U.S. Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Recommendations and Reports / Vol. 73 / No. 4
August 8, 2024

U.S. Medical Eligibility Criteria for Contraceptive Use, 2024

U.S. DEPARTMENT OF
HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE
CONTROL AND PREVENTION

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The *MMWR* series of publications is published by the Office of Science, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested citation: [Author names; first three, then et al., if more than six.] [Title]. MMWR Recomm Rep 2024;73(No. RR-#):[inclusive page numbers].

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U.S. Medical Eligibility Criteria for Contraceptive Use, 2024

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Summary

The 2024 U.S. Medical Eligibility Criteria for Contraceptive Use *(U.S. MEC) comprises recommendations for the use of specific contraceptive methods by persons who have certain characteristics or medical conditions. These recommendations for health care providers were updated by CDC after review of the scientific evidence and a meeting with national experts in Atlanta, Georgia, during January 25–27, 2023. The information in this report replaces the 2016 U.S. MEC* (CDC. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. MMWR 2016:65[No. RR-3]:1–103). *Notable updates include 1) the addition of recommendations for persons with chronic kidney disease; 2) revisions to the recommendations for persons with certain characteristics or medical conditions (i.e., breastfeeding, postpartum, postabortion, obesity, surgery, deep venous thrombosis or pulmonary embolism with or without anticoagulant therapy, thrombophilia, superficial venous thrombosis, valvular heart disease, peripartum cardiomyopathy, systemic lupus erythematosus, high risk for HIV infection, cirrhosis, liver tumor, sickle cell disease, solid organ transplantation, and drug interactions with antiretrovirals used for prevention or treatment of HIV infection); and 3) inclusion of new contraceptive methods, including new doses or formulations of combined oral contraceptives, contraceptive patches, vaginal rings, progestin-only pills, levonorgestrel intrauterine devices, and vaginal pH modulator. The recommendations in this report are intended to serve as a source of evidence-based clinical practice guidance for health care providers. The goals of these recommendations are to remove unnecessary medical barriers to accessing and using contraception and to support the provision of person-centered contraceptive counseling and services in a noncoercive manner. Health care providers should always consider the individual clinical circumstances of each person seeking contraceptive services. This report is not intended to be a substitute for professional medical advice for individual patients; when needed, patients should seek advice from their health care providers about contraceptive use.*

Introduction

U.S. Medical Eligibility Criteria for Contraceptive Use, 2024 (U.S. MEC) provides recommendations for health care providers for safe use of contraceptive methods for persons who have certain characteristics or medical conditions within the framework of removing unnecessary medical barriers to accessing and using contraception. U.S. MEC is a companion document to *U.S. Selected Practice Recommendations for Contraceptive Use, 2024* (U.S. SPR) (*1*), which provides recommendations for health care providers that address provision of contraceptive methods and management of side effects and issues related to contraceptive method use (*2*). Both U.S. MEC and U.S. SPR were adapted from global guidance developed by the World Health Organization (WHO) (*3*,*4*). WHO intended for the global guidance to be used by local or national policymakers, family planning program managers, and the scientific community as a reference when they develop family planning guidance at the country or program level (*3*).

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CDC first published U.S. MEC in 2010, after a formal process during 2008–2010 to adapt the global guidance for use in the United States, which included rigorous identification and critical appraisal of the scientific evidence through systematic reviews and input from national experts on how to translate that evidence into recommendations for U.S. health care providers (*5*); a subsequent update was published in 2016 (*6*).

U.S. MEC and U.S. SPR recommendations are components of quality contraceptive services and can be used in conjunction with other guidance documents such as *Providing Quality Family Planning Services: Recommendations of CDC and the U.S. Office of Population Affairs*, which provides recommendations for the content and delivery of services related to preventing or for achieving pregnancy (*7*–*9*). Evidence-based guidance can support health care providers when providing person-centered counseling and contraceptive services, including assisting persons in selecting and using contraceptive methods safely and effectively.

Equitable access to the full range of contraceptive methods for all those seeking care is an essential component of high-quality sexual and reproductive health care. Contraceptive services should be offered in a noncoercive manner that supports a person's values, goals, and reproductive autonomy

through a shared decision-making process with health care providers (*10*–*14*). Because of the history of and ongoing forced sterilization and reproductive coercion in the United States among persons of racial and ethnic minority groups, persons with disabilities, and other groups that have been marginalized, it is important that persons can select the method that best meets their needs to promote reproductive autonomy (*10*–*12*).

This report replaces the 2016 version of U.S. MEC (*6*) with new and revised recommendations, on the basis of new evidence and input from experts. This updated document uses gender-inclusive language throughout. However, when summarizing published evidence that describes study populations by specific genders, the wording of the primary studies has been maintained for accuracy. A summary of new and revised recommendations from the 2016 U.S. MEC is provided (Appendix A). Notable updates include

- addition of recommendations for persons with chronic kidney disease, specifically those with nephrotic syndrome, those receiving hemodialysis, and those receiving peritoneal dialysis;
- revisions to recommendations for persons with certain characteristics or medical conditions (i.e., breastfeeding, postpartum, postabortion, obesity, surgery, history of deep venous thrombosis or pulmonary embolism with or without anticoagulant therapy, thrombophilia, superficial venous thrombosis, valvular heart disease, peripartum cardiomyopathy, systemic lupus erythematosus, cirrhosis, liver tumor, sickle cell disease, and solid organ transplantation);
- revisions to recommendations for persons at high risk for HIV infection (this recommendation was developed and published in 2020) (*15*);
- revisions to recommendations for drug interactions with antiretrovirals to include prevention in addition to treatment for HIV infection (this recommendation was developed and published in 2020) (*15*); and
- inclusion of additional contraceptive methods, including new doses or formulations of combined oral contraceptives (COCs), contraceptive patches, vaginal rings, progestin-only pills (POPs), levonorgestrel intrauterine devices (LNG-IUDs), and vaginal pH modulator.

U.S. MEC recommendations are meant to serve as a source of evidence-based clinical guidance for health care providers and can support the provision of person-centered contraceptive counseling and services in a noncoercive manner. Health care providers should always consider the individual clinical circumstances of each person seeking contraceptive services. This report is not intended to be a substitute for professional medical advice for individual patients; when needed, patients should seek advice from their health care providers about contraceptive use.

Methods

Since publication of the 2016 U.S. MEC, CDC has monitored the literature for new evidence relevant to the recommendations through the WHO/CDC Continuous Identification of Research Evidence (CIRE) system (*16*). This system identifies new evidence as it is published and allows WHO and CDC to update systematic reviews and facilitate updates to recommendations as new evidence warrants. Automated searches are run in PubMed weekly, and the results are reviewed. Abstracts that meet specific criteria are added to the web-based CIRE system, which facilitates coordination and peer review of systematic reviews for both WHO and CDC. For this update, CDC reviewed all existing recommendations in the 2016 U.S. MEC for new evidence identified by CIRE that had the potential to lead to a changed recommendation. To obtain comments from the public about revisions to CDC's contraception recommendations (U.S. MEC and U.S. SPR), CDC published a notice in the Federal Register (86 FR 46703) on August 19, 2021, requesting public comment on content to consider for revision or addition to the recommendations and how to improve the implementation of the guidance documents (*17*). The comment period closed on October 18, 2021. CDC received 46 submissions from the general public, including private persons, professional organizations, academic institutions, and industry. CDC reviewed each of the submissions and carefully considered them when revising the recommendations.

During January 21, 25, and 26, 2022, CDC held virtual scoping meetings that included 27 participants with expertise in contraception, adolescent health, and thrombosis, as well as representatives from partner organizations, to solicit their individual input on the scope for updating both the 2016 U.S. MEC and 2016 U.S. SPR. The 27 invited participants represented various types of health care providers and health care provider organizations. Lists of participants and potential conflicts of interests are provided at the end of this report. Meeting participants discussed topics to be addressed in the update of U.S. MEC on the basis of the presentation of new evidence published since 2016 (identified through the CIRE system), submissions received through the Federal Register notice, and feedback CDC received from other sources (e.g., health care providers and others through e-mail, public inquiry, and questions received at conferences). CDC identified multiple topics to consider when updating the guidance, including revision of existing recommendations for certain characteristics or medical conditions (postpartum, postabortion, obesity, anticoagulant therapy, known thrombogenic mutations, viral hepatitis, cirrhosis, liver tumors, sickle cell disease, and solid organ transplantation), addition of recommendations for

new characteristics or medical conditions (chronic kidney disease and antiphospholipid syndrome), and addition of recommendations for new contraceptive methods (including new formulations of COCs, contraceptive patches, vaginal rings, POPs, LNG-IUDs, and vaginal pH modulator). CDC determined that all other recommendations in the 2016 U.S. MEC were up to date and consistent with the existing body of evidence for that recommendation.

In preparation for a subsequent expert meeting held during January 25–27, 2023, to review the scientific evidence for potential recommendations, CDC staff members and other invited authors conducted systematic reviews for each of the topics being considered. The purpose of these systematic reviews was to identify direct and indirect evidence about the safety of contraceptive method use by persons with selected characteristics or medical conditions (e.g., risk for disease progression or other adverse health effects in persons with chronic kidney disease who use combined hormonal contraceptives [CHCs]). Person-centered outcomes that might represent contraceptive users' values and preferences (e.g., method continuation and patient satisfaction) were considered where relevant and available for each of the systematic reviews. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed for reporting systematic reviews (*18*). The Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach was used to assess the certainty of the evidence (*19*,*20*). Certainty of evidence was rated as high, moderate, low, or very low depending on criteria including study design, risk for bias, indirectness, imprecision, and inconsistency. Outcomes evaluated in randomized clinical trials (RCTs) are considered to have high certainty of evidence and those in observational studies to have low certainty; these ratings are adjusted according to the previously mentioned criteria. When direct evidence was limited or not available, indirect evidence (e.g., evidence on proxy outcomes or among healthy persons) and theoretical issues were considered. Reviews are referenced and cited throughout this report; the full reviews will be submitted to peer-reviewed journals and will contain the details of each review, including the systematic review question, literature search protocol (registered in https://www.crd.york.ac.uk/PROSPERO), inclusion and exclusion criteria, evidence tables, and quality assessments. Brief summaries of the evidence and GRADE tables are included (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). CDC staff members continued to monitor new evidence identified through the CIRE system during the preparation for the January 2023 meeting.

In addition to the preparation of the systematic reviews, CDC included patient perspectives in the guideline update process to better consider how the resulting updated recommendations could meet patient preferences and needs. Consideration of patient perspectives can center discussions on the evidence in a person-centered care model, can support inclusion of patient perspectives along with provider perspectives on the evidence, and has the potential to shape recommendations (*14*,*21*,*22*). In November and December 2022, listening sessions were held with a different group of 18 participants, representing themselves or patient advocacy organizations, who provided perspectives from patient populations such as youths; lesbian, gay, bisexual, transgender, queer, and intersex (LGBTQI+) persons; persons with disabilities; and persons with chronic medical conditions. The goal of the listening sessions was to gather insights about participants' experiences, values, preferences, and information needs related to contraceptive choice and decision-making.

During January 25–27, 2023, in Atlanta, Georgia, CDC held a meeting with 40 participants who were invited to provide their individual perspectives on the scientific evidence presented and the implications for practice for U.S. MEC. Thirty-eight participants represented a wide range of expertise in contraception provision, research, and reproductive justice and included obstetricians and gynecologists, pediatricians, family physicians, internal medicine physicians, nurse practitioners, epidemiologists, and others with research and clinical practice expertise in contraceptive safety, effectiveness, and management. Two participants were patient representatives who provided their individual perspectives on the topics discussed throughout the meeting. Six additional participants with expertise relevant to specific topics on the meeting agenda provided information and participated in the discussion on their topic of expertise only (e.g., an expert in kidney disease was asked to provide general information about the condition and to assist in interpreting the evidence and any theoretical concerns on the use of contraceptive methods in persons with the condition). During the meeting, a summary of the information from the patient listening sessions was presented, and the two patient representatives presented information on their individual experiences and perspectives related to receipt of contraceptive services. The evidence from the systematic review for each topic was presented, including direct evidence and any indirect evidence or theoretical concerns. Meeting participants provided their individual perspectives on topics discussed throughout the meeting and on using the evidence to develop recommendations that would meet the needs of U.S. health care providers and the patients they serve. Participants also provided feedback on the certainty of evidence, the balance

of benefits and harms, and values and preferences. Areas of research that need additional investigation also were considered during the meeting. Lists of participants and potential conflicts of interest are provided at the end of this report.

After the January 2023 meeting, CDC determined the recommendations in this report, taking into consideration the individual perspectives provided by the meeting participants. Feedback also was received from a group of four external reviewers, composed of health care providers and researchers who had not participated in the scoping or update meetings. These external reviewers were asked to provide comments on the accuracy, feasibility, and clarity of the recommendations.

Keeping Guidance Up to Date

As with any evidence-based guidance document, a key challenge is keeping the recommendations up to date as new scientific evidence becomes available. Working with WHO, CDC uses the CIRE system to ensure that WHO and CDC guidance is based on the best available evidence and that a mechanism is in place to update guidance when new evidence becomes available (*16*). CDC will continue to work with WHO to identify and assess all new relevant evidence and determine whether changes in the recommendations are warranted. CDC will completely review U.S. MEC periodically. Updates to the guidance will published in CDC's *Morbidity and Mortality Weekly Report* (*MMWR*) and posted on the CDC website (https://www.cdc.gov/contraception/hcp/contraceptive-guidance).

As part of the process to update these recommendations, CDC identifies gaps in the evidence for the recommendations considered. Evidence is often limited on the safety of contraceptive methods among persons with certain characteristics or medical conditions. Generalizability of the published evidence to all persons seeking contraceptive services presents a challenge because of biases about who might be included in studies on contraceptive safety. New, high-quality research on contraception that addresses priority research gaps inclusive of diverse populations can further strengthen these recommendations and improve clinical practice.

How to Use This Document

The recommendations in this report are intended to help health care providers determine the safe use of contraceptive methods among persons with certain characteristics and medical conditions. Providers can use the information in these recommendations during contraceptive counseling with patients. The tables include recommendations for the use of contraceptive methods by persons with certain characteristics or medical conditions. Each condition is defined as representing either a person's characteristics (e.g., age or postpartum status) or a known medical condition (e.g., diabetes or hypertension). The recommendations refer to contraceptive methods being used for contraceptive purposes; the recommendations do not consider the use of contraceptive methods for treatment of medical conditions because the eligibility criteria in these situations might differ. The conditions affecting eligibility for the use of each contraceptive method are classified into one of four categories (Box 1).

Contraceptive Decision-Making

CDC acknowledges the paramount importance of personal autonomy in contraceptive decision-making. This is critically important because of the context of historical and ongoing contraceptive coercion and reproductive mistreatment in the United States, especially among communities that have been marginalized, including human rights violations such as forced sterilization and enrollment in contraceptive trials without informed consent (*10*–*12*). Coercive practices in the health care system can include provider bias for certain contraceptive methods over a patient's reproductive goals and preferences, lack of person-centered counseling and support, and policies or incentives for uptake of certain contraceptive methods (*11*). For health care providers and the settings in which they work, it is important to acknowledge the structural systems that drive inequities (e.g., discrimination because of race, ethnicity, disability, sex, gender, and sexual orientation), work to mitigate harmful impacts, and recognize that provider bias (unconscious or explicit) might affect contraceptive counseling and provision of services (*12*). All persons seeking contraceptive care need access to appropriate counseling and services that

BOX 1. Categories of medical eligibility criteria for contraceptive use

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

support the person's values, goals, and reproductive autonomy (*10*–*14*). Health care providers can support the contraceptive needs of all persons by using a person-centered framework and recognizing the many factors that influence individual decision-making about contraception (*10*,*12*,*14*).

The U.S. MEC and U.S. SPR recommendations can be used to support a person's contraceptive decision-making (Box 2). Persons should have equitable access to the full range of contraceptive methods and be given the information they need for contraceptive decision-making in a noncoercive manner. Patient-centeredness has been defined by the Institute of Medicine as "providing care that is respectful of and responsive to individual patient preferences, needs, and values and ensuring that patient values guide all clinical decisions" (*23*). Shared decision-making and person-centered approaches to providing health care recognize the expertise of both the medical provider and the patient (*10*,*12*,*23*).

Health care providers should always consider the individual clinical and social factors of each person seeking contraceptive services and discuss reproductive desires, expectations, preferences, and priorities regarding contraception. A person might consider and prioritize many elements when choosing an acceptable contraceptive method, such as safety, effectiveness (*24*), availability (including accessibility and affordability), side effects, user control, reversibility, and ease of removal or discontinuation. In addition, a person's health risks associated with pregnancy and access to comprehensive health care services should be considered in these discussions. A person-centered approach to contraceptive decision-making prioritizes a person's preferences and reproductive autonomy rather than a singular focus on pregnancy prevention and respects the person as the main decision-maker in contraceptive decisions, including the decision not to use contraception or to discontinue contraceptive method use (*12*,*25*). Voluntary informed choice of contraceptive methods is an essential guiding principle, and contraceptive counseling, where applicable, might be an important contributor to the successful use of contraceptive methods. Key resources provide additional information on person-centered contraceptive counseling and care (*7*,*10*,*12*,*26*).

Using U.S. MEC Categories in Practice

Health care providers can use the eligibility categories when assessing the safety of contraceptive method use for persons with certain characteristics or medical conditions. Category 1 comprises conditions for which no restrictions exist for use of the contraceptive method. However, category 1 does not imply that the method is the most appropriate choice for a person, who might be prioritizing other factors when considering contraception. Classification of a method or condition as category 2 indicates the method generally can be used, with additional discussion about risks and benefits, and careful follow-up might be required. For a method or condition classified as category 3, use of that method usually is not recommended unless other more appropriate methods are not available or acceptable. The severity of the condition and the availability, practicality, and acceptability of alternative methods should be considered, and careful follow-up is required. Hence, provision of a contraceptive method to a person with a condition classified as category 3 requires careful clinical judgment and might warrant additional counseling, consultation, or follow-up. Category 4 comprises conditions that represent an unacceptable health risk if the method is used. For example, a person who smokes and is aged <35 years generally can use COCs (category 2). However, for a person

BOX 2. Using the *U.S. Medical Eligibility Criteria for Contraceptive Use* and *U.S. Selected Practice Recommendations for Contraceptive Use* recommendations to support contraceptive decision-making

- CDC acknowledges the paramount importance of personal autonomy in contraceptive decision-making.
- Persons should have equitable access to the full range of contraceptive methods.
- Contraceptive services should be offered in a noncoercive manner that honors a person's values, goals, and reproductive autonomy.
- Shared decision-making and person-centered approaches recognize the expertise of both the health care provider and the person.
- A person-centered approach to contraceptive decision-making
  - prioritizes a person's preferences and reproductive autonomy rather than a singular focus on pregnancy prevention,
  - respects the person as the main decision-maker in contraceptive decisions, and
  - includes respecting the decision not to use contraception or to discontinue contraceptive method use.
- U.S. MEC and U.S. SPR recommendations can be used by health care providers to support persons in contraceptive decision-making.
- U.S. MEC and U.S. SPR recommendations can be used by health care providers to remove unnecessary medical barriers to accessing and using contraception.

Abbreviations: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*; U.S. SPR = *U.S. Selected Practice Recommendations for Contraceptive Use*.

aged ≥35 years who smokes <15 cigarettes per day, the use of COCs usually is not recommended unless other methods are not available or acceptable (category 3). A person aged ≥35 years who smokes ≥15 cigarettes per day should not use COCs because of unacceptable health risks, primarily the risk for myocardial infarction and stroke (category 4). The implementation of this clinical guidance might vary within different health systems, clinics, or settings. For example, in certain settings, category 3 might mean that a special consultation is warranted. Health departments and medical societies or organizations can provide information on implementation through additional guidance or clinical protocols.

The recommendations address medical eligibility criteria for the initiation and continued use of all contraceptive methods evaluated. The issue of medical eligibility criteria for continuation of a contraceptive method is clinically relevant whenever a medical condition develops or worsens during use of a contraceptive method. When the categories differ for initiation and continuation, these differences are noted. When different initiation and continuation recommendations are not given, the category is the same for initiation and continuation of use.

On the basis of this classification system, the eligibility criteria for initiating and continuing use of a specific contraceptive method are presented in tables (Appendices A, B, C, D, E, and J). In these tables, the first column indicates the condition. Multiple conditions are divided into subconditions to differentiate between varying condition types or severity. The next columns provide classifications of the condition for initiation, continuation, or both into categories 1, 2, 3, or 4 for specific contraceptive methods. For certain conditions, the last column further clarifies the numeric category in cases where the numeric classification does not adequately capture the recommendation. These clarifications are considered a necessary element of the recommendation. The last column also summarizes the evidence for the recommendation if evidence exists. The recommendations for which no evidence is cited might be based on information from sources other than systematic reviews and might take into account individual perspectives from either the WHO or U.S. expert meetings in which these recommendations were developed. For certain recommendations, comments in the third column can provide additional rationale or other information about the recommendation. Information provided along with the numeric recommendation (i.e., clarifications, evidence, and comments) is additional detail that providers can use as part of their counseling and referrals, as needed.

U.S. MEC recommendations comprise one aspect of contraceptive counseling. All persons should be counseled about the full range of contraceptive options for which they are medically eligible. Voluntary informed choice of contraceptive methods is an essential guiding principle of these recommendations, and person-centered contraceptive counseling can help to ensure a person's contraceptive needs are met successfully.

Recommendations for Use of Contraceptive Methods

The classifications for whether persons with certain characteristics or medical conditions can safely use specific contraceptive methods are provided for intrauterine devices (IUDs), including the copper IUD (Cu-IUD) and LNG-IUD (Appendix B); progestin-only contraceptives (POCs), including progestin-only implants, depot medroxyprogesterone acetate injections, and POPs (Appendix C); CHCs, including COCs, combined transdermal patches, and combined vaginal rings (Appendix D); barrier contraceptive methods, including external (male) and internal (female) condoms, spermicides and vaginal pH modulator, and diaphragm with spermicide or cervical cap with spermicide (Appendix E); fertility awareness–based methods (Appendix F); lactational amenorrhea method (Appendix G); coitus interruptus (Appendix H); permanent contraception, including tubal surgery and vasectomy (Appendix I); and emergency contraception, including emergency use of the Cu-IUD and emergency contraceptive pills (Appendix J). A table at the end of this report summarizes the classifications for the hormonal and intrauterine methods (Appendix K).

Prevention of Sexually Transmitted Infections

All patients, regardless of contraceptive choice, should be counseled about the use of condoms and the risk for sexually transmitted infections (STIs), including HIV infection (*27*). Most contraceptive methods, such as hormonal methods, IUDs, and permanent contraception do not protect against STIs, including HIV infection. Consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*27*). Although evidence is limited, use of internal (female) condoms can provide protection from acquisition and transmission of STIs (*27*). Patients also should be counseled that pre-exposure prophylaxis (PrEP), when taken as prescribed, is highly effective for preventing HIV infection (*28*). Additional information about prevention and treatment of STIs is available from CDC's *Sexually Transmitted Infections Treatment Guidelines* (https://www.cdc.gov/std/treatment-guidelines/default.htm) (*27*), and information on PrEP for prevention of HIV infection is available from the U.S.

Public Health Service's *Preexposure Prophylaxis for the Prevention of HIV Infection in the United States — 2021 Update: A Clinical Practice Guideline* (https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf) (*28*).

Pregnancy and Increased Health Risk

Discussion of health risks associated with pregnancy is an important aspect of contraceptive counseling. For persons with certain medical conditions, pregnancy poses increased health risks. Conditions included in U.S. MEC that are associated with increased risk for adverse health events as a result of pregnancy are identified throughout the document (Box 3). This is not a comprehensive list of all conditions that could lead to adverse events during pregnancy. Certain medical conditions included in U.S. MEC recommendations also are treated with teratogenic drugs, which could have adverse effects when used during pregnancy. When applying U.S. MEC classifications during person-centered counseling, health care providers should discuss the risks of a particular contraceptive method as well as the health risks associated with pregnancy. Even though permanent contraception and long-acting, reversible contraceptive methods are highly effective, persons should be provided with the full range of contraceptive options and supported in their autonomous decisions about pregnancy planning and contraceptive choices. Discussions about pregnancy should include reviewing access to comprehensive health care services and subspecialists for a high-risk pregnancy (*29*).

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BOX 3. Conditions included in *U.S. Medical Eligibility Criteria for Contraceptive Use* associated with increased risk for adverse health events as a result of pregnancy*

- Breast cancer
- Chronic kidney disease: with current nephrotic syndrome, receiving hemodialysis, or receiving peritoneal dialysis
- Complicated valvular heart disease
- Cystic fibrosis
- Decompensated cirrhosis
- Deep venous thrombosis/pulmonary embolism
- Diabetes: insulin dependent; with nephropathy, retinopathy, or neuropathy or other vascular disease; or of >20 years' duration
- Endometrial cancer
- Epilepsy
- Gestational trophoblastic disease
- Hepatocellular adenoma and malignant liver tumors (hepatocellular carcinoma)
- History of bariatric surgery within the past 2 years
- HIV infection: not clinically well or not receiving antiretroviral therapy
- Hypertension (systolic ≥160 mm Hg or diastolic ≥100 mm Hg)
- Ischemic heart disease
- Ovarian cancer
- Peripartum cardiomyopathy
- Schistosomiasis with fibrosis of the liver
- Sickle cell disease
- Solid organ transplantation within the past 2 years
- Stroke
- Systemic lupus erythematosus
- Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome)
- Tuberculosis

* Even though permanent contraception and long-acting, reversible contraceptive methods are highly effective, persons should be provided with the full range of contraceptive options and supported in their autonomous decisions about pregnancy planning and contraceptive choices. Discussions about pregnancy should include reviewing access to comprehensive health care services and subspecialists for a high-risk pregnancy.

Acknowledgments

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Conflicts of interest for invited meeting participants, January 21, 2022, virtual: Michael Streiff, consultation for Bayer, Janssen, Pfizer, and Portola, recipient of grants to support research from Boehringer Ingelheim, Janssen, Novo Nordisk, Portola, Sanofi, Tremeau pharmaceuticals, conducted lectures for Bayer, Pfizer, and Portola; Alison Edelman, consultant for American College of Obstetricians and Gynecologists (ACOG), supports medical eligibility criteria activities for World Health Organization, Oregon Health & Science University receives research funding from Merck and HRA Pharma; Andrew Kaunitz, University of Florida College of Medicine receives financial support for clinical trials sponsored by Merck and Mithra; Carolyn Westhoff, editor of Contraception, consultant for Merck and Bayer, member of a number of data safety and monitoring boards for overseeing phase 4 Food and Drug Administration–mandated studies of new contraceptives, Columbia University receives research funding for clinical trials for each new contraceptive discussed.

CDC Subject Matter Experts and Attendees, January 21, 2022, Virtual

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Conflicts of interest for invited meeting participants, January 25–26, 2022, virtual: Elise Berlan, Nexplanon clinical trainer for Merck/Organon, received research funding from Merck/Organon; Nicole Chaisson, Nexplanon trainer for Organon; Alison Edelman, consultant for American College of Obstetricians and Gynecologists (ACOG), supports medical eligibility criteria activities for World Health Organization, Oregon Health & Science University receives research funding from Merck and HRA Pharma; Carolyn Westhoff, editor of *Contraception*, consultant for Merck and Bayer, member of a number of data safety and monitoring boards for overseeing phase 4 Food and Drug Administration–mandated studies of new contraceptives, Columbia University receives research funding for clinical trials for each new contraceptive discussed.

CDC Subject Matter Experts and Attendees, January 25–26, 2022, Virtual

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Conflicts of interest for invited meeting participants, January 25–27, 2023, Atlanta, Georgia: Elise Berlan, Nexplanon clinical trainer for Merck/Organon; Nicole Chaisson, Nexplanon clinical trainer for Merck/Organon; Mitchell Creinin, received honorarium from Gedeon Richter, Mayne, and Organon, served on advisory board for Gedeon Richter, GlaxoSmithKline, OLIC, and Organon, consulted for Danco, Estetra SRL, FHI360, Mayne, and Medicines360, University of California-Davis, receives contraceptive research funding from Chemo Research SL, Evofem, Medicines360, Merck, Sebela, and National Institutes of Health National Institute of Child Health and Human Development; Alison Edelman, receives travel reimbursement from American College of Obstetricians and Gynecologists, World Health Organization, CDC, and Gynuity for committee activities, receives royalties from Up to Date, Inc., Oregon Health & Science University receives research funding from Oregon Health & Science University Foundation, Merck, HRA Pharma, Bill & Melinda Gates Foundation, and National Institutes of Health; Emily Godfrey, works with Organon and received honoraria as Nexplanon trainer; Andrew Kaunitz, consultant to Mithra, University of Florida receives research support from Bayer, Merck, Mithra, and Mylan; Aaron Lazorwitz, receives research support from Organon for investigator-initiated research with the etonogestrel contraceptive implant; Yvanna Marlin-Guanga, employed under CommunicateHealth, contractor for U.S. Medical Eligibility Criteria for Contraceptive Use and U.S. Selected Practice Recommendations for Contraceptive Use January 2023 meeting; Rachel Martin, employed under CommunicateHealth, contractor for U.S. Medical Eligibility Criteria for Contraceptive Use and U.S. Selected Practice Recommendations for Contraceptive Use January 2023 meeting; Lydia Pecker, consulted for Novo Nordisk and Global Blood Therapeutics, receives research support from Alexion, National Institutes of Health National Heart, Lung, and Blood Institute, Mellon Foundation, American Society of Hematology, and Doris Duke Foundation; Michael Streiff, consultant for CSL Behring data safety monitoring board member, Janssen consultant on management of cancer-associated thromboembolism, and Pfizer consultant on anticoagulation for venous thromboembolism; Katharine White, receives research support through institution from Bayer, Merck, and Evofem; Tracey Wilkinson, receives project funding from Bayer, Cooper Surgical, and Organon, and nonpaid consultant for HRA Pharma.

CDC Subject Matter Experts and Attendees, January 25–27, 2023, Atlanta, Georgia

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Conflicts of Interest

All authors have completed and submitted the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest. No potential conflicts of interest were disclosed. To promote transparency, all meeting participants were asked to disclose potential conflicts of interest to CDC before the expert meeting and to report potential conflicts of interest during the introductory portion of the expert meeting. All potential conflicts of interest disclosed by meeting participants are listed. No participants were excluded from discussion based on potential conflicts of interest. CDC staff members who ultimately decided and developed these recommendations have no financial interests or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters relevant to these recommendations.

References

1. Curtis KM, Nguyen AT, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2024. MMWR Recomm Rep 2024;73(No. RR-3):1–77.
2. Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2016. MMWR Recomm Rep 2016;65(No. RR-4):1–66. PMID:27467319 https://doi.org/10.15585/mmwr.rr6504a1
3. World Health Organization. Medical eligibility criteria for contraceptive use. Geneva, Switzerland: World Health Organization; 2015. https://www.who.int/publications/i/item/9789241549158
4. World Health Organization. Selected practice recommendations for contraceptive use. Geneva, Switzerland: World Health Organization; 2016. https://www.who.int/publications/i/item/9789241565400
5. CDC. U.S. medical eligibility criteria for contraceptive use, 2010. MMWR Recomm Rep 2010;59(No. RR-4):1–86. PMID:20559203
6. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. MMWR Recomm Rep 2016;65(No. RR-3):1–103. PMID:27467196 https://doi.org/10.15585/mmwr.rr6503a1
7. Gavin L, Moskosky S, Carter M, et al.; CDC. Providing quality family planning services: recommendations of CDC and the U.S. Office of Population Affairs. MMWR Recomm Rep 2014;63(No. RR-4):1–54. PMID:24759690

8. Gavin L, Pazol K. Update: providing quality family planning services—recommendations from CDC and the U.S. Office of Population Affairs, 2015. MMWR Morb Mortal Wkly Rep 2016;65:231–4. PMID:26963363 https://doi.org/10.15585/mmwr.mm6509a3
9. Gavin L, Pazol K, Ahrens K. Update: providing quality family planning services—recommendations from CDC and the U.S. Office of Population Affairs. MMWR Morb Mortal Wkly Rep 2017;66:1383–5. PMID:29267259 https://doi.org/10.15585/mmwr.mm6650a4
10. American College of Obstetricians and Gynecologists' Committee on Health Care for Underserved Women, Contraceptive Equity Expert Work Group, and Committee on Ethics. Patient-centered contraceptive counseling: ACOG committee statement number 1. Obstet Gynecol 2022;139:350–3. PMID:35061341 https://doi.org/10.1097/AOG.0000000000004659
11. American Public Health Association. Opposing coercion in contraceptive access and care to promote reproductive health equity. Washington, DC: American Public Health Association; 2021. https://www.apha.org/Policies-and-Advocacy/Public-Health-Policy-Statements/Policy-Database/2022/01/07/Contraceptive-Access
12. Holt K, Reed R, Crear-Perry J, Scott C, Wulf S, Dehlendorf C. Beyond same-day long-acting reversible contraceptive access: a person-centered framework for advancing high-quality, equitable contraceptive care. Am J Obstet Gynecol 2020;222(4S):S878.e1–e6. PMID:31809706 https://doi.org/10.1016/j.ajog.2019.11.1279
13. United Nations Population Fund. Programme of Action of the International Conference on Population and Development. Cairo, Egypt: United Nations; 1995. https://unfpa.org/sites/default/files/pub-pdf/programme_of_action_Web%20ENGLISH.pdf
14. World Health Organization. Framework for ensuring human rights in the provision of contraceptive information and services. Geneva, Switzerland: World Health Organization; 2014. https://www.who.int/publications/i/item/9789241507745
15. Tepper NK, Curtis KM, Cox S, Whiteman MK. Update to U.S. medical eligibility criteria for contraceptive use, 2016: updated recommendations for the use of contraception among women at high risk for HIV infection. MMWR Morb Mortal Wkly Rep 2020;69:405–10. PMID:32271729 https://doi.org/10.15585/mmwr.mm6914a3
16. Mohllajee AP, Curtis KM, Flanagan RG, Rinehart W, Gaffield ML, Peterson HB. Keeping up with evidence a new system for WHO's evidence-based family planning guidance. Am J Prev Med 2005;28:483–90. PMID:15894153 https://doi.org/10.1016/j.amepre.2005.02.008
17. CDC. Updating CDC's contraception guidance documents: U.S. medical eligibility criteria for contraceptive use and U.S. selected practice recommendations for contraceptive use. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.govinfo.gov/content/pkg/FR-2021-08-31/pdf/2021-18769.pdf
18. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. PMID:33782057 https://doi.org/10.1136/bmj.n71
19. Guyatt G, Oxman AD, Akl EA, et al. GRADE guidelines: 1. Introduction-GRADE evidence profiles and summary of findings tables. J Clin Epidemiol 2011;64:383–94. PMID:21195583 https://doi.org/10.1016/j.jclinepi.2010.04.026
20. Guyatt GH, Oxman AD, Vist GE, et al.; GRADE Working Group. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. BMJ 2008;336:924–6. PMID:18436948 https://doi.org/10.1136/bmj.39489.470347.AD
21. Armstrong MJ, Rueda JD, Gronseth GS, Mullins CD. Framework for enhancing clinical practice guidelines through continuous patient engagement. Health Expect 2017;20:3–10. PMID:27115476 https://doi.org/10.1111/hex.12467
22. Institute of Medicine Committee on Standards for Developing Trustworthy Clinical Practice Guidelines. Clinical practice guidelines we can trust. In: Graham R, Mancher M, Miller Wolman D, Greenfield S, Steinberg E, eds. Clinical practice guidelines we can trust. Washington, DC: National Academies Press; 2011.
23. Institute of Medicine. Crossing the quality chasm: a new health system for the 21st century. Washington, DC: National Academies Press; 2001.
24. Bradley SEK, Polis CB, Micks EA, Steiner MJ. Effectiveness, safety, and comparative side effects. In: Cason P, Cwiak C, Edelman A, Kowal D, Marrazzo JM, Nelson AL, et al., editors. Contracept Technol. 22nd ed. Burlington, MA: Jones-Bartlett Learning; 2023.
25. Potter JE, Stevenson AJ, Coleman-Minahan K, et al. Challenging unintended pregnancy as an indicator of reproductive autonomy. Contraception 2019;100:1–4. PMID:30851238 https://doi.org/10.1016/j.contraception.2019.02.005
26. Reproductive Health National Training Center. Contraceptive counseling and education eLearning. Washington, DC: US Department of Health and Human Services, Office of the Assistant Secretary for Health, Office of Population Affairs, Office on Women's Health; 2022. https://rhntc.org/resources/contraceptive-counseling-and-education-elearning
27. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70:1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
28. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
29. Blackwell S, Louis JM, Norton ME, et al. Reproductive services for women at high risk for maternal mortality: a report of the workshop of the Society for Maternal-Fetal Medicine, the American College of Obstetricians and Gynecologists, the Fellowship in Family Planning, and the Society of Family Planning. Am J Obstet Gynecol 2020;222:B2–18. PMID:32252942 https://doi.org/10.1016/j.ajog.2019.12.008

Appendix A: Summary of Changes from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

The classification additions, deletions, and modifications from the 2016 *U.S. Medical Eligibility Criteria for Contraceptive Use* (U.S. MEC) are summarized in this appendix (Box A1) (Tables A1, A2, and A3). For conditions for which classifications changed for one or more contraceptive methods or for which the condition description underwent a substantive modification, the changes or modifications are noted (Tables A1, A2, and A3). Conditions that do not appear in this table remain unchanged from the 2016 U.S. MEC.

BOX A1. Categories for classifying intrauterine devices and hormonal contraceptives

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE A1. Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
Breastfeeding							
a. <21 days postpartum	—	—	2	2	2	4	Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
b. 21 to <30 days postpartum							
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	2	2	2	3	**CHC:** For persons with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Breastfeeding:** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
ii. Without other risk factors for VTE	—	—	2	2	2	3	Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
c. 30–42 days postpartum							
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1	2*	1	3	**CHC:** For persons with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Breastfeeding:** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
ii. Without other risk factors for VTE	—	—	1	1	1	2	Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
d. >42 days postpartum	—	—	1	1	1	2	Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
Postpartum (nonbreastfeeding)							
a. <21 days postpartum	—	—	1	2*	1	4	—
b. 21–42 days postpartum							
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1	2*	1	3	**CHC:** For persons with other risk factors for VTE, these risk factors might increase the classification to a category 4.
ii. Without other risk factors for VTE	—	—	1	1	1	2	—
c. >42 days postpartum	—	—	1	1	1	1	—
Postpartum (including cesarean delivery, breastfeeding, or nonbreastfeeding)							
a. <10 minutes after delivery of the placenta	2*	2*	—	—	—	—	**IUD:** Postpartum placement of IUDs is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Breastfeeding:** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
b. 10 minutes after delivery of the placenta to <4 weeks	2	2	—	—	—	—	**IUD:** Postpartum placement of IUDs is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Breastfeeding:** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
c. ≥4 weeks	1	1	—	—	—	—	**IUD:** Postpartum placement of IUDs is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Breastfeeding:** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*1*) or up to age 2 years or longer (*2*).
d. Postpartum sepsis	4	4	—	—	—	—	—
Postabortion (spontaneous or induced)							
a. First trimester abortion							
i. Procedural (surgical)*	1	1	1	1	1	1	**IUD:** IUDs may be placed immediately after abortion completion. **POC:** POCs may be started immediately after abortion completion or at time of medication abortion initiation. **DMPA**: After a first trimester medication abortion that did not include mifepristone, there is no restriction for the use of DMPA (category 1). After a first trimester medication abortion that included mifepristone, there is no restriction for use of DMPA after abortion completion (category 1) and benefits generally outweigh risks with DMPA use immediately at time of medication abortion initiation (category 2). Concurrent administration of DMPA with mifepristone might slightly decrease medication abortion effectiveness and increase risk for ongoing pregnancy. Risk for ongoing pregnancy with concurrent administration of DMPA with mifepristone should be considered along with personal preference and access to follow-up abortion and contraceptive care.* **CHC:** CHCs may be started immediately after abortion completion or at time of medication abortion initiation.
ii. Medication*	1	1	1	1/2*	1	1	
iii. Spontaneous abortion with no intervention*	1	1	1	1	1	1	
b. Second trimester abortion							
i. Procedural (surgical)*	2	2	1	1	1	1	**IUD:** IUDs may be placed immediately after abortion completion. **POC:** POCs may be started immediately after abortion completion or at time of medication abortion initiation. **CHC:** CHCs may be started immediately after abortion completion or at time of medication abortion initiation.
ii. Medication*	2	2	1	1	1	1	
iii. Spontaneous abortion with no intervention*	2	2	1	1	1	1	
c. Immediate postseptic abortion	4	4	1	1	1	1	**POC:** POCs may be started immediately after abortion completion or at time of medication abortion initiation. **CHC:** CHCs may be started immediately after abortion completion or at time of medication abortion initiation.

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
Obesity							
a. BMI ≥30 kg/m^2	1	1	1	1	1	2	**CHC:** Risk for thrombosis increases with multiple risk factors, such as obesity, older age (e.g., ≥40 years), diabetes, smoking, family history of thrombosis, and dyslipidemia. When a person has multiple risk factors, any of which alone would increase risk for thrombosis, use of CHCs might increase thrombosis risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two category 2 risk factors might not necessarily warrant a higher category.*
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	1	2	1	2	**CHC:** Risk for thrombosis increases with multiple risk factors, such as obesity, older age (e.g., ≥40 years), diabetes, smoking, family history of thrombosis, and dyslipidemia. When a person has multiple risk factors, any of which alone would increase risk for thrombosis, use of CHCs might increase thrombosis risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two category 2 risk factors might not necessarily warrant a higher category.*
Surgery							
a. Minor surgery without immobilization	1	1	1	1	1	1	—
b. Major surgery							
i. Without prolonged immobilization	1	1	1	1	1	2	—
ii. With prolonged immobilization	1	1*	1*	2	1*	4	—

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
Deep venous thrombosis/ Pulmonary embolism							
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).							
a. Current or history of DVT/PE, receiving anticoagulant therapy (therapeutic dose) (e.g., acute DVT/PE or long-term therapeutic dose)*	2	2	2	2	2	3*	**Cu-IUD:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. Cu-IUDs might worsen bleeding.* **LNG-IUD:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. LNG-IUDs can be of benefit in preventing or treating this complication. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.* **POC:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. POCs can be of benefit in preventing or treating these complications; benefits might vary by POC dose and formulation. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.* **CHC:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. CHCs can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.* **CHC:** When a patient discontinues therapeutic dose of anticoagulant therapy, careful consideration should be given to transitioning from CHCs to a progestin-only or nonhormonal method, if acceptable to the patient.*

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
b. History of DVT/PE, receiving anticoagulant therapy (prophylactic dose)*							**Cu-IUD:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. Cu-IUDs might worsen bleeding.* **LNG-IUD:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. LNG-IUDs can be of benefit in preventing or treating this complication. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.* **POC:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. POCs can be of benefit in preventing or treating these complications; benefits might vary by POC dose and formulation. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.* **CHC:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. CHCs can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.*
i. Higher risk for recurrent DVT/PE (one or more risk factors)*	2	2	2	3*	2	4	
• Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome)*							
• Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer*							
• History of recurrent DVT/PE*							
ii. Lower risk for recurrent DVT/PE (no risk factors)*	2	2	2	2	2	3	
c. History of DVT/PE, not receiving anticoagulant therapy*							
i. Higher risk for recurrent DVT/PE (one or more risk factors)*	1	2	2	3*	2	4	
• History of estrogen-associated DVT/PE							
• Pregnancy-associated DVT/PE*							
• Idiopathic DVT/PE*							
• Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, or antithrombin deficiencies; or antiphospholipid syndrome)*							
• Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer*							
• History of recurrent DVT/PE*							
ii. Lower risk for recurrent DVT/PE (no risk factors)*	1	2	2	2	2	3	
d. Family history (first-degree relatives)	1	1	1	1	1	2	

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC	Clarification
Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	2	3*	2	4	Routine screening in the general population before contraceptive initiation is not recommended. If a person has current or history of DVT/PE, see recommendations for DVT/PE.* Classification of antiphospholipid syndrome includes presence of a clinical feature (e.g., thrombosis or obstetric morbidity) and persistently abnormal antiphospholipid antibody test on two or more occasions at least 12 weeks apart (*3*).*
Superficial venous disorders							
a. Varicose veins	1	1	1	1	1	1	—
b. Superficial venous thrombosis (acute or history)	1	1	1	2*	1	3	**CHC:** Superficial venous thrombosis might be associated with an increased risk for VTE. If a person has risk factors for concurrent DVT (e.g., thrombophilia or cancer) or has current or history of DVT, see recommendations for DVT/PE. Superficial venous thrombosis associated with a peripheral intravenous catheter is less likely to be associated with additional thrombosis and use of CHCs may be considered.
Valvular heart disease Complicated valvular heart disease is a condition associated with increased risk for adverse health events as a result of pregnancy (Box 3).							
a. Uncomplicated	1	1	1	1	1	2	—
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1	1	2*	1	4	
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).							
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: no limitation of activities or slight, mild limitation of activity) (*4*)							
i. <6 months	2	2	1	2*	1	4	—
ii. ≥6 months	2	2	1	2*	1	3	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: marked limitation of activity or should be at complete rest) (*4*)	2	2	2	3*	2	4	
Chronic kidney disease*	Initiation / Continuation		Initiation / Continuation				
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).							
a. Current nephrotic syndrome*	1* / 1*		2* / 2*		2*	3*	2* DRSP POP with known hyperkalemia: 4*	4*	**DRSP POP:** Persons with known hyperkalemia should not use DRSP POPs because of the risk for worsening hyperkalemia (category 4). For persons with CKD without known hyperkalemia (category 2), consider checking serum potassium level during first cycle of DRSP POPs.*

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD		LNG-IUD		Implant	DMPA		POP	CHC	Clarification
b. Hemodialysis*	1*	1*	2*	2*	2*	3*		2* DRSP POP with known hyperkalemia: 4*	4*	**DRSP POP:** Persons with known hyperkalemia should not use DRSP POPs because of the risk for worsening hyperkalemia (category 4). For persons with CKD without known hyperkalemia (category 2), consider checking serum potassium level during first cycle of DRSP POPs.*
c. Peritoneal dialysis*	2*	1*	2*	2*	2*	3*		2* DRSP POP with known hyperkalemia: 4*	4*	**DRSP POP:** Persons with known hyperkalemia should not use DRSP POPs because of the risk for worsening hyperkalemia (category 4). For persons with CKD without known hyperkalemia (category 2), consider checking serum potassium level during first cycle of DRSP POPs.*
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).						Initiation	Continuation			—
a. Positive (or unknown) antiphospholipid antibodies	1	1	2*		2*	3	3	2*	4	Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors.
b. Severe thrombocytopenia	3	2	2		2	3	2	2	2	Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Severe thrombocytopenia increases the risk for bleeding. The category should be assessed according to the severity of thrombocytopenia and its clinical manifestations. In persons with very severe thrombocytopenia who are at risk for spontaneous bleeding, consultation with a specialist and certain pretreatments might be warranted.
c. Immunosuppressive therapy	2	1	2		2	2	2	2	2	Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors.
d. None of the above	1	1	2		2	2	2	2	2	Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors.

See table footnotes on page 20.

TABLE A1. (*Continued*) Summary of changes in classifications for hormonal contraceptive methods and intrauterine devices from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC	Clarification
High risk for HIV infection	Initiation	Continuation	Initiation	Continuation					**IUD:** Many persons at high risk for HIV infection are also at risk for other STIs (see recommendations for Sexually transmitted infections in U.S. MEC and recommendations on STI screening before IUD placement in U.S. SPR [https://www.cdc.gov/contraception/hcp/usspr]) (*5*).*
	1*	1*	1*	1*	1	1	1	1	
Cirrhosis									
Decompensated cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Compensated (normal liver function)	1		1		1	1	1	1	—
b. Decompensated (impaired liver function)	1		2*		2*	3	2*	4	
Liver tumors									
Hepatocelluar adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Benign									
i. Focal nodular hyperplasia	1		2		2	2	2	2	
ii. Hepatoceullular adenoma	1		2*		2*	3	2*	4	
b. Malignant (hepatocellular carcinoma)	1		3		3	3	3	4	
Sickle cell disease	2		1		1	2/3*	1	4*	**DMPA:** The category should be assessed according to the severity of the condition and risk for thrombosis.*
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
Solid organ transplantation	Initiation	Continuation	Initiation	Continuation				—	
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. No graft failure	1*	1*	1*	1*	2	2/3*	2	2	**DMPA:** DMPA use among persons receiving long-term immunosuppressive therapy with a history of, or risk factors for, nontraumatic fractures is classified as category 3. Otherwise, DMPA use for persons with solid organ transplantation is classified as category 2.* **CHC:** Persons with transplant due to Budd-Chiari syndrome should not use CHCs because of the increased risk for thrombosis.*
b. Graft failure	2*	1*	2*	1*	2	2/3*	2	4	**DMPA:** DMPA use among persons receiving long-term immunosuppressive therapy with a history of, or risk factors for, nontraumatic fractures is classified as category 3. Otherwise, DMPA use for persons with solid organ transplantation is classified as category 2.*
Antiretrovirals used for prevention (PrEP) or treatment of HIV infection*,†									
See the following guidelines for the most up-to-date recommendations on drug-drug interactions between hormonal contraception and antiretrovirals: 1) Recommendations for the Use of Antiretroviral Drugs During Pregnancy and Interventions to Reduce Perinatal HIV Transmission in the United States (https://clinicalinfo.hiv.gov/en/guidelines/perinatal/prepregnancy-counseling-childbearing-age-overview?view=full#table-3) (*6*) and 2) Guidelines for the Use of Antiretroviral Agents in Adults and Adolescents With HIV (https://clinicalinfo.hiv.gov/en/guidelines/hiv-clinical-guidelines-adult-and-adolescent-arv/drug-interactions-overview?view=full) (*7*).								

Abbreviations: ARV = antiretroviral; BMI = body mass index; CHC = combined hormonal contraceptive; CKD = chronic kidney disease; Cu-IUD = copper intrauterine device; DMPA = depot medroxyprogesterone acetate; DRSP = drospirenone; DVT = deep venous thrombosis; IUD = intrauterine device; LNG-IUD = levonorgestrel intrauterine device; PE = pulmonary embolism; POC = progestin-only contraceptive; POP = progestin-only pill; PrEP = pre-exposure prophylaxis; SLE = systemic lupus erythematous; STI = sexually transmitted infection; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*; U.S. SPR = *U.S. Selected Practice Recommendations for Contraceptive Use*; VTE = venous thromboembolism.

* Indicates a condition for which the classification changed for one or more contraceptive methods or for which the condition description underwent a substantive modification.

† U.S. MEC recommendations for concurrent use of hormonal contraceptives or IUDs and ARVs for treatment of HIV infection also apply to use of ARVs for PrEP.

TABLE A2. Summary of changes for barrier methods from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Condom	Spermicide/Vaginal pH modulator*,†	Diaphragm/Cap (with spermicide)	Clarification
Chronic kidney disease* This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Current nephrotic syndrome*	1*	1*	1*	—
b. Hemodialysis*	1*	1*	1*	—
c. Peritoneal dialysis*	1*	1*	1*	—
Cervical cancer (awaiting treatment)	1	Vaginal pH modulator: 1* Spermicide: 2	1	The cap should not be used. Diaphragm use has no restrictions.
High risk for HIV infection	1	Vaginal pH modulator: 1* Spermicide: 4	4	—
HIV infection For persons with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	Vaginal pH modulator: 1* Spermicide: 3	3	
Antiretrovirals used for prevention (PrEP) or treatment of HIV infection*,§	1	1/3/4*	3/4	No drug interaction between ARV therapy and barrier method use is known. HIV infection is classified as category 1 for vaginal pH modulator and category 3 for spermicide and diaphragm or cap (see recommendations for HIV infection). High risk for HIV infection is classified as category 1 for vaginal pH modulator and category 4 for spermicide and diaphragm or cap (see recommendations for High risk for HIV infection).*

Abbreviations: ARV = antiretroviral; PrEP = pre-exposure prophylaxis.

* Indicates a condition for which the classification changed for one or more contraceptive methods or for which the condition description underwent a substantive modification.

† The contraceptive method "Spermicide" has been changed to "Spermicide/Vaginal pH modulator." Recommendations for "Spermicide/Vaginal pH modulator" are the same as those previously for "Spermicide," with exceptions noted.

§ *U.S. Medical Eligibility Criteria for Contraceptive Use* recommendations for concurrent use of barrier methods and ARVs for treatment of HIV infection also apply to use of ARVs for PrEP.

TABLE A3. Summary of changes for emergency contraception from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Condition	Category				Clarification
	Cu-IUD	UPA	LNG	COC	
Solid organ transplantation					
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. No graft failure	1*	1	1	1	—
b. Graft failure	2*	1	1	1	—

Abbreviations: COC = combined oral contraceptive; Cu-IUD = copper intrauterine device; LNG = levonorgestrel; UPA = ulipristal acetate.

* Indicates a condition for which the classification changed for one or more contraceptive methods or for which the condition description underwent a substantive modification.

References

1. US Department of Agriculture; US Department of Health and Human Services. Dietary guidelines for Americans, 2020–2025. 9th ed. Washington, DC: US Department of Agriculture and US Department of Health and Human Services; 2020. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf
2. Meek JY, Noble L; Section on Breastfeeding. Policy statement: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057988. PMID:35921640 https://doi.org/10.1542/peds.2022-057988
3. Barbhaiya M, Zuily S, Naden R, et al.; ACR/EULAR APS Classification Criteria Collaborators. The 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria. Arthritis Rheumatol 2023;75:1687–702. PMID:37635643 https://doi.org/10.1002/art.42624
4. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
5. Curtis KM, Nguyen AT, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2024. MMWR Recomm Rep 2024;73(No. RR-3):1–77.
6. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Recommendations for the use of antiretroviral drugs during pregnancy and interventions to reduce perinatal HIV transmission in the United States. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/en/guidelines/perinatal/recommendations-arv-drugs-pregnancy-overview
7. Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in adults and adolescents with HIV. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/sites/default/files/guidelines/documents/adult-adolescent-arv/guidelines-adult-adolescent-arv.pdf

Appendix B: Classifications for Intrauterine Devices

Classifications for intrauterine devices (IUDs) are for the copper (380 mm^2) and levonorgestrel (13.5 mg, 19.5 mg, or 52 mg) IUDs (Box B1) (Table B1). IUDs do not protect against sexually transmitted infections (STIs), including HIV infection, and patients using IUDs should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*1*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*1*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*2*).

BOX B1. Categories for classifying intrauterine devices

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE B1. Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
Personal Characteristics and Reproductive History			
Pregnancy	4	4	**Clarification:** The IUD is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion.
Age			
a. Menarche to <20 years	2	2	**Comment:** Concern exists both about the risk for expulsion from nulliparity and for STIs from sexual behavior in younger age groups (see U.S. SPR for recommendations on STI screening before IUD placement (https://www.cdc.gov/contraception/hcp/usspr) (*3*).
b. ≥20 years	1	1	—
Parity			
a. Nulliparous	2	2	**Evidence:** Data conflict about whether IUD use is associated with infertility among nulliparous women, although well-conducted studies suggest no increased risk (*4–12*).
b. Parous	1	1	—
Postpartum (including cesarean delivery, breastfeeding, or nonbreastfeeding)			
a. <10 minutes after delivery of the placenta	2	2	**Clarification:** Postpartum placement of IUDs is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*13*) or up to age 2 years or longer (*14*). **Evidence:** Studies suggest that immediate postplacental (<10 minutes) and early postpartum (10 minutes up until 72 hours) placement of Cu-IUDs and LNG-IUDs is associated with increased risk for expulsion compared with interval placement (i.e., not related to pregnancy). A meta-analysis found an increased risk for expulsion with immediate postplacental placement (8.6%; range = 0%–31.9%) and early postpartum placement (25.1%; range = 3.5%–46.7%) compared with interval placement (1.6%; range = 0%–4.8%) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Although immediate postplacental placement at the time of cesarean delivery might have increased risk for expulsion compared with interval placement, risk appears lower than that for placement at the time of vaginal delivery. Evidence for infection, perforation, and removals for pain or bleeding are limited; however, these events are rare (*15–67*). **Evidence (breastfeeding):** Two RCTs found conflicting results on breastfeeding outcomes when LNG-IUDs were initiated immediately postpartum compared with 6–8 weeks postpartum. Initiation of LNG-IUDs immediately postpartum had no other harmful effect on infant health, growth, or development (*19,68*). Breastfeeding women using IUDs do not have an increased risk for certain IUD-related adverse events including expulsion, infection, pain, or bleeding compared with nonbreastfeeding women. The risk for perforation is increased independently among breastfeeding women and among women ≤36 weeks postpartum, compared with nonpostpartum women; however, the absolute risk for perforation remains low (*15–67,69*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without risk factors for breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
b. 10 minutes after delivery of the placenta to <4 weeks	2	2	**Clarification:** Postpartum placement of IUDs is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*13*) or up to age 2 years or longer (*14*). **Evidence:** Studies suggest that immediate postplacental (<10 minutes) and early postpartum (10 minutes up until 72 hours) placement of Cu-IUDs and LNG-IUDs is associated with increased risk for expulsion compared with interval placement (i.e., not related to pregnancy). A meta-analysis found an increased risk for expulsion with immediate postplacental placement (8.6%; range = 0%–31.9%) and early postpartum placement (25.1%; range = 3.5%–46.7%) compared with interval placement (1.6%; range = 0%–4.8%) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Although immediate postplacental placement at the time of cesarean delivery might have increased risk for expulsion compared with interval placement, risk appears lower than that for placement at the time of vaginal delivery. Evidence for infection, perforation, and removals for pain or bleeding are limited; however, these events are rare (*15–67*). **Evidence (breastfeeding):** Two RCTs found conflicting results on breastfeeding outcomes when LNG-IUDs were initiated immediately postpartum compared with 6–8 weeks postpartum. Initiation of LNG-IUDs immediately postpartum had no other harmful effect on infant health, growth, or development (*19,68*). Breastfeeding women using IUDs do not have an increased risk for certain IUD-related adverse events including expulsion, infection, pain, or bleeding compared with nonbreastfeeding women. The risk for perforation is increased independently among breastfeeding women and among women ≤36 weeks postpartum, compared with nonpostpartum women; however, the absolute risk for perforation remains low (*15–67,69*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without risk factors for breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
c. ≥4 weeks	1	1	**Clarification:** Postpartum placement of IUDs is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*13*) or up to age 2 years or longer (*14*). **Evidence (breastfeeding):** Initiation of LNG-IUDs at 4 weeks postpartum or later demonstrated no detrimental effect on breastfeeding outcomes and no harmful effect on infant health, growth, or development (*19,68*). Breastfeeding women using IUDs do not have an increased risk for certain IUD-related adverse events including expulsion, infection, pain, or bleeding compared with nonbreastfeeding women. The risk for perforation is increased independently among breastfeeding women and among women ≤36 weeks postpartum, compared with nonpostpartum women; however, the absolute risk for perforation remains low (*15–67,69*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without risk factors for breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
d. Postpartum sepsis	4	4	**Comment:** Theoretical concern exists that postpartum placement of an IUD in a person with recent chorioamnionitis or current endometritis might be associated with increased complications.

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
Postabortion (spontaneous or induced)			
a. First trimester abortion			**Clarification:** IUDs may be placed immediately after abortion completion. **Evidence:** Risk for complications from immediate versus delayed placement of an IUD after abortion did not differ. Expulsion was greater when an IUD was placed after a second trimester procedural abortion than when placed after a first trimester procedural abortion. Safety or expulsion for postabortion placement of an LNG-IUD did not differ from that of a Cu-IUD (*70*).
i. Procedural (surgical)	1	1	
ii. Medication	1	1	
iii. Spontaneous abortion with no intervention	1	1	
b. Second trimester abortion			
i. Procedural (surgical)	2	2	
ii. Medication	2	2	
iii. Spontaneous abortion with no intervention	2	2	
c. Immediate postseptic abortion	4	4	**Comment:** Placement of an IUD might substantially worsen the condition.
Past ectopic pregnancy	1	1	**Comment:** The absolute risk for ectopic pregnancy is extremely low because of the high effectiveness of IUDs. However, when a person becomes pregnant during IUD use, the relative likelihood of ectopic pregnancy increases substantially.
History of pelvic surgery (see recommendations for Postpartum [including cesarean delivery])	1	1	—
Smoking			
a. Age <35 years	1	1	—
b. Age ≥35 years			
i. <15 cigarettes per day	1	1	—
ii. ≥15 cigarettes per day	1	1	—
Obesity			
a. BMI ≥30 kg/m^2	1	1	—
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	—
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	—
Surgery			
a. Minor surgery without immobilization	1	1	—
b. Major surgery			
i. Without prolonged immobilization	1	1	—
ii. With prolonged immobilization	1	1	**Evidence:** No direct evidence was identified on risk for thrombosis with POC use among those undergoing major surgery (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Cardiovascular Disease			
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	2	—

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
Hypertension			
Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Adequately controlled hypertension	1	1	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive.
b. Elevated blood pressure levels (properly taken measurements)			**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive.
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	1	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1	2	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
c. Vascular disease	1	2	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive. **Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	—
Deep venous thrombosis/ Pulmonary embolism			
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Current or history of DVT/PE, receiving anticoagulant therapy (therapeutic dose) (e.g., acute DVT/PE or long-term therapeutic dose)	2	2	**Clarification (Cu-IUD):** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. Cu-IUDs might worsen bleeding. **Clarification (LNG-IUD):** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. LNG-IUDs can be of benefit in preventing or treating this complication. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis. **Evidence:** Limited evidence was identified on use of POCs or Cu-IUDs among women with acute DVT/PE receiving anticoagulant therapy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). In one study among women with a history of acute VTE currently receiving therapeutic anticoagulant therapy (i.e., rivaroxaban or enoxaparin/vitamin K antagonist [warfarin or acenocoumarol]), the incidence of recurrent VTE was similar among estrogen users (CHC or estrogen-only pills), POC users, and women not on hormonal therapy (*71*). Limited evidence suggests that placement of a Cu-IUD or LNG-IUD does not increase risk for bleeding complications in women receiving anticoagulant therapy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
b. History of DVT/PE, receiving anticoagulant therapy (prophylactic dose)			**Clarification (Cu-IUD):** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. Cu-IUDs might worsen bleeding.
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	2	**Clarification (LNG-IUD):** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding. LNG-IUDs can be of benefit in preventing or treating this complication. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis. **Evidence:** Limited evidence suggests that placement of the LNG-IUD does not increase risk for bleeding complications in women receiving anticoagulant therapy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	
c. History of DVT/PE, not receiving anticoagulant therapy			
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	1	2	—
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	2	—
d. Family history (first-degree relatives)	1	1	—
Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	**Clarification:** Routine screening in the general population before contraceptive initiation is not recommended. **Clarification:** If a person has current or history of DVT/PE, see recommendations for DVT/PE. **Clarification**: Classification of antiphospholipid syndrome includes presence of a clinical feature (e.g., thrombosis or obstetric morbidity) and persistently abnormal antiphospholipid antibody test on two or more occasions at least 12 weeks apart (*72*). **Evidence:** Limited evidence was identified on LNG-IUD use among persons with thrombophilia. Among women with factor V Leiden mutation, one study found that women using LNG-IUD had similar risk for venous thrombosis as those not using hormonal contraception (*73*). No evidence was identified on POC use among persons with prothrombin gene mutation, protein S deficiency, protein C deficiency, antithrombin deficiency, or antiphospholipid syndrome (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Superficial venous disorders			
a. Varicose veins	1	1	—
b. Superficial venous thrombosis (acute or history)	1	1	—

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category: Cu-IUD	Category: LNG-IUD		Clarification/Evidence/Comment
		Initiation	Continuation	
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	3	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2		**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				**Comment:** According to the American Heart Association, administration of prophylactic antibiotics solely to prevent endocarditis is not recommended for patients who undergo genitourinary tract procedures, including placement or removal of IUDs (*74*).
a. Uncomplicated	1	1		
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1		
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				**Evidence:** No direct evidence exists on the safety of IUDs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies did not demonstrate any cases of arrhythmia or infective endocarditis in women with cardiac disease who used IUDs (*75*). **Comment:** IUD placement might induce cardiac arrhythmias in healthy persons; persons with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: no limitation of activities or slight, mild limitation of activity) (*76*)				
i. <6 months	2	2		
ii. ≥6 months	2	2		
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: marked limitation of activity or should be at complete rest) (*76*)	2	2		
Renal Disease				
Chronic kidney disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation / Continuation	Initiation	Continuation	
a. Current nephrotic syndrome	1 / 1	2	2	**Comment:** A person might have CKD without current nephrotic syndrome, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, SLE). See recommendations for other conditions if they apply.
b. Hemodialysis	1 / 1	2	2	**Evidence:** No comparative studies were identified on the safety of IUD use among persons with CKD on hemodialysis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). One case report of LNG-IUD use in a person with CKD on hemodialysis reported improved abnormal uterine bleeding and anemia (*77*). **Comment:** A person might have CKD without hemodialysis, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, and SLE). See recommendations for other conditions if they apply.
c. Peritoneal dialysis	2 / 1	2	2	**Evidence:** No comparative studies were identified on IUD use among persons with CKD on peritoneal dialysis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Four case reports of IUD use among women with CKD on peritoneal dialysis identified one case of peritoneal allergic reaction (78), three cases of peritonitis (*78–80*) and one case of TOA (*78*). **Comment:** A person might have CKD without peritoneal dialysis, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, and SLE). See recommendations for other conditions if they apply.

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category				Clarification/Evidence/Comment
	Cu-IUD		LNG-IUD		
Rheumatic Diseases					
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation			—
a. Positive (or unknown) antiphospholipid antibodies	1	1	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*81–99*). **Evidence:** No direct evidence was identified on POC use among persons with SLE with antiphospholipid antibodies (*100*) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Severe thrombocytopenia	3	2	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*81–99*). **Clarification:** Severe thrombocytopenia increases the risk for bleeding. The category should be assessed according to the severity of thrombocytopenia and its clinical manifestations. In persons with very severe thrombocytopenia who are at risk for spontaneous bleeding, consultation with a specialist and certain pretreatments might be warranted. **Evidence:** The LNG-IUD might be a useful treatment for menorrhagia in women with severe thrombocytopenia (*94*).
c. Immunosuppressive therapy	2	1	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*81–99*).
d. None of the above	1	1	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*81–99*).
Rheumatoid arthritis	Initiation	Continuation	Initiation	Continuation	—
a. Not receiving immunosuppressive therapy	1	1	1	1	—
b. Receiving immunosuppressive therapy	2	1	2	1	—
Neurologic Conditions					
Headaches					
a. Nonmigraine (mild or severe)		1		1	—
b. Migraine					
i. Without aura (includes menstrual migraine)		1		1	**Evidence:** No studies directly examined the risk for stroke among women with migraine using LNG-IUDs (*101*). Limited evidence demonstrated that women using LNG-IUDs do not have an increased risk for ischemic stroke compared with women not using hormonal contraceptives (*102*). **Comment:** Menstrual migraine is a subtype of migraine without aura. For more information see the International Headache Society's *International Classification of Headache Disorders, 3rd ed.* (https://ichd-3.org) (*103*).
ii. With aura		1		1	
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		1		1	—
Multiple sclerosis					
a. Without prolonged immobility		1		1	—
b. With prolonged immobility		1		1	—
Depressive Disorders					
Depressive disorders		1		1	**Clarification:** If a person is receiving psychotropic medications or St. John's wort, see recommendations for Drug Interactions. **Evidence:** The frequency of psychiatric hospitalizations for women with bipolar disorder or depression did not significantly differ among women using DMPA, LNG-IUD, Cu-IUD, or sterilization (*104*).

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category: Cu-IUD		Category: LNG-IUD		Clarification/Evidence/Comment
Reproductive Tract Infections and Disorders					
Vaginal bleeding patterns			Initiation	Continuation	—
a. Irregular pattern without heavy bleeding	1		1	1	
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2		1	2	**Clarification:** Unusually heavy bleeding should raise suspicion of a serious underlying condition. **Evidence:** Evidence from studies examining the treatment effects of the LNG-IUD among women with heavy or prolonged bleeding reported no increase in adverse effects and found the LNG-IUD to be beneficial in treating menorrhagia (*105–112*).
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	Initiation 4	Continuation 2	Initiation 4	Continuation 2	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. The IUD does not need to be removed before evaluation.
Endometriosis	2		1		**Evidence:** LNG-IUD use among women with endometriosis decreased dysmenorrhea, pelvic pain, and dyspareunia (*113–117*).
Benign ovarian tumors (including cysts)	1		1		—
Severe dysmenorrhea	2		1		**Comment:** Dysmenorrhea might intensify with Cu-IUD use. LNG-IUD use has been associated with reduction of dysmenorrhea.
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. Suspected gestational trophoblastic disease (immediate postevacuation)					**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.
i. Uterine size first trimester	1		1		**Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*118*).
ii. Uterine size second trimester	2		2		**Comment:** The risk for expulsion immediately postevacuation for gestational trophoblastic disease is unknown. Expulsion is greater after IUD placement immediately postevacuation for a spontaneous or induced abortion in the second trimester compared with IUD placement after a first trimester abortion.
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)	Initiation	Continuation	Initiation	Continuation	—
i. Undetectable or nonpregnant β-hCG levels	1	1	1	1	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*118*). **Comment:** Once β-hCG levels have decreased to nonpregnant levels, the risk for disease progression is likely to be very low.
ii. Decreasing β-hCG levels	2	1	2	1	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Clarification:** For persons at higher risk for disease progression, the benefits of effective contraception must be weighed against the potential need for early IUD removal. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*118*).
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	2	1	2	1	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*118*).
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	4	2	4	2	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*118*). **Comment:** For persons with suspected or confirmed intrauterine disease, an IUD should not be placed because of theoretical risk for perforation, infection, and hemorrhage. For persons who already have an IUD in place, individual circumstance along with the benefits of effective contraception must be weighed against theoretical risks of either removal or continuation of the IUD.

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category				Clarification/Evidence/Comment
	Cu-IUD		LNG-IUD		
Cervical ectropion	1		1		—
Cervical intraepithelial neoplasia	1		2		**Comment:** Theoretical concern exists that LNG-IUDs might enhance progression of cervical intraepithelial neoplasia.
Cervical cancer (awaiting treatment)	Initiation 4	Continuation 2	Initiation 4	Continuation 2	**Comment:** Concern exists about the increased risk for infection and bleeding at placement. The IUD most likely will need to be removed at the time of treatment but until then, the person is at risk for pregnancy.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. Undiagnosed mass	1		2		**Clarification (LNG-IUD):** Evaluation of mass should be pursued as early as possible.
b. Benign breast disease	1		1		—
c. Family history of cancer	1		1		—
d. Breast cancer					**Comment:** Breast cancer is a hormonally sensitive tumor. Concerns about progression of the disease might be less with LNG-IUDs than with COCs or higher-dose POCs.
i. Current	1		4		
ii. Past and no evidence of current disease for 5 years	1		3		
Endometrial hyperplasia	1		1		**Evidence:** Among women with endometrial hyperplasia, no adverse health events occurred with LNG-IUD use; most women experienced disease regression (*119*).
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation 4	Continuation 2	Initiation 4	Continuation 2	**Comment:** Concern exists about the increased risk for infection, perforation, and bleeding at placement. The IUD most likely will need to be removed at the time of treatment, but until then, the person is at risk for pregnancy.
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1		1		**Comment:** Persons with ovarian cancer who undergo fertility-sparing treatment and need contraception can use an IUD.
Uterine fibroids	2		2		**Evidence:** Among women with uterine fibroids using an LNG-IUD, most experienced improvements in serum levels of hemoglobin, hematocrit, and ferritin and in menstrual blood loss (*120*). Rates of LNG-IUD expulsion were higher in women with uterine fibroids (11%) than in women without fibroids (0%–3%); these findings were either not statistically significant or significance testing was not conducted (*120*). Rates of expulsion found in noncomparative studies ranged from 0%–20% (*120*). **Comment:** Persons with heavy or prolonged bleeding should be assigned the category for that condition.
Anatomical abnormalities					
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD placement)	4		4		**Comment:** An anatomical abnormality that distorts the uterine cavity might preclude proper IUD placement.
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD placement	2		2		—
Pelvic inflammatory disease	Initiation	Continuation	Initiation	Continuation	
a. Current PID	4	2	4	2	**Clarification (continuation):** Treat the PID using appropriate antibiotics. The IUD usually does not need to be removed if the person wants to continue using it. Continued use of an IUD depends on the person's informed choice and current risk factors for STIs and PID. **Evidence:** Among IUD users treated for PID, clinical course did not differ regardless of whether the IUD was removed or left in place (*121*).
b. Past PID					**Comment:** IUDs do not protect against STIs, including HIV infection, or PID. In persons at low risk for STIs, IUD placement poses little risk for PID.
i. With subsequent pregnancy	1	1	1	1	
ii. Without subsequent pregnancy	2	2	2	2	
Sexually transmitted infections	Initiation	Continuation	Initiation	Continuation	
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2	4	2	**Clarification (continuation):** Treat the STI using appropriate antibiotics. The IUD usually does not need to be removed if the person wants to continue using it. Continued use of an IUD depends on the person's informed choice and current risk factors for STIs and PID. **Evidence:** Among women who had an IUD placed, the absolute risk for subsequent PID was low among women with STI at the time of placement but greater than among women with no STI at the time of IUD placement (*122–128*).

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category: Cu-IUD		LNG-IUD		Clarification/Evidence/Comment
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	—
c. Other factors related to STIs	2	2	2	2	**Clarification (initiation):** Most persons do not require additional STI screening at the time of IUD placement. If a person with risk factors for STIs has not been screened for gonorrhea and chlamydia according to CDC STI treatment guidelines (*1*), screening may be performed at the time of IUD placement and placement should not be delayed. **Evidence:** Women who undergo same-day STI screening and IUD placement have low incidence rates of PID. Algorithms for predicting PID among women with risk factors for STIs have poor predictive value. Risk for PID among women with risk factors for STIs is low (*129*).
HIV					
	Initiation	Continuation	Initiation	Continuation	**Clarification:** Many persons at high risk for HIV infection are also at risk for other STIs (see recommendations for Sexually transmitted infections in U.S. MEC and recommendations on STI screening before IUD placement in U.S. SPR (https://www.cdc.gov/contraception/hcp/usspr) (*3*). **Evidence:** High-quality evidence from one RCT, along with low-quality evidence from two observational studies, suggested no increased risk for HIV acquisition with Cu-IUD use. No studies were identified for LNG-IUDs (*130–132*).
High risk for HIV infection	1	1	1	1	
HIV infection For persons with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					**Evidence:** Among IUD users, limited evidence demonstrates a low risk for PID among HIV-infected women using IUDs and no higher risk for pelvic infectious complications in HIV-infected than in HIV-noninfected women or among women with varying degrees of HIV severity. IUD use did not adversely affect progression of HIV infection during 6–45 months of follow-up or when compared with hormonal contraceptive use among HIV-infected women. Furthermore, IUD use among HIV-infected women was not associated with increased risk for transmission to sex partners or with increased genital viral shedding (*133*).
a. Clinically well receiving ARV therapy	1	1	1	1	
b. Not clinically well or not receiving ARV therapy	2	1	2	1	
Other Infections					
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. Uncomplicated		1		1	—
b. Fibrosis of the liver (if severe, see recommendations for Cirrhosis)		1		1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation	Initiation	Continuation	
a. Nonpelvic	1	1	1	1	—
b. Pelvic	4	3	4	3	**Comment:** Placement of an IUD might substantially worsen the condition.
Malaria		1		1	—
Endocrine Conditions					
Diabetes Insulin-dependent diabetes diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. History of gestational disease		1		1	—
b. Nonvascular disease					**Evidence:** Limited evidence on the use of the LNG-IUD among women with insulin-dependent or non–insulin-dependent diabetes suggests that these methods have little effect on short-term or long-term diabetes control (e.g., glycosylated hemoglobin levels), hemostatic markers, or lipid profile (*134,135*).
i. Non-insulin dependent		1		2	
ii. Insulin dependent		1		2	
c. Nephropathy, retinopathy, or neuropathy		1		2	—
d. Other vascular disease or diabetes of >20 years' duration		1		2	—

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
Thyroid disorders			
a. Simple goiter	1	1	—
b. Hyperthyroid	1	1	—
c. Hypothyroid	1	1	—
Gastrointestinal Conditions			
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	1	**Evidence:** Although two case reports described three women with IBD who experienced exacerbation of disease 5 days–25 months after LNG-IUD placement (*136*), no comparative studies have examined the safety of IUD use among women with IBD (*136*).
Gallbladder disease			
a. Asymptomatic	1	2	—
b. Symptomatic			
i. Current	1	2	—
ii. Treated by cholecystectomy	1	2	—
iii. Medically treated	1	2	—
History of cholestasis			
a. Pregnancy related	1	1	—
b. Past COC related	1	2	**Comment:** Concern exists that history of COC related cholestasis might predict subsequent cholestasis with LNG use. Whether risk exists with use of LNG-IUD is unclear.
Viral hepatitis			
a. Acute or flare	1	1	**Evidence:** No direct evidence was identified on IUD use among persons with viral hepatitis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Chronic	1	1	**Evidence:** No direct evidence was identified on IUD use among persons with viral hepatitis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Cirrhosis Decompensated cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Compensated (normal liver function)	1	1	**Evidence:** No direct evidence was identified on IUD use among persons with cirrhosis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Decompensated (impaired liver function)	1	2	**Evidence:** No direct evidence was identified on IUD use among persons with cirrhosis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Hepatic metabolism of exogenous hormones might be impaired in persons with liver dysfunction, which could lead to increased progestin levels in circulation and progestin-related side effects and adverse events, which might vary by dose and formulation. Any progestin-related hepatotoxicity might be less tolerated in persons with existing liver dysfunction.
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Benign			
i. Focal nodular hyperplasia	1	2	**Evidence:** Limited evidence suggests that progestin use does not influence either progression or regression of focal nodular hyperplasia (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
ii. Hepatocellular adenoma	1	2	**Evidence:** Limited evidence suggests that hepatocellular adenomas generally regress or remain stable during progestin use (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Malignant (hepatocellular carcinoma)	1	3	**Evidence:** No direct evidence was identified on IUD use among persons with hepatocellular carcinoma (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Respiratory Conditions			
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	**Clarification:** Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for persons with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions.
Hematologic Conditions			
Thalassemia	2	1	**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	2	1	**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category				Clarification/Evidence/Comment
	Cu-IUD		LNG-IUD		
Iron deficiency anemia	2		1		**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Solid Organ Transplantation					
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation	Initiation	Continuation	**Evidence:** Limited evidence suggests that LNG-IUD use among solid organ transplantation recipients does not increase risk for pelvic infections or decrease contraceptive effectiveness over time or compared with persons without solid organ transplantation No evidence was identified for Cu-IUD (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
a. No graft failure	1	1	1	1	
b. Graft failure	2	1	2	1	
Drug Interactions					
Antiretrovirals used for prevention (PrEP) or treatment of HIV	Initiation	Continuation	Initiation	Continuation	**Clarification:** No known interaction exists between ARV therapy and IUD use. However, for persons with HIV infection, IUD placement is classified as category 2 if the person is not clinically well or not receiving ARV therapy. Otherwise, both placement and continuation are classified as category 1 (see recommendations for HIV infection). For persons at high risk for HIV infection, IUDs are category 1 for initiation and continuation (see recommendations for High risk for HIV infection).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)					
i. Abacavir (ABC)	1/2	1	1/2	1	
ii. Tenofovir (TDF)	1/2	1	1/2	1	
iii. Zidovudine (AZT)	1/2	1	1/2	1	
iv. Lamivudine (3TC)	1/2	1	1/2	1	
v. Didanosine (DDI)	1/2	1	1/2	1	
vi. Emtricitabine (FTC)	1/2	1	1/2	1	
vii. Stavudine (D4T)	1/2	1	1/2	1	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)					
i. Efavirenz (EFV)	1/2	1	1/2	1	
ii. Etravirine (ETR)	1/2	1	1/2	1	
iii. Nevirapine (NVP)	1/2	1	1/2	1	
iv. Rilpivirine (RPV)	1/2	1	1/2	1	
c. Ritonavir-boosted protease inhibitors					
i. Ritonavir-boosted atazanavir (ATV/r)	1/2	1	1/2	1	
ii. Ritonavir-boosted darunavir (DRV/r)	1/2	1	1/2	1	
iii. Ritonavir-boosted fosamprenavir (FPV/r)	1/2	1	1/2	1	
iv. Ritonavir-boosted lopinavir (LPV/r)	1/2	1	1/2	1	
v. Ritonavir-boosted saquinavir (SQV/r)	1/2	1	1/2	1	
vi. Ritonavir-boosted tipranavir (TPV/r)	1/2	1	1/2	1	
d. Protease inhibitors without ritonavir					
i. Atazanavir (ATV)	1/2	1	1/2	1	
ii. Fosamprenavir (FPV)	1/2	1	1/2	1	
iii. Indinavir (IDV)	1/2	1	1/2	1	
iv. Nelfinavir (NFV)	1/2	1	1/2	1	
e. CCR5 co-receptor antagonists					
i. Maraviroc (MVC)	1/2	1	1/2	1	
f. HIV integrase strand transfer inhibitors					
i. Raltegravir (RAL)	1/2	1	1/2	1	
ii. Dolutegravir (DTG)	1/2	1	1/2	1	
iii. Elvitegravir (EVG)	1/2	1	1/2	1	
g. Fusion inhibitors					
i. Enfuvirtide	1/2	1	1/2	1	

See table footnotes on page 36.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper intrauterine device and levonorgestrel intrauterine device

Condition	Category		Clarification/Evidence/Comment
	Cu-IUD	LNG-IUD	
Anticonvulsant therapy			
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	1	1	**Evidence:** Limited evidence suggests use of certain anticonvulsants does not interfere with the contraceptive effectiveness of the LNG-IUD (*137,138*).
b. Lamotrigine	1	1	**Evidence:** No drug interactions have been reported among women with epilepsy who are receiving lamotrigine and using the LNG-IUD (*138,139*).
Antimicrobial therapy			
a. Broad-spectrum antibiotics	1	1	—
b. Antifungals	1	1	—
c. Antiparasitics	1	1	—
d. Rifampin or rifabutin therapy	1	1	**Evidence:** One cross-sectional survey found that rifabutin had no impact on the effectiveness of the LNG-IUD (*137*).
Psychotropic medications			
a. Selective serotonin reuptake inhibitors (SSRIs)	1	1	**Comment:** For many common psychotropic agents, limited or no theoretical concern exists for clinically significant drug interactions when co-administered with hormonal contraceptives. However, either no or very limited data exist examining potential interactions for these classes of medications
St. John's wort	1	1	—

Abbreviations: ARV = antiretroviral; BMI = body mass index; CHC = combined hormonal contraceptive; CKD = chronic kidney disease; COC = combined oral contraceptive; Cu-IUD = copper intrauterine device; DMPA = depot medroxyprogesterone acetate; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; IBD = inflammatory bowel disease; IUD = intrauterine device; LDL = low-density lipoprotein; LNG = levonorgestrel; LNG-IUD = levonorgestrel intrauterine device; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; POC = progestin-only contraceptive; PrEP = pre-exposure prophylaxis; RCT = randomized clinical trial; SLE = systemic lupus erythematous; STI = sexually transmitted infection; TOA = tubo-ovarian abscess; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*; U.S. SPR = *U.S. Selected Practice Recommendations for Contraceptive Use*; VTE = venous thromboembolism.

References

1. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
2. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
3. Curtis KM, Nguyen AT, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2024. MMWR Recomm Rep 2024;73(No. RR-3):1–77.
4. Cramer DW, Schiff I, Schoenbaum SC, et al. Tubal infertility and the intrauterine device. N Engl J Med 1985;312:941–7. PMID:3974684 https://doi.org/10.1056/NEJM198504113121502
5. Daling JR, Weiss NS, Metch BJ, et al. Primary tubal infertility in relation to the use of an intrauterine device. N Engl J Med 1985;312:937–41. PMID:3974683 https://doi.org/10.1056/NEJM198504113121501
6. Daling JR, Weiss NS, Voigt LF, McKnight B, Moore DE. The intrauterine device and primary tubal infertility. N Engl J Med 1992;326:203–4. PMID:1727559 https://doi.org/10.1056/NEJM199201163260314
7. Delbarge W, Bátár I, Bafort M, et al. Return to fertility in nulliparous and parous women after removal of the GyneFix intrauterine contraceptive system. Eur J Contracept Reprod Health Care 2002;7:24–30. PMID:12041861 https://doi.org/10.1080/ejc.7.1.24.30
8. Doll H, Vessey M, Painter R. Return of fertility in nulliparous women after discontinuation of the intrauterine device: comparison with women discontinuing other methods of contraception. BJOG 2001;108:304–14. PMID:11281473 https://doi.org/10.1111/j.1471-0528.2001.00075.x
9. Hubacher D, Lara-Ricalde R, Taylor DJ, Guerra-Infante F, Guzmán-Rodríguez R. Use of copper intrauterine devices and the risk of tubal infertility among nulligravid women. N Engl J Med 2001;345:561–7. PMID:11529209 https://doi.org/10.1056/NEJMoa010438
10. Skjeldestad FE, Bratt H. Return of fertility after use of IUDs (Nova-T, MLCu250 and MLCu375). Adv Contracept 1987;3:139–45. PMID:3630823 https://doi.org/10.1007/BF01890702
11. Urbach DR, Marrett LD, Kung R, Cohen MM. Association of perforation of the appendix with female tubal infertility. Am J Epidemiol 2001;153:566–71. PMID:11257064 https://doi.org/10.1093/aje/153.6.566
12. Wilson JC. A prospective New Zealand study of fertility after removal of copper intrauterine contraceptive devices for conception and because of complications: a four-year study. Am J Obstet Gynecol 1989;160:391–6. PMID:2916624 https://doi.org/10.1016/0002-9378(89)90455-9
13. US Department of Agriculture; US Department of Health and Human Services. Dietary guidelines for Americans, 2020–2025. 9th ed. Washington, DC: US Department of Agriculture and US Department of Health and Human Services; 2020. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf
14. Meek JY, Noble L; Section on Breastfeeding. Policy statement: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057988. PMID:35921640 https://doi.org/10.1542/peds.2022-057988
15. Annus J, Brat T, Diethelm MP, et al.; World Health Organization Special. Comparative multicentre trial of three IUDs inserted immediately following delivery of the placenta. Contraception 1980;22:9–18. PMID:7418410 https://doi.org/10.1016/0010-7824(80)90112-2
16. Apelo RA, Waszak CS. Postpartum IUD insertions in Manila, Philippines. Adv Contracept 1985;1:319–28. PMID:3842222 https://doi.org/10.1007/BF01849307
17. Baldwin MK, Edelman AB, Lim JY, Nichols MD, Bednarek PH, Jensen JT. Intrauterine device placement at 3 versus 6 weeks postpartum: a randomized trial. Contraception 2016;93:356–63. PMID:26686914 https://doi.org/10.1016/j.contraception.2015.12.006

18. Bonilla Rosales F, Aguilar Zamudio ME, Cázares Montero ML, Hernández Ortiz ME, Luna Ruiz MA. [Factors for expulsion of intrauterine device Tcu380A applied immediately postpartum and after a delayed period]. Rev Med Inst Mex Seguro Soc 2005;43:5–10. PMID:15998475
19. Braniff K, Gomez E, Muller R. A randomised clinical trial to assess satisfaction with the levonorgestrel-releasing intrauterine system inserted at caesarean section compared to postpartum placement. Aust N Z J Obstet Gynaecol 2015;55:279–83. PMID:26053465 https://doi.org/10.1111/ajo.12335
20. Bryant AG, Kamanga G, Stuart GS, Haddad LB, Meguid T, Mhango C. Immediate postpartum versus 6-week postpartum intrauterine device insertion: a feasibility study of a randomized controlled trial. Afr J Reprod Health 2013;17:72–9. PMID:24069753
21. Caliskan E, Oztürk N, Dilbaz BO, Dilbaz S. Analysis of risk factors associated with uterine perforation by intrauterine devices. Eur J Contracept Reprod Health Care 2003;8:150–5. PMID:14667326 https://doi.org/10.1080/ejc.8.3.150.155
22. Celen S, Möröy P, Sucak A, Aktulay A, Danişman N. Clinical outcomes of early postplacental insertion of intrauterine contraceptive devices. Contraception 2004;69:279–82. PMID:15033401 https://doi.org/10.1016/j.contraception.2003.12.004
23. Çelen Ş, Sucak A, Yıldız Y, Danışman N. Immediate postplacental insertion of an intrauterine contraceptive device during cesarean section. Contraception 2011;84:240–3. PMID:21843687 https://doi.org/10.1016/j.contraception.2011.01.006
24. Chen BA, Reeves MF, Hayes JL, Hohmann HL, Perriera LK, Creinin MD. Postplacental or delayed insertion of the levonorgestrel intrauterine device after vaginal delivery: a randomized controlled trial. Obstet Gynecol 2010;116:1079–87. PMID:20966692 https://doi.org/10.1097/AOG.0b013e3181f73fac
25. Chen JH, Wu SC, Shao WQ, et al. The comparative trial of TCu 380A IUD and progesterone-releasing vaginal ring used by lactating women. Contraception 1998;57:371–9. PMID:9693396 https://doi.org/10.1016/S0010-7824(98)00043-2
26. Cohen R, Sheeder J, Arango N, Teal SB, Tocce K. Twelve-month contraceptive continuation and repeat pregnancy among young mothers choosing postdelivery contraceptive implants or postplacental intrauterine devices. Contraception 2016;93:178–83. PMID:26475368 https://doi.org/10.1016/j.contraception.2015.10.001
27. Dahlke JD, Terpstra ER, Ramseyer AM, Busch JM, Rieg T, Magann EF. Postpartum insertion of levonorgestrel—intrauterine system at three time periods: a prospective randomized pilot study. Contraception 2011;84:244–8. PMID:21843688 https://doi.org/10.1016/j.contraception.2011.01.007
28. Dias T, Abeykoon S, Kumarasiri S, Gunawardena C, Padeniya T, D'Antonio F. Use of ultrasound in predicting success of intrauterine contraceptive device insertion immediately after delivery. Ultrasound Obstet Gynecol 2015;46:104–8. PMID:25418016 https://doi.org/10.1002/uog.14733
29. El-Shafei MMMA, Hassan EO, El-Boghdad L, El-Lakkany N. Postpartum and postabortion intrauterine device insertion unmet needs of safe reproductive health: three years experience of Mansoura University Hospital. J Egypt 2000;26:253–62.
30. Elsedeek MS. Puerperal and menstrual bleeding patterns with different types of contraceptive device fitted during elective cesarean delivery. Int J Gynaecol Obstet 2012;116:31–4. PMID:22036512 https://doi.org/10.1016/j.ijgo.2011.07.036
31. Elsedeek MS. Five-year follow-up of two types of contraceptive device fitted during elective cesarean delivery. Int J Gynaecol Obstet 2015;130:179–82. PMID:25957802 https://doi.org/10.1016/j.ijgo.2015.02.031
32. Eroğlu K, Akkuzu G, Vural G, et al. Comparison of efficacy and complications of IUD insertion in immediate postplacental/early postpartum period with interval period: 1 year follow-up. Contraception 2006;74:376–81. PMID:17046378 https://doi.org/10.1016/j.contraception.2006.07.003
33. Gueye M, Gaye YF, Diouf AA, et al. [Trancesarean intra-uterine device. Pilot study performed at Dakar teaching hospital]. J Gynecol Obstet Biol Reprod (Paris) 2013;42:585–90. PMID:23850420 https://doi.org/10.1016/j.jgyn.2013.06.003
34. Gupta S, Malik S, Sinha R, Shyamsunder S, Mittal MK. Association of the position of the Copper T 380A as determined by the ultrasonography following its insertion in the immediate postpartum period with the subsequent complications: an observational study. J Obstet Gynaecol India 2014;64:349–53. PMID:25368459 https://doi.org/10.1007/s13224-014-0532-5
35. Hagbard L, Ingemanson CA, Sorbe B. Early postpartum insertion of copper IUD. Contraception 1978;17:355–63. PMID:648157 https://doi.org/10.1016/0010-7824(78)90081-1
36. Hayes JL, Cwiak C, Goedken P, Zieman M. A pilot clinical trial of ultrasound-guided postplacental insertion of a levonorgestrel intrauterine device. Contraception 2007;76:292–6. PMID:17900440 https://doi.org/10.1016/j.contraception.2007.06.003
37. Jatlaoui TC, Marcus M, Jamieson DJ, Goedken P, Cwiak C. Postplacental intrauterine device insertion at a teaching hospital. Contraception 2014;89:528–33. PMID:24565735 https://doi.org/10.1016/j.contraception.2013.10.008
38. Jatlaoui TC, Whiteman MK, Jeng G, et al. Intrauterine device expulsion after postpartum placement: a systematic review and meta-analysis. Obstet Gynecol 2018;132:895–905. PMID:30204688 https://doi.org/10.1097/AOG.0000000000002822
39. Kumar S, Sethi R, Balasubramaniam S, et al. Women's experience with postpartum intrauterine contraceptive device use in India. Reprod Health 2014;11:32. PMID:24755312 https://doi.org/10.1186/1742-4755-11-32
40. Laes E, Lehtovirta P, Weintraub D, Pyörälä T, Luukkainen T. Early puerperal insertions of copper-T-200. Contraception 1975;11:289–95. PMID:1116368 https://doi.org/10.1016/0010-7824(75)90037-2
41. Lara Ricalde R, Menocal Tobías G, Ramos Pérez C, Velázquez Ramírez N. [Random comparative study between intrauterine device Multiload Cu375 and TCu 380a inserted in the postpartum period]. Ginecol Obstet Mex 2006;74:306–11. PMID:16970116
42. Lavin P, Bravo C, Waszak C. Comparison of T Cu 200 and Progestasert IUDs. Contracept Deliv Syst 1983;4:143–7. PMID:12338635
43. Lavin P, Waszak C, Bravo C. Preliminary report on a postpartum CuT 200 study, Santiago, Chile. Int J Gynaecol Obstet 1983;21:71–5. PMID:6133798 https://doi.org/10.1016/0020-7292(83)90073-5
44. Lester F, Kakaire O, Byamugisha J, et al. Intracesarean insertion of the Copper T380A versus 6 weeks postcesarean: a randomized clinical trial. Contraception 2015;91:198–203. PMID:25499587 https://doi.org/10.1016/j.contraception.2014.12.002
45. Letti Müller AL, Lopes Ramos JG, Martins-Costa SH, et al. Transvaginal ultrasonographic assessment of the expulsion rate of intrauterine devices inserted in the immediate postpartum period: a pilot study. Contraception 2005;72:192–5. PMID:16102554 https://doi.org/10.1016/j.contraception.2005.03.014
46. Levi E, Cantillo E, Ades V, Banks E, Murthy A. Immediate postplacental IUD insertion at cesarean delivery: a prospective cohort study. Contraception 2012;86:102–5. PMID:22264666 https://doi.org/10.1016/j.contraception.2011.11.019
47. Levi EE, Stuart GS, Zerden ML, Garrett JM, Bryant AG. Intrauterine device placement during cesarean delivery and continued use 6 months postpartum. Obstet Gynecol 2015;126:5–11. PMID:26241250 https://doi.org/10.1097/AOG.0000000000000882

48. Mishra S. Evaluation of safety, efficacy, and expulsion of post-placental and intra-cesarean insertion of intrauterine contraceptive devices (PPIUCD). J Obstet Gynaecol India 2014;64:337–43. PMID:25368457 https://doi.org/10.1007/s13224-014-0550-3
49. Morrison C, Waszak C, Katz K, Diabaté F, Mate EM. Clinical outcomes of two early postpartum IUD insertion programs in Africa. Contraception 1996;53:17–21. PMID:8631184 https://doi.org/10.1016/0010-7824(95)00254-5
50. Nelson AL, Chen S, Eden R. Intraoperative placement of the Copper T-380 intrauterine devices in women undergoing elective cesarean delivery: a pilot study. Contraception 2009;80:81–3. PMID:19501220 https://doi.org/10.1016/j.contraception.2009.01.014
51. Newton J, Harper M, Chan KK. Immediate post-placental insertion of intrauterine contraceptive devices. Lancet 1977;2:272–4. PMID:69881 https://doi.org/10.1016/S0140-6736(77)90955-2
52. Prema K, Gayathri TL, Philips FS. Comparative study of early postpartum, postabortal and interval insertion of Cu T 200 mm2 device. J Obstet Gynaecol India 1978;28:946–8. PMID:571822
53. Puzey M. Mirena at caesarean section. Eur J Contracept Reprod Health Care 2005;10:164–7. PMID:16318963 https://doi.org/10.1080/13625180500233851
54. Ragab A, Hamed HO, Alsammani MA, et al. Expulsion of Nova-T380, Multiload 375, and Copper-T380A contraceptive devices inserted during cesarean delivery. Int J Gynaecol Obstet 2015;130:174–8. PMID:25975871 https://doi.org/10.1016/j.ijgo.2015.03.025
55. Shukla M, Qureshi S; Chandrawati. Post-placental intrauterine device insertion—a five year experience at a tertiary care centre in north India. Indian J Med Res 2012;136:432–5. PMID:23041736
56. Singal S, Bharti R, Dewan R, et al. Clinical outcome of postplacental Copper T 380A insertion in women delivering by caesarean section. J Clin Diagn Res 2014;8:OC01–04. PMID:25386484
57. Stuart GS, Bryant AG, O'Neill E, Doherty IA. Feasibility of postpartum placement of the levonorgestrel intrauterine system more than 6 h after vaginal birth. Contraception 2012;85:359–62. PMID:22067759 https://doi.org/10.1016/j.contraception.2011.08.005
58. Stuart GS, Lesko CR, Stuebe AM, Bryant AG, Levi EE, Danvers AI. A randomized trial of levonorgestrel intrauterine system insertion 6 to 48 h compared to 6 weeks after vaginal delivery; lessons learned. Contraception 2015;91:284–8. PMID:25553871 https://doi.org/10.1016/j.contraception.2014.12.009
59. Thiery M, Van Kets H, Van der Pas H. Immediate post-placental IUD insertion: the expulsion problem. Contraception 1985;31:331–49. PMID:4006467 https://doi.org/10.1016/0010-7824(85)90002-2
60. Van Der Pas MT, Delbeke L, Van Dets H. Comparative performance of two copper-wired IUDs (ML Cu 250 and T Cu 200: immediate postpartum and interval insertion. Contracept Deliv Syst 1980;1:27–35. PMID:12261715
61. Welkovic S, Costa LO, Faúndes A, de Alencar Ximenes R, Costa CF. Post-partum bleeding and infection after post-placental IUD insertion. Contraception 2001;63:155–8. PMID:11368989 https://doi.org/10.1016/S0010-7824(01)00180-9
62. Whitaker AK, Endres LK, Mistretta SQ, Gilliam ML. Postplacental insertion of the levonorgestrel intrauterine device after cesarean delivery vs. delayed insertion: a randomized controlled trial. Contraception 2014;89:534–9. PMID:24457061 https://doi.org/10.1016/j.contraception.2013.12.007
63. Woo CJ, Alamgir H, Potter JE. Women's experiences after Planned Parenthood's exclusion from a family planning program in Texas. Contraception 2016;93:298–302. PMID:26680757 https://doi.org/10.1016/j.contraception.2015.12.004
64. Wu SC; Research Group on Failure Causes and Prevention Measures of Intrauterine Device. [Efficacy of intrauterine device TCu380A when inserted in four different periods]. Zhonghua Fu Chan Ke Za Zhi 2009;44:431–5. PMID:19953943
65. Xu J, Yang X, Gu X, et al. Comparison between two techniques used in immediate postplacental insertion of TCu 380A intrauterine device: 36-month follow-up. Reprod Contracept 1999;10:156–62. PMID:12349462
66. Xu J, Zhuang L, Yu G. [Comparison of two techniques used in immediate postplacental insertion of TCu 380A intrauterine device: 12 month follow-up of 910 cases]. Zhonghua Fu Chan Ke Za Zhi 1997;32:354–7. PMID:9596916
67. Xu JX, Rivera R, Dunson TR, et al. A comparative study of two techniques used in immediate postplacental insertion (IPPI) of the Copper T-380A IUD in Shanghai, People's Republic of China. Contraception 1996;54:33–8. PMID:8804806 https://doi.org/10.1016/0010-7824(96)00117-5
68. Phillips SJ, Tepper NK, Kapp N, Nanda K, Temmerman M, Curtis KM. Progestogen-only contraceptive use among breastfeeding women: a systematic review. Contraception 2016;94:226–52. PMID:26410174 https://doi.org/10.1016/j.contraception.2015.09.010
69. Berry-Bibee EN, Tepper NK, Jatlaoui TC, Whiteman MK, Jamieson DJ, Curtis KM. The safety of intrauterine devices in breastfeeding women: a systematic review. Contraception 2016;94:725–38. PMID:27421765 https://doi.org/10.1016/j.contraception.2016.07.006
70. Steenland MW, Tepper NK, Curtis KM, Kapp N. Intrauterine contraceptive insertion postabortion: a systematic review. Contraception 2011;84:447–64. PMID:22018119 https://doi.org/10.1016/j.contraception.2011.03.007
71. Martinelli I, Lensing AW, Middeldorp S, et al. Recurrent venous thromboembolism and abnormal uterine bleeding with anticoagulant and hormone therapy use. Blood 2016;127:1417–25. PMID:26696010 https://doi.org/10.1182/blood-2015-08-665927
72. Barbhaiya M, Zuily S, Naden R, et al.; ACR/EULAR APS Classification Criteria Collaborators. The 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria. Arthritis Rheumatol 2023;75:1687–702. PMID:37635643 https://doi.org/10.1002/art.42624
73. Bergendal A, Persson I, Odeberg J, et al. Association of venous thromboembolism with hormonal contraception and thrombophilic genotypes. Obstet Gynecol 2014;124:600–9. PMID:25162263 https://doi.org/10.1097/AOG.0000000000000411
74. Wilson W, Taubert KA, Gewitz M, et al.; American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee; American Heart Association Council on Cardiovascular Disease in the Young; American Heart Association Council on Clinical Cardiology; American Heart Association Council on Cardiovascular Surgery and Anesthesia; Quality of Care and Outcomes Research Interdisciplinary Working Group. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation 2007;116:1736–54. PMID:17446442 https://doi.org/10.1161/CIRCULATIONAHA.106.183095
75. Tepper NK, Paulen ME, Marchbanks PA, Curtis KM. Safety of contraceptive use among women with peripartum cardiomyopathy: a systematic review. Contraception 2010;82:95–101. PMID:20682147 https://doi.org/10.1016/j.contraception.2010.02.004
76. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
77. Fedele L, Gammaro L, Bianchi S. Levonorgestrel-releasing intrauterine device for the treatment of menometrorrhagia in a woman on hemodialysis. N Engl J Med 1999;341:541. PMID:10447446 https://doi.org/10.1056/NEJM199908123410718

78. Chen Y, Li Z, Mo L, et al. Eosinophilia in peritoneal effluent due to a levonorgestrel-releasing intrauterine system in a woman on peritoneal dialysis. Perit Dial Int 2017;37:349–50. PMID:28512169 https://doi.org/10.3747/pdi.2016.00154
79. Korzets A, Chagnac A, Ori Y, Zevin D, Levi J. Pneumococcal peritonitis complicating CAPD—was the indwelling intrauterine device to blame? Clin Nephrol 1991;35:24–5. PMID:2007293
80. Plaza MM. Intrauterine device-related peritonitis in a patient on CAPD. Perit Dial Int 2002;22:538–9. PMID:12322833 https://doi.org/10.1177/089686080202200420
81. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81. PMID:15226516 https://doi.org/10.1093/rheumatology/keh282
82. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9. PMID:15280571 https://doi.org/10.1093/rheumatology/keh331
83. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8. PMID:12180730
84. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7. PMID:11665973 https://doi.org/10.1002/1529-0131(200110)44:10<2331::AID-ART395>3.0.CO;2-I
85. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30. PMID:8448613 https://doi.org/10.1093/rheumatology/32.3.227
86. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15. PMID:9048514 https://doi.org/10.1093/oxfordjournals.aje.a009122
87. McAlindon T, Giannotta L, Taub N, D'Cruz D, Hughes G. Environmental factors predicting nephritis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4. PMID:8257208 https://doi.org/10.1136/ard.52.10.720
88. McDonald J, Stewart J, Urowitz MB, Gladman DD. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60. PMID:1540039 https://doi.org/10.1136/ard.51.1.56
89. Mintz G, Gutiérrez G, Delezé M, Rodríguez E. Contraception with progestagens in systemic lupus erythematosus. Contraception 1984;30:29–38. PMID:6434228 https://doi.org/10.1016/0010-7824(84)90076-3
90. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45. PMID:7654797 https://doi.org/10.1002/art.1790080305
91. Petri M. Lupus in Baltimore: evidence-based 'clinical pearls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3. PMID:16425579 https://doi.org/10.1191/0961203305lu2230xx
92. Sánchez-Guerrero J, Uribe AG, Jiménez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49. PMID:16354890 https://doi.org/10.1056/NEJMoa050817
93. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12. PMID:16082635 https://doi.org/10.1002/art.21314
94. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3. PMID:16202726 https://doi.org/10.1016/j.ajog.2005.05.002
95. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6. PMID:12465147
96. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5. PMID:1251849 https://doi.org/10.1016/0002-9343(76)90431-9
97. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33. PMID:1771400 https://doi.org/10.3109/03009749109096822
98. Jungers P, Dougados M, Pélissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23. PMID:7092961 https://doi.org/10.1002/art.1780250603
99. Petri M, Kim MY, Kalunian KC, et al.; OC-SELENA Trial. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8. PMID:16354891 https://doi.org/10.1056/NEJMoa051135
100. Culwell KR, Curtis KM, Del Carmen Cravioto M. Safety of contraceptive method use among women with systemic lupus erythematosus: a systematic review. Obstet Gynecol 2009;114:341–53. PMID:19622996 https://doi.org/10.1097/AOG.0b013e3181ae9c64
101. Tepper NK, Whiteman MK, Zapata LB, Marchbanks PA, Curtis KM. Safety of hormonal contraceptives among women with migraine: asystematic review. Contraception 2016;94:630–40. PMID:27153744 https://doi.org/10.1016/j.contraception.2016.04.016
102. Tepper NK, Whiteman MK, Marchbanks PA, James AH, Curtis KM. Progestin-only contraception and thromboembolism: asystematic review. Contraception 2016;94:678–700. PMID:27153743 https://doi.org/10.1016/j.contraception.2016.04.014
103. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders. 3rd ed. Cephalalgia 2018;38:1–211. https://www.ichd-3.org/wp-content/uploads/2018/01/The-International-Classification-of-Headache-Disorders-3rd-Edition-2018.pdf
104. Pagano HP, Zapata LB, Berry-Bibee EN, Nanda K, Curtis KM. Safety of hormonal contraception and intrauterine devices among women with depressive and bipolar disorders: a systematic review. Contraception 2016;94:641–9. PMID:27364100 https://doi.org/10.1016/j.contraception.2016.06.012
105. Barrington JW, Arunkalaivanan AS, Abdel-Fattah M. Comparison between the levonorgestrel intrauterine system (LNG-IUS) and thermal balloon ablation in the treatment of menorrhagia. Eur J Obstet Gynecol Reprod Biol 2003;108:72–4. PMID:12694974 https://doi.org/10.1016/S0301-2115(02)00408-6
106. Gupta B, Mittal S, Misra R, Deka D, Dadhwal V. Levonorgestrel-releasing intrauterine system vs. transcervical endometrial resection for dysfunctional uterine bleeding. Int J Gynaecol Obstet 2006;95:261–6. PMID:16999960 https://doi.org/10.1016/j.ijgo.2006.07.004
107. Hurskainen R, Teperi J, Rissanen P, et al. Quality of life and cost-effectiveness of levonorgestrel-releasing intrauterine system versus hysterectomy for treatment of menorrhagia: a randomised trial. [see comment]. Lancet 2001;357:273–7. PMID:11214131 https://doi.org/10.1016/S0140-6736(00)03615-1
108. Istre O, Trolle B. Treatment of menorrhagia with the levonorgestrel intrauterine system versus endometrial resection. Fertil Steril 2001;76:304–9. PMID:11476777 https://doi.org/10.1016/S0015-0282(01)01909-4
109. Koh SC, Singh K. The effect of levonorgestrel-releasing intrauterine system use on menstrual blood loss and the hemostatic, fibrinolytic/inhibitor systems in women with menorrhagia. J Thromb Haemost 2007;5:133–8. PMID:17010149 https://doi.org/10.1111/j.1538-7836.2006.02243.x

110. Lethaby AE, Cooke I, Rees M. Progesterone/progestogen releasing intrauterine systems versus either placebo or any other medication for heavy menstrual bleeding. Cochrane Database Syst Rev 2000;(2):CD002126. PMID:10796865
111. Magalhães J, Aldrighi JM, de Lima GR. Uterine volume and menstrual patterns in users of the levonorgestrel-releasing intrauterine system with idiopathic menorrhagia or menorrhagia due to leiomyomas. Contraception 2007;75:193–8. PMID:17303488 https://doi.org/10.1016/j.contraception.2006.11.004
112. Stewart A, Cummins C, Gold L, Jordan R, Phillips W. The effectiveness of the levonorgestrel-releasing intrauterine system in menorrhagia: a systematic review. BJOG 2001;108:74–86. PMID:11213008 https://doi.org/10.1111/j.1471-0528.2001.00020.x
113. Fedele L, Bianchi S, Zanconato G, Portuese A, Raffaelli R. Use of a levonorgestrel-releasing intrauterine device in the treatment of rectovaginal endometriosis. Fertil Steril 2001;75:485–8. PMID:11239528 https://doi.org/10.1016/S0015-0282(00)01759-3
114. Lockhat FBE, Emembolu J, Konje JC. The effect of a levonorgestrel intrauterine system (LNG-IUS) on symptomatic endometriosis. Fertil Steril 2002;77(Suppl 1):S24. https://doi.org/10.1016/S0015-0282(01)03086-2
115. Petta CA, Ferriani RA, Abrao MS, et al. Randomized clinical trial of a levonorgestrel-releasing intrauterine system and a depot GnRH analogue for the treatment of chronic pelvic pain in women with endometriosis. Hum Reprod 2005;20:1993–8. PMID:15790607 https://doi.org/10.1093/humrep/deh869
116. Vercellini P, Aimi G, Panazza S, De Giorgi O, Pesole A, Crosignani PG. A levonorgestrel-releasing intrauterine system for the treatment of dysmenorrhea associated with endometriosis: a pilot study. Fertil Steril 1999;72:505–8. PMID:10519624 https://doi.org/10.1016/S0015-0282(99)00291-5
117. Vercellini P, Frontino G, De Giorgi O, Aimi G, Zaina B, Crosignani PG. Comparison of a levonorgestrel-releasing intrauterine device versus expectant management after conservative surgery for symptomatic endometriosis: a pilot study. Fertil Steril 2003;80:305–9. PMID:12909492 https://doi.org/10.1016/S0015-0282(03)00608-3
118. Gaffield ME, Kapp N, Curtis KM. Combined oral contraceptive and intrauterine device use among women with gestational trophoblastic disease. Contraception 2009;80:363–71. PMID:19751859 https://doi.org/10.1016/j.contraception.2009.03.022
119. Whiteman MK, Zapata LB, Tepper NK, Marchbanks PA, Curtis KM. Use of contraceptive methods among women with endometrial hyperplasia: a systematic review. Contraception 2010;82:56–63. PMID:20682143 https://doi.org/10.1016/j.contraception.2010.02.005
120. Zapata LB, Whiteman MK, Tepper NK, Jamieson DJ, Marchbanks PA, Curtis KM. Intrauterine device use among women with uterine fibroids: a systematic review. Contraception 2010;82:41–55. PMID:20682142 https://doi.org/10.1016/j.contraception.2010.02.011
121. Tepper NK, Steenland MW, Gaffield ME, Marchbanks PA, Curtis KM. Retention of intrauterine devices in women who acquire pelvic inflammatory disease: a systematic review. Contraception 2013;87:655–60. PMID:23040135 https://doi.org/10.1016/j.contraception.2012.08.011
122. Faúndes A, Telles E, Cristofoletti ML, Faúndes D, Castro S, Hardy E. The risk of inadvertent intrauterine device insertion in women carriers of endocervical Chlamydia trachomatis. Contraception 1998;58:105–9. PMID:9773265 https://doi.org/10.1016/S0010-7824(98)00064-X
123. Ferraz do Lago R, Simões JA, Bahamondes L, Camargo RP, Perrotti M, Monteiro I. Follow-up of users of intrauterine device with and without bacterial vaginosis and other cervicovaginal infections. Contraception 2003;68:105–9. PMID:12954522 https://doi.org/10.1016/S0010-7824(03)00109-4
124. Morrison CS, Sekadde-Kigondu C, Miller WC, Weiner DH, Sinei SK. Use of sexually transmitted disease risk assessment algorithms for selection of intrauterine device candidates. Contraception 1999;59:97–106. PMID:10361624 https://doi.org/10.1016/S0010-7824(99)00006-2
125. Pap-Akeson M, Solheim F, Thorbert G, Akerlund M. Genital tract infections associated with the intrauterine contraceptive device can be reduced by inserting the threads into the uterine cavity. Br J Obstet Gynaecol 1992;99:676–9. PMID:1390474 https://doi.org/10.1111/j.1471-0528.1992.tb13854.x
126. Sinei SK, Schulz KFLP, Lamptey PR, et al. Preventing IUCD-related pelvic infection: the efficacy of prophylactic doxycycline at insertion. Br J Obstet Gynaecol 1990;97:412–9. PMID:2196934 https://doi.org/10.1111/j.1471-0528.1990.tb01828.x
127. Skjeldestad FE, Halvorsen LE, Kahn H, Nordbø SA, Saake K. IUD users in Norway are at low risk for genital C. trachomatis infection. Contraception 1996;54:209–12. PMID:8922873 https://doi.org/10.1016/S0010-7824(96)00190-4
128. Walsh TL, Bernstein GS, Grimes DA, Frezieres R, Bernstein L, Coulson AH; IUD Study Group. Effect of prophylactic antibiotics on morbidity associated with IUD insertion: results of a pilot randomized controlled trial. Contraception 1994;50:319–27. PMID:7813220 https://doi.org/10.1016/0010-7824(94)90019-1
129. Jatlaoui TC, Simmons KB, Curtis KM. The safety of intrauterine contraception initiation among women with current asymptomatic cervical infections or at increased risk of sexually transmitted infections. Contraception 2016;94:701–12. PMID:27263041 https://doi.org/10.1016/j.contraception.2016.05.013
130. Curtis KM, Hannaford PC, Rodriguez MI, Chipato T, Steyn PS, Kiarie JN. Hormonal contraception and HIV acquisition among women: an updated systematic review. BMJ Sex Reprod Health 2020;46:8–16. PMID:31919239 https://doi.org/10.1136/bmjsrh-2019-200509
131. Hannaford PC, Ti A, Chipato T, Curtis KM. Copper intrauterine device use and HIV acquisition in women: a systematic review. BMJ Sex Reprod Health 2020;46:17–25. PMID:31919240 https://doi.org/10.1136/bmjsrh-2019-200512
132. Tepper NK, Curtis KM, Cox S, Whiteman MK. Update to U.S. medical eligibility criteria for contraceptive use, 2016: updated recommendations for the use of contraception among women at high risk for HIV infection. MMWR Morb Mortal Wkly Rep 2020;69:405–10. PMID:32271729 https://doi.org/10.15585/mmwr.mm6914a3
133. Tepper NK, Curtis KM, Nanda K, Jamieson DJ. Safety of intrauterine devices among women with HIV: a systematic review. Contraception 2016;94:713–24. PMID:27343750 https://doi.org/10.1016/j.contraception.2016.06.011
134. Grigoryan OR, Grodnitskaya EE, Andreeva EN, Shestakova MV, Melnichenko GA, Dedov II. Contraception in perimenopausal women with diabetes mellitus. Gynecol Endocrinol 2006;22:198–206. PMID:16723306 https://doi.org/10.1080/09513590600624317
135. Rogovskaya S, Rivera R, Grimes DA, et al. Effect of a levonorgestrel intrauterine system on women with type 1 diabetes: a randomized trial. Obstet Gynecol 2005;105:811–5. PMID:15802410 https://doi.org/10.1097/01.AOG.0000156301.11939.56
136. Zapata LB, Paulen ME, Cansino C, Marchbanks PA, Curtis KM. Contraceptive use among women with inflammatory bowel disease: a systematic review. Contraception 2010;82:72–85. PMID:20682145 https://doi.org/10.1016/j.contraception.2010.02.012
137. Bounds W, Guillebaud J. Observational series on women using the contraceptive Mirena concurrently with anti-epileptic and other enzyme-inducing drugs. J Fam Plann Reprod Health Care 2002;28:78–80. PMID:12396777 https://doi.org/10.1783/147118902101195992
138. Gaffield ME, Culwell KR, Lee CR. The use of hormonal contraception among women taking anticonvulsant therapy. Contraception 2011;83:16–29. PMID:21134499 https://doi.org/10.1016/j.contraception.2010.06.013
139. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7. PMID:16146436 https://doi.org/10.1111/j.1528-1167.2005.10105.x

Appendix C: Classifications for Progestin-Only Contraceptives

Classifications for progestin-only contraceptives (POCs) include those for progestin-only implants (68 mg etonogestrel), progestin-only injectables (depot medroxyprogesterone acetate [DMPA], 150 mg intramuscular [DMPA-IM] or 104 mg subcutaneous [DMPA-SC]), and progestin-only pills (POPs) (containing norethindrone, norgestrel, or drospirenone [DRSP]) (Box C1) (Table C1). DMPA-SC can be administered by a health care provider or through self-administration. Recommendations in this report and *U.S. Selected Practice Recommendations for Contraceptive Use, 2024* (*1*) for provider-administered DMPA (IM or SC) also apply to self-administered DMPA-SC. POCs do not protect against sexually transmitted infections (STIs), including HIV infection, and patients using POCs should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*2*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*2*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*3*).

BOX C1. Categories for classifying progestin-only contraceptives

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE C1. Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Personal Characteristics and Reproductive History				
Pregnancy	NA	NA	NA	**Clarification:** Use of POCs is not required. No known harm to the patient, the course of pregnancy, or the fetus occurs if POCs are inadvertently used during pregnancy. However, the relation between DMPA use during pregnancy and its effects on the fetus remains unclear.
Age				**Evidence:** Most studies have found that women lose BMD during DMPA use but recover BMD after discontinuation (*4*). Limited evidence demonstrates a weak association with fracture. However, one large study suggests that women who choose DMPA might be at higher risk for fracture before initiation (*5*). It is unclear whether adult women with long durations of DMPA use can regain BMD to baseline levels before entering menopause and whether adolescents can reach peak bone mass after discontinuation of DMPA. The relation between these changes in BMD during the reproductive years and future fracture risk is unknown. Studies generally find no effect of POCs other than DMPA on BMD (*4*–*52*).
a. Menarche to <18 years	1	2	1	
b. 18–45 years	1	1	1	
c. >45 years	1	2	1	
Parity				
a. Nulliparous	1	1	1	—
b. Parous	1	1	1	—
Breastfeeding				
a. <21 days postpartum	2	2	2	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*53*) or up to age 2 years or longer (*54*). **Evidence (breastfeeding):** Two small, RCTs found no adverse impact on breastfeeding with initiation of etonogestrel implants within 48 hours postpartum. Other studies found that initiation of POPs, injectables, and implants at ≤6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*55*,*56*). **Evidence:** Limited evidence suggests that DMPA use might further elevate risk for VTE among postpartum women compared with non-use (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
b. 21 to <30 days postpartum				
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	2	2	2	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*53*) or up to age 2 years or longer (*54*). **Evidence (breastfeeding):** Two small, RCTs found no adverse impact on breastfeeding with initiation of etonogestrel implants within 48 hours postpartum. Other studies found that initiation of POPs, injectables, and implants at ≤6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*55*,*56*). **Evidence:** Limited evidence suggests that DMPA use might further elevate risk for VTE among postpartum women compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
ii. Without other risk factors for VTE	2	2	2	
c. 30–42 days postpartum				
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	1	2	1	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*53*) or up to age 2 years or longer (*54*). **Evidence (breastfeeding):** Two small, RCTs found no adverse impact on breastfeeding with initiation of etonogestrel implants within 48 hours postpartum. Other studies found that initiation of POPs, injectables, and implants at ≤6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*55*,*56*). **Evidence**: Limited evidence suggests that DMPA use might further elevate risk for VTE among postpartum women compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
ii. Without other risk factors for VTE	1	1	1	

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
d. >42 days postpartum	1	1	1	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*53*) or up to age 2 years or longer (*54*). **Evidence:** Overall, studies found that initiation of POPs, injectables, and implants at >6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*56*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
Postpartum (nonbreastfeeding)				
a. <21 days postpartum	1	2	1	**Evidence:** Limited evidence suggests that DMPA use might further elevate risk for VTE among postpartum women compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. 21–42 days postpartum				
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	1	2	1	**Evidence:** Limited evidence suggests that DMPA use might further elevate risk for VTE among postpartum women compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
ii. Without other risk factors for VTE	1	1	1	—
c. >42 days postpartum	1	1	1	—

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Postabortion (spontaneous or induced)				
a. First trimester abortion				**Clarification:** POCs may be started immediately after abortion completion or at time of medication abortion initiation.
i. Procedural (surgical)	1	1	1	
ii. Medication	1	1/2	1	
iii. Spontaneous abortion with no intervention	1	1	1	**Clarification (DMPA):** After a first trimester medication abortion that did not include mifepristone, there is no restriction for the use of DMPA (category 1). After a first trimester medication abortion that included mifepristone, there is no restriction for use of DMPA after abortion completion (category 1) and benefits generally outweigh risks with DMPA use immediately at time of medication abortion initiation (category 2). Concurrent administration of DMPA with mifepristone might slightly decrease medication abortion effectiveness and increase risk for ongoing pregnancy. Risk for ongoing pregnancy with concurrent administration of DMPA with mifepristone should be considered along with personal preference and access to follow-up abortion and contraceptive care. **Evidence:** Limited evidence suggests decreased first trimester medication abortion effectiveness with concurrent administration of DMPA with mifepristone (immediate) versus DMPA administration after abortion completion (delayed). In one study, the risk for ongoing pregnancy, while overall low, was higher with immediate (3.6%) versus delayed (0.9%) DMPA administration (difference 2.7%; 90% CI = 0.4–5.6%) (*57*). This difference was not seen with other progestin-only methods (*58*). Evidence suggests that there is no increased risk for adverse events when POCs are initiated after first trimester procedural or medication abortion (immediately or delayed) (*58*) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Second trimester abortion				**Clarification:** POCs may be started immediately after abortion completion or at time of medication abortion initiation.
i. Procedural (surgical)	1	1	1	
ii. Medication	1	1	1	
iii. Spontaneous abortion with no intervention	1	1	1	
c. Immediate postseptic abortion	1	1	1	**Clarification:** POCs may be started immediately after abortion completion or at time of medication abortion initiation.
Past ectopic pregnancy	1	1	2	**Comment:** POP users have a higher absolute rate of ectopic pregnancy than do users of other POCs but still lower than those using no method.
History of pelvic surgery	1	1	1	—
Smoking				
a. Age <35 years	1	1	1	—
b. Age ≥35 years				
i. <15 cigarettes per day	1	1	1	—
ii. ≥15 cigarettes per day	1	1	1	—
Obesity				
a. BMI ≥30 kg/m^2	1	1	1	—
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	2	1	**Evidence:** Among adult women, generally no association has been found between baseline weight and weight gain among DMPA users compared with nonusers. Evidence is mixed for adolescent DMPA users, with certain studies observing greater weight gain among users with obesity compared with those without obesity but other studies demonstrating no association; methodologic differences across studies might account for the differences in findings. Data on other POC methods and other adverse outcomes including weight gain are limited (*59*–*76*).

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent laparoscopic placement of an adjustable gastric band (*77*).
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	3	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent a biliopancreatic diversion; however, evidence from pharmacokinetic studies suggested conflicting results regarding oral contraceptive effectiveness among women who underwent a jejunoileal bypass (*77*). **Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications such as long-term diarrhea, vomiting, or both.
Surgery				
a. Minor surgery without immobilization	1	1	1	—
b. Major surgery				
i. Without prolonged immobilization	1	1	1	—
ii. With prolonged immobilization	1	2	1	**Evidence:** No direct evidence was identified on risk for thrombosis with POC use among those undergoing major surgery. Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with prolonged immobilization after major surgery.
Cardiovascular Disease				
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	2	3	2	**Clarification:** When multiple major risk factors exist, risk for cardiovascular disease might increase substantially. Certain POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs. The effects of DMPA might persist for some time after discontinuation. **Clarification:** The recommendations apply to known pre-existing medical conditions or characteristics. Few if any screening tests are needed before initiation of contraception. See U.S. SPR (https://www.cdc.gov/contraception/hcp/usspr) (*1*).

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Adequately controlled hypertension	1	2	1	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive. **Clarification:** Persons adequately treated for hypertension are at lower risk for acute myocardial infarction and stroke than are untreated persons. Although no data exist, POC users with adequately controlled and monitored hypertension should be at lower risk for acute myocardial infarction and stroke than are untreated hypertensive POC users.
b. Elevated blood pressure levels (properly taken measurements)				**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive. **Evidence:** Limited evidence suggests that among women with hypertension, those who used POPs or progestin-only injectables had a small increased risk for cardiovascular events compared with women who did not use these methods (*78*).
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	2	1	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	2	3	2	
c. Vascular disease	2	3	2	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive. **Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. However, little concern exists about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	—
Deep venous thrombosis/ Pulmonary embolism This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
a. Current or history of DVT/PE, receiving anticoagulant therapy (therapeutic dose) (e.g., acute DVT/PE or long-term therapeutic dose)	2	2	2	**Clarification**: Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. POCs can be of benefit in preventing or treating these complications; benefits might vary by POC dose and formulation. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis. **Evidence**: Limited evidence was identified on use of POCs among women with acute DVT/PE receiving anticoagulant therapy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). In one study among women with a history of acute VTE currently receiving therapeutic anticoagulant therapy (i.e., rivaroxaban or enoxaparin/vitamin K antagonist [warfarin or acenocoumarol]), the incidence of recurrent VTE was similar among estrogen users (CHC or estrogen-only pills), POC users, and women not on hormonal therapy (*79*). Limited evidence suggests that intramuscular injections of DMPA in women receiving chronic anticoagulation therapy do not pose a significant risk for hematoma at the injection site or increase the risk for heavy or irregular vaginal bleeding (*80*).
b. History of DVT/PE, receiving anticoagulant therapy (prophylactic dose)				**Clarification:** Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. POCs can be of benefit in preventing or treating these complications; benefits might vary by POC dose and formulation. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis. **Evidence**: Limited evidence was identified on use of POCs among women with acute DVT/PE receiving anticoagulant therapy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with a history of DVT/PE and at higher risk for recurrent DVT/PE. Limited evidence suggests that intramuscular injections of DMPA in women receiving chronic anticoagulation therapy do not pose a significant risk for hematoma at the injection site or increase the risk for heavy or irregular vaginal bleeding (*80*).
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	3	2	
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	2	
c. History of DVT/PE, not receiving anticoagulant therapy				

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	3	2	**Evidence:** Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with a history of DVT/PE and at higher risk for recurrent DVT/PE.
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	2	—
d. Family history (first-degree relatives)	1	1	1	—
Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	2	3	2	**Clarification:** Routine screening in the general population before contraceptive initiation is not recommended. **Clarification**: If a person has current or history of DVT/PE, see recommendations for DVT/PE. **Clarification**: Classification of antiphospholipid syndrome includes presence of a clinical feature (e.g., thrombosis or obstetric morbidity) and persistently abnormal antiphospholipid antibody test on two or more occasions at least 12 weeks apart (*81*). **Evidence:** Among women with factor V Leiden mutation, one study found that women using POCs had an increased risk for venous thrombosis compared with non-users without the mutation, with the highest relative risk for DMPA users (*82*). Women with prothrombin gene mutation using POCs did not have an increased risk for venous thrombosis compared with nonusers without the mutation (*82*). No evidence was identified on POC use among persons with protein S deficiency, protein C deficiency, antithrombin deficiency, or antiphospholipid syndrome (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category					Clarification/Evidence/Comment
	Implant		DMPA	POP		
Superficial venous disorders						
a. Varicose veins	1		1	1		—
b. Superficial venous thrombosis (acute or history)	1		2	1		**Evidence:** No direct evidence was identified on risk for thrombosis with POC use among persons with superficial venous thrombosis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with superficial venous thrombosis are at higher risk for venous thrombosis than the general population (*83*). Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with non-use (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with acute or history of superficial venous thrombosis.
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation		Initiation	Continuation	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. However, little concern exists about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
	2	3	3	2	3	
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation		Initiation	Continuation	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. However, little concern exists about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
	2	3	3	2	3	
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 3).						
a. Uncomplicated	1		1	1		—
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1		2	1		**Evidence:** No direct evidence was identified on risk for thrombosis with POC use among persons with valvular heart disease (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with complicated valvular heart disease.
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).						**Evidence:** No direct evidence was identified on the safety of POC use among persons with peripartum cardiomyopathy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension, thromboembolism, and heart failure in women with cardiac disease using POPs and DMPA (*84*). Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with peripartum cardiomyopathy. **Comment:** Progestin-only implants might induce cardiac arrhythmias in healthy persons; persons with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: no limitation of activities or slight, mild limitation of activity) (*85*)						
i. <6 months	1		2	1		
ii. ≥6 months	1		2	1		
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: marked limitation of activity or should be at complete rest) (*85*)	2		3	2		

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Renal Disease				
Chronic kidney disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Current nephrotic syndrome	2	3	2 DRSP POP with known hyperkalemia: 4	**Clarification (DRSP POP):** Persons with known hyperkalemia should not use DRSP POPs because of the risk for worsening hyperkalemia (category 4). For persons with CKD without known hyperkalemia (category 2), consider checking serum potassium level during first cycle of DRSP POPs. **Evidence:** No direct evidence was identified on POC use among persons with CKD with current nephrotic syndrome (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with severe CKD or nephrotic syndrome are at higher risk for thrombosis than the general population (*86–90*). Use of DMPA, which has been associated with increased risk for thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among those with CKD with current nephrotic syndrome. Persons with severe CKD have a higher prevalence of fracture than the general population (*91–93*). Use of DMPA, which has been associated with small changes in bone mineral density (*4*) might further elevate risk for fracture among persons with CKD with current nephrotic syndrome. **Comment:** A person might have CKD without current nephrotic syndrome, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, SLE). See recommendations for other conditions if they apply.
b. Hemodialysis	2	3	2 DRSP POP with known hyperkalemia: 4	**Clarification (DRSP POP):** Persons with known hyperkalemia should not use DRSP POPs because of the risk for worsening hyperkalemia (category 4). For persons with CKD without known hyperkalemia (category 2), consider checking serum potassium level during first cycle of DRSP POPs. **Evidence:** No direct evidence was identified on POC use among persons with CKD on hemodialysis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with CKD on dialysis are at higher risk for thrombosis than the general population (*94–96*). Use of DMPA, which has been associated with increased risk for thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among those with CKD on dialysis. Persons with CKD on dialysis have a higher prevalence of fracture than the general population (*97–99*). Use of DMPA, which has been associated with small changes in bone mineral density (*4*), might further elevate risk for fracture among persons with CKD on dialysis. **Comment:** A person might have CKD without hemodialysis, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, SLE). See recommendations for other conditions if they apply.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category				Clarification/Evidence/Comment
	Implant	DMPA		POP	
c. Peritoneal dialysis	2	3		2 DRSP POP with known hyperkalemia: 4	**Clarification (DRSP POP):** Persons with known hyperkalemia should not use DRSP POPs because of the risk for worsening hyperkalemia (category 4). For persons with CKD without known hyperkalemia (category 2), consider checking serum potassium level during first cycle of DRSP POPs. **Evidence:** No direct evidence was identified on POC use among persons with CKD on peritoneal dialysis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with CKD on dialysis are at higher risk for thrombosis than the general population (*94–96*). Use of DMPA, which has been associated with increased risk for thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among those with CKD on dialysis. Persons with CKD on dialysis have a higher prevalence of fracture than the general population (*97–99*). Use of DMPA, which has been associated with small changes in bone mineral density (*4*), might further elevate risk for fracture among persons with CKD on dialysis. **Comment:** A person might have CKD without peritoneal dialysis but might have other conditions often associated with CKD (e.g., diabetes, hypertension, and SLE). See recommendations for other conditions if they apply.
Rheumatic Diseases					
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		Initiation	Continuation		—
a. Positive (or unknown) antiphospholipid antibodies	2	3	3	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*100–118*). **Evidence:** No direct evidence was identified on POC use among persons with SLE with antiphospholipid antibodies (*119*) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with SLE with antiphospholipid antibodies are at higher risk for thrombosis than the general population (*120,121*). Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with SLE with antiphospholipid antibodies.
b. Severe thrombocytopenia	2	3	2	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*100–118*). **Comment:** Severe thrombocytopenia increases the risk for bleeding. POCs might be useful in treating heavy or prolonged bleeding in persons with severe thrombocytopenia. However, given the increased or erratic bleeding that might be seen on initiation of DMPA and its irreversibility for 11–13 weeks after administration, initiation of this method in persons with severe thrombocytopenia should be done with caution.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
c. Immunosuppressive therapy	2	2	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*100–118*).
d. None of the above	2	2	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*100–118*).
Rheumatoid arthritis				
a. Not receiving immunosuppressive therapy	1	2	1	**Evidence:** Limited evidence demonstrates no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives, progesterone, or estrogen (*122*).
b. Receiving immunosuppressive therapy	1	2/3	1	**Clarification (DMPA):** DMPA use among persons receiving long-term corticosteroid therapy with a history of, or with risk factors for, nontraumatic fractures is classified as category 3. Otherwise, DMPA use for persons with rheumatoid arthritis is classified as category 2. **Evidence:** Limited evidence demonstrates no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives, progesterone, or estrogen (*122*).
Neurologic Conditions				
Headaches				
a. Nonmigraine (mild or severe)	1	1	1	—
b. Migraine				
i. Without aura (includes menstrual migraine)	1	1	1	**Evidence:** No studies directly examined the risk for stroke among women with migraine using POCs (*123*). Limited evidence demonstrated that women using POPs, DMPA, or implants do not have an increased risk for ischemic stroke compared with nonusers (*124*). **Comment:** Menstrual migraine is a subtype of migraine without aura. For more information, see the International Headache Society's *International Classification of Headache Disorders, 3rd ed.* (https://ichd-3.org) (*125*).
ii. With aura	1	1	1	
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	**Clarification:** If a person is taking anticonvulsants, see recommendations for Drug Interactions. Certain anticonvulsants lower POC effectiveness.
Multiple sclerosis				
a. Without prolonged immobility	1	2	1	**Evidence:** Limited evidence demonstrates that use of COCs or oral contraceptives (type not specified) among women with multiple sclerosis does not worsen the clinical course of disease (*126*). **Comment:** Persons with multiple sclerosis might have compromised bone health from disease-related disability, immobility, and use of corticosteroids. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
b. With prolonged immobility	1	2	1	
Depressive Disorders				
Depressive disorders	1	1	1	**Clarification:** If a person is taking psychotropic medications or St. John's wort, see recommendations for Drug Interactions. **Evidence:** The frequency of psychiatric hospitalizations for women with bipolar disorder or depression did not significantly differ among women using DMPA, LNG-IUD, Cu-IUD, or sterilization (*127*).

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Reproductive Tract Infections and Disorders				
Vaginal bleeding patterns				
a. Irregular pattern without heavy bleeding	2	2	2	**Comment:** Irregular menstrual bleeding patterns are common among healthy persons. POC use frequently induces an irregular bleeding pattern. Implant use might induce irregular bleeding patterns, especially during the first 3–6 months, although these patterns might persist longer.
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2	2	2	**Clarification:** Unusually heavy bleeding should raise the suspicion of a serious underlying condition.
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	3	3	2	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. **Comment:** POCs might cause irregular bleeding patterns, which might mask symptoms of underlying pathologic conditions. The effects of DMPA might persist for some time after discontinuation.
Endometriosis	1	1	1	—
Benign ovarian tumors (including cysts)	1	1	1	—
Severe dysmenorrhea	1	1	1	—
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.
a. Suspected gestational trophoblastic disease (immediate postevacuation)				
i. Uterine size first trimester	1	1	1	
ii. Uterine size second trimester	1	1	1	
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)				
i. Undetectable or nonpregnant β–hCG levels	1	1	1	
ii. Decreasing β–hCG levels	1	1	1	
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	1	1	1	
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	1	1	1	
Cervical ectropion	1	1	1	—
Cervical intraepithelial neoplasia	2	2	1	**Evidence:** Among women with persistent human papillomavirus infection, long-term DMPA use (≥5 years) might increase the risk for carcinoma in situ and invasive carcinoma (*128*).
Cervical cancer (awaiting treatment)	2	2	1	**Comment:** Theoretical concern exists that POC use might affect prognosis of the existing disease. While awaiting treatment, POCs may be used. In general, treatment of this condition can render a person infertile.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Undiagnosed mass	2	2	2	**Clarification:** Evaluation of mass should be pursued as early as possible.
b. Benign breast disease	1	1	1	—
c. Family history of cancer	1	1	1	—
d. Breast cancer				
i. Current	4	4	4	**Comment:** Breast cancer is a hormonally sensitive tumor, and the prognosis for persons with current or recent breast cancer might worsen with POC use.
ii. Past and no evidence of current disease for 5 years	3	3	3	
Endometrial hyperplasia	1	1	1	—
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	**Comment:** While awaiting treatment, POCs may be used. In general, treatment of this condition renders a person infertile.
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	**Comment:** While awaiting treatment, POCs may be used. In general, treatment of this condition renders a person infertile.
Uterine fibroids	1	1	1	**Comment:** POCs do not appear to cause growth of uterine fibroids.
Pelvic inflammatory disease				**Comment:** Whether POCs, like COCs, reduce the risk for PID among persons with STIs is unknown; however, they do not protect against HIV infection or lower genital tract STIs.
a. Current PID	1	1	1	
b. Past PID				
i. With subsequent pregnancy	1	1	1	
ii. Without subsequent pregnancy	1	1	1	
Sexually transmitted infections				
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	1	1	1	—
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	—
c. Other factors related to STIs	1	1	1	—
HIV				
High risk for HIV infection	1	1	1	**Evidence:** High-quality evidence from one RCT observed no statistically significant differences in HIV acquisition between DMPA-IM versus Cu-IUD, DMPA-IM versus LNG implant, and Cu-IUD versus LNG implant. Of the low-to-moderate-quality evidence from 14 observational studies, certain studies suggested a possible increased risk for HIV infection with progestin-only injectable use, which was most likely due to unmeasured confounding. Low-quality evidence from three observational studies did not suggest an increased HIV infection risk for implant users. No studies of sufficient quality were identified for POPs (*129–131*).
HIV infection For persons with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	**Clarification:** Drug interactions might exist between hormonal contraceptives and ARV drugs (see recommendations for Drug Interactions). **Evidence:** Overall, evidence does not support an association between POC use and progression of HIV infection. Limited direct evidence on an association between POC use and transmission of HIV to noninfected partners, as well as studies measuring genital viral shedding as a proxy for infectivity, have had mixed results. Studies measuring whether hormonal contraceptive methods affect plasma HIV viral load generally have found no effect (*132–134*).

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Other Infections				
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Uncomplicated	1	1	1	**Evidence:** Among women with uncomplicated schistosomiasis, limited evidence demonstrated that DMPA use had no adverse effects on liver function (*135*).
b. Fibrosis of the liver (if severe, see recommendations for Cirrhosis)	1	1	1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				**Clarification:** If a person is taking rifampin, see recommendations for Drug Interactions. Rifampin is likely to decrease the effectiveness of certain POCs.
a. Nonpelvic	1	1	1	
b. Pelvic	1	1	1	
Malaria	1	1	1	—
Endocrine Conditions				
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. History of gestational disease	1	1	1	**Evidence:** POCs had no adverse effects on serum lipid levels in women with a history of gestational diabetes in two small studies (*136,137*). Limited evidence is inconsistent about the development of noninsulin-dependent diabetes among users of POCs with a history of gestational diabetes (*138–141*).
b. Nonvascular disease				
i. Non-insulin dependent	2	2	2	**Evidence:** Among women with insulin-dependent or noninsulin-dependent diabetes, limited evidence on use of POCs (POPs, DMPA, and LNG implant) suggests that these methods have little effect on short-term or long-term diabetes control (e.g., glycosylated hemoglobin levels), hemostatic markers, or lipid profile (*142–145*).
ii. Insulin dependent	2	2	2	
c. Nephropathy, retinopathy or neuropathy	2	3	2	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. The effects of DMPA might persist for some time after discontinuation. Certain POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs.
d. Other vascular disease or diabetes of >20 years' duration	2	3	2	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. The effects of DMPA might persist for some time after discontinuation. Certain POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs.
Thyroid disorders				
a. Simple goiter	1	1	1	—
b. Hyperthyroid	1	1	1	—
c. Hypothyroid	1	1	1	—

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Gastrointestinal Conditions				
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	2	2	**Evidence:** Risk for disease relapse among women with IBD using oral contraceptives (most studies did not specify formulation) did not increase significantly from that for nonusers (*146*). **Comment:** Absorption of POPs among persons with IBD might be reduced if the person has substantial malabsorption caused by severe disease or small bowel surgery. Women with IBD have a higher prevalence of osteoporosis and osteopenia than the general population. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
Gallbladder disease				
a. Asymptomatic	2	2	2	—
b. Symptomatic				
i. Current	2	2	2	—
ii. Treated by cholecystectomy	2	2	2	—
iii. Medically treated	2	2	2	—
History of cholestasis				
a. Pregnancy related	1	1	1	
b. Past COC related	2	2	2	**Comment:** Theoretical concern exists that a history of COC-related cholestasis might predict subsequent cholestasis with POC use. However, this has not been documented.
Viral hepatitis				
a. Acute or flare	1	1	1	**Evidence**: No direct evidence was identified on POC use among persons with viral hepatitis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Chronic	1	1	1	**Evidence**: No evidence was identified on POC use among persons with viral hepatitis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Cirrhosis Decompensated cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Compensated (normal liver function)	1	1	1	**Evidence**: No direct evidence was identified on POC use among persons with cirrhosis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Decompensated (impaired liver function)	2	3	2	**Evidence:** No direct evidence was identified on POC use among persons with cirrhosis. (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). DMPA use has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Hepatic metabolism of exogenous hormones might be impaired in persons with liver dysfunction, which could lead to increased progestin levels in circulation and progestin-related side effects and adverse events (e.g., thrombosis), which might vary by dose and formulation. Any progestin-related hepatotoxicity might be less tolerated in persons with existing liver dysfunction.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Liver tumors				
Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Benign				
i. Focal nodular hyperplasia	2	2	2	**Evidence:** Limited evidence suggests that progestin use does not influence either progression or regression of focal nodular hyperplasia (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
ii. Hepatocellular adenoma	2	3	2	**Evidence:** Limited evidence suggests that hepatocellular adenomas generally regress or remain stable during progestin use (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Malignant (hepatocellular carcinoma)	3	3	3	**Evidence:** No direct evidence was identified on POC use among persons with hepatocellular carcinoma (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Respiratory Conditions				
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	1	**Clarification:** Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for persons with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions. **Clarification:** Certain drugs to treat cystic fibrosis (e.g., lumacaftor) might reduce effectiveness of hormonal contraceptives, including oral, injectable, transdermal, and implantable contraceptives. **Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with cystic fibrosis is not associated with worsening of disease severity. Very limited evidence suggests that cystic fibrosis does not impair the effectiveness of hormonal contraception (*147*). **Comment:** Persons with cystic fibrosis have a higher prevalence of osteopenia, osteoporosis, and fragility fractures than the general population. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
Hematologic Conditions				
Thalassemia	1	1	1	—
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2/3	1	**Clarification (DMPA):** The category should be assessed according to the severity of the condition and risk for thrombosis. **Evidence:** Limited evidence suggests that POC use does not increase risk for thrombosis among persons with sickle cell disease (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with sickle cell disease are at higher risk for stroke and venous thrombosis than the general population (*148–151*). Use of DMPA, which has been associated with a higher risk for venous thrombosis compared with nonuse (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516), might further elevate risk for thrombosis among persons with sickle cell disease. POC might be beneficial in reducing clinical symptoms (e.g., pain crises) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Iron deficiency anemia	1	1	1	**Comment:** Changes in the menstrual pattern associated with POC use have little effect on hemoglobin levels.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
Solid Organ Transplantation				
Solid organ transplantation				
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. No graft failure	2	2/3	2	**Clarification (DMPA):** DMPA use among persons receiving long-term immunosuppressive therapy with a history of, or risk factors for, nontraumatic fractures is classified as category 3. Otherwise, DMPA use for persons with solid organ transplantation is classified as category 2. **Evidence:** One study observed no differences in transplant-related adverse outcomes (e.g., infection, graft failure, and graft rejection) or occurrence of pregnancy between transplant recipients using the implant and those using no hormonal method (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). No direct evidence was identified on bone health or fracture with use of POCs, including DMPA, among persons with solid organ transplantation. Persons with solid organ transplantation have a higher prevalence of osteoporosis and fracture than the general population, especially in the early posttransplantation period (*152*). Use of DMPA, which has been associated with small changes in bone mineral density compared with nonuse (*4*) might further elevate risk for fracture among persons with solid organ transplantation.
b. Graft failure	2	2/3	2	
Drug Interactions				
Antiretrovirals used for prevention (PrEP) or treatment of HIV infection				**Comment:** These recommendations generally are for ARV agents used alone. However, most persons receiving ARV are using multiple drugs in combination. In general, whether interactions between ARVs and hormonal contraceptives differ when ARVs are given alone or in combination is unknown.
See the following guidelines for the most up-to-date recommendations on drug-drug interactions between hormonal contraception and antiretrovirals: 1) Recommendations for the Use of Antiretroviral Drugs During Pregnancy and Interventions to Reduce Perinatal HIV Transmission in the United States (https://clinicalinfo.hiv.gov/en/guidelines/perinatal/prepregnancy-counseling-childbearing-age-overview?view=full#table-3) (*153*) and 2) Guidelines for the Use of Antiretroviral Agents in Adults and Adolescents with HIV (https://clinicalinfo.hiv.gov/en/guidelines/hiv-clinical-guidelines-adult-and-adolescent-arv/drug-interactions-overview?view=full) (*154*).				
a. Nucleoside reverse transcriptase inhibitors (NRTIs)				
i. Abacavir (ABC)	1	1	1	**Evidence:** NRTIs do not appear to have significant risk for interactions with hormonal contraceptive methods (*155–160*).
ii. Tenofovir (TDF)	1	1	1	
iii. Zidovudine (AZT)	1	1	1	
iv. Lamivudine (3TC)	1	1	1	
v. Didanosine (DDI)	1	1	1	
vi. Emtricitabine (FTC)	1	1	1	
vii. Stavudine (D4T)	1	1	1	

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)				
i. Efavirenz (EFV)	2	1	2	**Clarification:** Evidence suggests drug interactions between EFV and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One study found that women using etonogestrel implants with EFV had a higher pregnancy rate than women not using ARVs, although confidence intervals overlapped and absolute pregnancy rates were still lower than for other hormonal methods; another study found that etonogestrel levels were decreased and 5% of women had presumptive ovulation while using etonogestrel implants with EFV (*161*,*162*). Three studies of women using LNG implants demonstrated increased pregnancy rates for women using EFV-containing ARV therapy compared with no ARV use, although absolute pregnancy rates were still lower than for other hormonal methods in one study (*162*–*164*); another study of LNG implant users found no difference in pregnancy rates with EFV compared with no EFV (165). No significant effects were found on pregnancy rates, DMPA levels, EFV levels, or HIV disease progression in women using DMPA and EFV compared with DMPA alone (*162*,*165*–*169*). No significant effects were found on HIV disease progression in women using LNG implants and EFV compared with no ARVs (*164*). No data have assessed effectiveness of contraceptive implants during later years of use when progestin concentrations are lower and risk for failure from drug interactions might be greater.
ii. Etravirine (ETR)	1	1	1	—
iii. Nevirapine (NVP)	1	1	1	**Evidence:** Five studies found no significant increase in pregnancy rates among women using implants and NVP compared with implants alone (*162*–*165*,*170*). Four studies found no significant increase in pregnancy rates among women using DMPA or other contraceptive injectables and NVP compared with DMPA or other contraceptive injectables alone (*162*,*165*,*168*,*171*). One study found no ovulations or changes in DMPA concentrations (*166*). No effect was found on HIV disease progression with use of NVP and DMPA or LNG implants (*164*,*166*,*168*–*170*,*172*). No data have assessed effectiveness of contraceptive implants during later years of use when progestin concentrations are lower and risk for failure from drug interactions might be greater.
iv. Rilpivirine (RPV)	1	1	1	—
c. Ritonavir-boosted protease inhibitors				
i. Ritonavir-boosted atazanavir (ATV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA. **Evidence:** One pharmacokinetic study demonstrated increased progestin concentrations with use of POPs and ATV/r compared with POPs alone (*173*).
ii. Ritonavir-boosted darunavir (DRV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
iii. Ritonavir-boosted fosamprenavir (FPV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
iv. Ritonavir-boosted lopinavir (LPV/r)	1	1	1	**Evidence:** One study demonstrated no pregnancies, no ovulations, no change in LPV/r level, and no change in HIV disease progression in women using DMPA (*174*); another study found a small increase in pregnancy rate in women using DMPA with LPV/r compared with no ARV therapy, however confidence intervals overlapped (*162*). Two studies found no increased risk for pregnancy in women using implants (*162,163*). Two studies found contraceptive hormones increased in women using LPV/r with DMPA or etonogestrel implants (*161,174*).
v. Ritonavir-boosted saquinavir (SQV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
vi. Ritonavir-boosted tipranavir (TPV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
d. Protease inhibitors without ritonavir				
i. Atazanavir (ATV)	1	1	1	**Comment:** When ATV is administered with cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels might vary when combined with other ARVs.
ii. Fosamprenavir (FPV)	2	2	2	**Clarification:** Theoretical concern exists that interactions between FPV and hormonal contraceptives leading to decreased levels of FPV might diminish effectiveness of the ARV drug. The drug interaction likely involves CYP3A4 pathways; POCs have less effect on CYP3A4 enzymes than CHCs.
iii. Indinavir (IDV)	1	1	1	—
iv. Nelfinavir (NFV)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA. Concern exists that interactions between NFV and POCs might decrease NFV levels. **Evidence:** One study found no pregnancies, no ovulations, no change in DMPA concentrations and no change in HIV disease progression with use of DMPA and NFV compared with DMPA alone; NFV concentrations were decreased with concomitant DMPA use (*166,168*).
e. CCR5 co-receptor antagonists				
i. Maraviroc (MVC)	1	1	1	—

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
f. HIV integrase strand transfer inhibitors				
i. Raltegravir (RAL)	1	1	1	—
ii. Dolutegravir (DTG)	1	1	1	—
iii. Elvitegravir (EVG)	1	1	1	**Comment:** When EVG is administered with cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels might vary when combined with other ARVs.
g. Fusion inhibitors				
i. Enfuvirtide	1	1	1	—
Anticonvulsant therapy				
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	2	1	3	**Clarification:** Although the interaction of certain anticonvulsants with POPs and etonogestrel implants is not harmful, it is likely to reduce the effectiveness of POPs and etonogestrel implants. Whether increasing the hormone dose of POPs alleviates this concern remains unclear. Use of other contraceptives should be encouraged for persons who are long-term users of any of these drugs. Use of DMPA is a category 1 because its effectiveness is not decreased by use of certain anticonvulsants. **Evidence:** Use of certain anticonvulsants might decrease the effectiveness of POCs (*175–178*).
b. Lamotrigine	1	1	1	**Evidence:** No drug interactions have been reported among women with epilepsy receiving lamotrigine and POCs (*178,179*).
Antimicrobial therapy				
a. Broad-spectrum antibiotics	1	1	1	—
b. Antifungals	1	1	1	—
c. Antiparasitics	1	1	1	—
d. Rifampin or rifabutin therapy	2	1	3	**Clarification:** Although the interaction of rifampin or rifabutin with POPs and etonogestrel implants is not harmful, it is likely to reduce the effectiveness of POPs and etonogestrel implants. Use of other contraceptives should be encouraged for persons who are long-term users of any of these drugs. Use of DMPA is a category 1 because its effectiveness is not decreased by use of rifampin or rifabutin. Whether increasing the hormone dose of POPs alleviates this concern remains unclear.
Psychotropic medications				**Comment:** For many common psychotropic agents, limited or no theoretical concern exits for clinically significant drug interactions when co-administered with hormonal contraceptives. However, either no or very limited data exist examining potential interactions for these classes of medications.
a. Selective serotonin reuptake inhibitors (SSRIs)	1	1	1	**Evidence:** No evidence specifically examined the use of POCs with SSRIs. Limited clinical and pharmacokinetic data do not demonstrate concern for SSRIs decreasing the effectiveness of oral contraceptives. Limited evidence suggests that for women taking SSRIs, the use of hormonal contraceptives was not associated with differences in effectiveness of the SSRI for treatment or in adverse events when compared with women not taking hormonal contraceptives (*180*). **Comment:** Drugs that are inhibitors of CYP3A4 or CYP2C9 theoretically have the potential to increase levels of contraceptive steroid, which might increase adverse events. Fluvoxamine is an SSRI known to be a moderate inhibitor of both 3A4 and 2C9; however, no clinical or pharmacokinetic studies were identified to explore potential drug-drug interactions.

See table footnotes on page 63.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarification/Evidence/Comment
	Implant	DMPA	POP	
St. John's wort	2	1	2	**Evidence:** No evidence specifically examined the use of POCs with St. John's wort. Although clinical data are limited, studies with pharmacokinetic and pharmacodynamics outcomes raise concern that St. John's wort might decrease effectiveness of hormonal contraceptives, including increased risk for breakthrough bleeding and ovulation and increased metabolism of estrogen and progestin. Any interactions might be dependent on the dose of St. John's wort, and the concentration of active ingredients across types of St. John's wort preparations might vary (*181*). **Comment:** Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.

Abbreviations: ARV = antiretroviral; BMD = bone mineral density; BMI = body mass index; CHC = combined hormonal contraceptive; CKD = chronic kidney disease; COC = combined oral contraceptive; Cu-IUD = copper intrauterine device; CYP = cytochrome P450; DMPA = depot medroxyprogesterone acetate; DRSP = drospirenone; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; IBD = inflammatory bowel disease; IM = intramuscular; LDL = low-density lipoprotein; LNG = levonorgestrel; LNG-IUD = levonorgestrel intrauterine device; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; POC = progestin-only contraceptive; POP = progestin-only pill; PrEP = pre-exposure prophylaxis; RCT = randomized clinical trial; SLE = systemic lupus erythematosus; STI = sexually transmitted infection; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*; U.S. SPR = *U.S. Selected Practice Recommendations for Contraceptive Use*; VTE = venous thromboembolism.

References

1. Curtis KM, Nguyen AT, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2024. MMWR Recomm Rep 2024;73(No. RR-3):1–77.
2. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
3. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
4. Curtis KM, Martins SL. Progestogen-only contraception and bone mineral density: a systematic review. Contraception 2006;73:470–87. PMID:16627031 https://doi.org/10.1016/j.contraception.2005.12.010
5. Lanza LL, McQuay LJ, Rothman KJ, et al. Use of depot medroxyprogesterone acetate contraception and incidence of bone fracture. Obstet Gynecol 2013;121:593–600. PMID:23635623 https://doi.org/10.1097/AOG.0b013e318283d1a1
6. Harel Z, Riggs S, Vaz R, Flanagan P, Harel D, Machan JT. Bone accretion in adolescents using the combined estrogen and progestin transdermal contraceptive method Ortho Evra: a pilot study. J Pediatr Adolesc Gynecol 2010;23:23–31. PMID:19647454 https://doi.org/10.1016/j.jpag.2009.04.008
7. Kaunitz AM, Miller PD, Rice VM, Ross D, McClung MR. Bone mineral density in women aged 25–35 years receiving depot medroxyprogesterone acetate: recovery following discontinuation. Contraception 2006;74:90
9. PMID:16860045 https://doi.org/10.1016/j.contraception.2006.03.010
8. Kaunitz AM, Arias R, McClung M. Bone density recovery after depot medroxyprogesterone acetate injectable contraception use. Contraception 2008;77:67–76. PMID:18226668 https://doi.org/10.1016/j.contraception.2007.10.005
9. Kaunitz AM, Darney PD, Ross D, Wolter KD, Speroff L. Subcutaneous DMPA vs. intramuscular DMPA: a 2-year randomized study of contraceptive efficacy and bone mineral density. Contraception 2009;80:7–17. PMID:19501210 https://doi.org/10.1016/j.contraception.2009.02.005
10. Lappe JM, Stegman MR, Recker RR. The impact of lifestyle factors on stress fractures in female Army recruits. Osteoporos Int 2001;12:35–42. PMID:11305081 https://doi.org/10.1007/s001980170155
11. Lara-Torre E, Edwards CP, Perlman S, Hertweck SP. Bone mineral density in adolescent females using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2004;17:17–21. PMID:15010034 https://doi.org/10.1016/j.jpag.2003.11.017
12. Lopez LM, Chen M, Mullins S, Curtis KM, Helmerhorst FM. Steroidal contraceptives and bone fractures in women: evidence from observational studies. Cochrane Database Syst Rev 2012;8:CD009849. PMID:22895991
13. Lopez LM, Grimes DA, Schulz KF, Curtis KM. Steroidal contraceptives: effect on bone fractures in women. Cochrane Database Syst Rev 2011;(7):CD006033. PMID:21735401
14. Meier C, Brauchli YB, Jick SS, Kraenzlin ME, Meier CR. Use of depot medroxyprogesterone acetate and fracture risk. J Clin Endocrinol Metab 2010;95:4909–16. PMID:20685865 https://doi.org/10.1210/jc.2010-0032
15. Merki-Feld GS, Neff M, Keller PJ. A 2-year prospective study on the effects of depot medroxyprogesterone acetate on bone mass-response to estrogen and calcium therapy in individual users. Contraception 2003;67:79–86. PMID:12586317 https://doi.org/10.1016/S0010-7824(02)00460-2
16. Monteiro-Dantas C, Espejo-Arce X, Lui-Filho JF, Fernandes AM, Monteiro I, Bahamondes L. A three-year longitudinal evaluation of the forearm bone density of users of etonogestrel- and levonorgestrel-releasing contraceptive implants. Reprod Health 2007;4:11. PMID:17997844 https://doi.org/10.1186/1742-4755-4-11
17. Naessen T, Olsson SE, Gudmundson J. Differential effects on bone density of progestogen-only methods for contraception in premenopausal women. Contraception 1995;52:35–9. PMID:8521712 https://doi.org/10.1016/0010-7824(95)00121-P

18. Sanches L, Marchi NM, Castro S, Juliato CT, Villarroel M, Bahamondes L. Forearm bone mineral density in postmenopausal former users of depot medroxyprogesterone acetate. Contraception 2008;78:365–9. PMID:18929732 https://doi.org/10.1016/j.contraception.2008.07.013
19. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Injectable hormone contraception and bone density: results from a prospective study. Epidemiology 2002;13:581–7. PMID:12192229 https://doi.org/10.1097/00001648-200209000-00015
20. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Change in bone mineral density among adolescent women using and discontinuing depot medroxyprogesterone acetate contraception. Arch Pediatr Adolesc Med 2005;159:139–44. PMID:15699307 https://doi.org/10.1001/archpedi.159.2.139
21. Segall-Gutierrez P, Agarwal R, Ge M, Lopez C, Hernandez G, Stanczyk FZ. A pilot study examining short-term changes in bone mineral density among class 3 obese users of depot-medroxyprogesterone acetate. Eur J Contracept Reprod Health Care 2013;18:199–205. PMID:23530919 https://doi.org/10.3109/13625187.2013.774358
22. Tang OS, Tang G, Yip PS, Li B. Further evaluation on long-term depot-medroxyprogesterone acetate use and bone mineral density: a longitudinal cohort study. Contraception 2000;62:161–4. PMID:11137068 https://doi.org/10.1016/S0010-7824(00)00168-2
23. Vestergaard P, Rejnmark L, Mosekilde L. The effects of depot medroxyprogesterone acetate and intrauterine device use on fracture risk in Danish women. Contraception 2008;78:459–64. PMID:19014791 https://doi.org/10.1016/j.contraception.2008.07.014
24. Viola AS, Castro S, Marchi NM, Bahamondes MV, Viola CF, Bahamondes L. Long-term assessment of forearm bone mineral density in postmenopausal former users of depot medroxyprogesterone acetate. Contraception 2011;84:122–7. PMID:21757052 https://doi.org/10.1016/j.contraception.2010.11.007
25. Walsh JS, Eastell R, Peel NF. Depot medroxyprogesterone acetate use after peak bone mass is associated with increased bone turnover but no decrease in bone mineral density. Fertil Steril 2010;93:697–701. PMID:19013564 https://doi.org/10.1016/j.fertnstert.2008.10.004
26. Wetmore CM, Ichikawa L, LaCroix AZ, Ott SM, Scholes D. Association between caffeine intake and bone mass among young women: potential effect modification by depot medroxyprogesterone acetate use. Osteoporos Int 2008;19:519–27. PMID:18004611 https://doi.org/10.1007/s00198-007-0473-2
27. Wong AY, Tang LC, Chin RK. Levonorgestrel-releasing intrauterine system (Mirena) and Depot medroxyprogesterone acetate (Depoprovera) as long-term maintenance therapy for patients with moderate and severe endometriosis: a randomised controlled trial. Aust N Z J Obstet Gynaecol 2010;50:273–9. PMID:20618247 https://doi.org/10.1111/j.1479-828X.2010.01152.x
28. Yang KY, Kim YS, Ji YI, Jung MH. Changes in bone mineral density of users of the levonorgestrel-releasing intrauterine system. J Nippon Med Sch 2012;79:190–4. PMID:22791119 https://doi.org/10.1272/jnms.79.190
29. Zhang MH, Zhang W, Zhang AD, Yang Y, Gai L. Effect of depot medroxyprogesterone acetate on bone mineral density in adolescent women. Chin Med J (Engl) 2013;126:4043–7. PMID:24229671 https://doi.org/10.3760/cma.j.issn.0366-6999.20130885
30. Bahamondes MV, Monteiro I, Castro S, Espejo-Arce X, Bahamondes L. Prospective study of the forearm bone mineral density of long-term users of the levonorgestrel-releasing intrauterine system. Hum Reprod 2010;25:1158–64. PMID:20185512 https://doi.org/10.1093/humrep/deq043
31. Banks E, Berrington A, Casabonne D. Overview of the relationship between use of progestogen-only contraceptives and bone mineral density. BJOG 2001;108:1214–21. PMID:11843382 https://doi.org/10.1111/j.1471-0528.2001.00296.x
32. Beerthuizen R, van Beek A, Massai R, Mäkäräinen L, Hout J, Bennink HC. Bone mineral density during long-term use of the progestagen contraceptive implant Implanon compared to a non-hormonal method of contraception. Hum Reprod 2000;15:118–22. PMID:10611199 https://doi.org/10.1093/humrep/15.1.118
33. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in adolescents using norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives for contraception. Contraception 2007;75:438–43. PMID:17519149 https://doi.org/10.1016/j.contraception.2007.02.001
34. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in a cohort of adolescents during use of norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives and after discontinuation of norethisterone enanthate. Contraception 2009;79:345–9. PMID:19341845 https://doi.org/10.1016/j.contraception.2008.11.009
35. Berenson AB, Breitkopf CR, Grady JJ, Rickert VI, Thomas A. Effects of hormonal contraception on bone mineral density after 24 months of use. Obstet Gynecol 2004;103:899–906. PMID:15121563 https://doi.org/10.1097/01.AOG.0000117082.49490.d5
36. Berenson AB, Rahman M, Breitkopf CR, Bi LX. Effects of depot medroxyprogesterone acetate and 20-microgram oral contraceptives on bone mineral density. Obstet Gynecol 2008;112:788–99. PMID:18827121 https://doi.org/10.1097/AOG.0b013e3181875b78
37. Busen NH, Britt RB, Rianon N. Bone mineral density in a cohort of adolescent women using depot medroxyprogesterone acetate for one to two years. J Adolesc Health 2003;32:257–9. PMID:12667729 https://doi.org/10.1016/S1054-139X(02)00567-0
38. Caird LE, Reid-Thomas V, Hannan WJ, Gow S, Glasier AF. Oral progestogen-only contraception may protect against loss of bone mass in breast-feeding women. Clin Endocrinol (Oxf) 1994;41:739–45. PMID:7889609 https://doi.org/10.1111/j.1365-2265.1994.tb02788.x
39. Clark MK, Sowers M, Levy B, Nichols S. Bone mineral density loss and recovery during 48 months in first-time users of depot medroxyprogesterone acetate. Fertil Steril 2006;86:1466–74. PMID:16996507 https://doi.org/10.1016/j.fertnstert.2006.05.024
40. Cromer BA, Lazebnik R, Rome E, et al. Double-blinded randomized controlled trial of estrogen supplementation in adolescent girls who receive depot medroxyprogesterone acetate for contraception. Am J Obstet Gynecol 2005;192:42–7. PMID:15672001 https://doi.org/10.1016/j.ajog.2004.07.041
41. Cromer BA, Blair JM, Mahan JD, Zibners L, Naumovski Z. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. J Pediatr 1996;129:671–6. PMID:8917232 https://doi.org/10.1016/S0022-3476(96)70148-8
42. Cromer BA, Bonny AE, Stager M, et al. Bone mineral density in adolescent females using injectable or oral contraceptives: a 24-month prospective study. Fertil Steril 2008;90:2060–7. PMID:18222431 https://doi.org/10.1016/j.fertnstert.2007.10.070

43. Cromer BA, Stager M, Bonny A, et al. Depot medroxyprogesterone acetate, oral contraceptives and bone mineral density in a cohort of adolescent girls. J Adolesc Health 2004;35:434–41. PMID:15581522 https://doi.org/10.1016/j.jadohealth.2004.07.005
44. Cundy T, Ames R, Horne A, et al. A randomized controlled trial of estrogen replacement therapy in long-term users of depot medroxyprogesterone acetate. J Clin Endocrinol Metab 2003;88:78–81. PMID:12519833 https://doi.org/10.1210/jc.2002-020874
45. Cundy T, Cornish J, Evans MC, Roberts H, Reid IR. Recovery of bone density in women who stop using medroxyprogesterone acetate. BMJ 1994;308:247–8. PMID:8111260 https://doi.org/10.1136/bmj.308.6923.247
46. Cundy T, Cornish J, Roberts H, Reid IR. Menopausal bone loss in long-term users of depot medroxyprogesterone acetate contraception. Am J Obstet Gynecol 2002;186:978–83. PMID:12015524 https://doi.org/10.1067/mob.2002.122420
47. Di X, Li Y, Zhang C, Jiang J, Gu S. Effects of levonorgestrel-releasing subdermal contraceptive implants on bone density and bone metabolism. Contraception 1999;60:161–6. PMID:10640160 https://doi.org/10.1016/S0010-7824(99)00080-3
48. Díaz S, Reyes MV, Zepeda A, et al. Norplant((R)) implants and progesterone vaginal rings do not affect maternal bone turnover and density during lactation and after weaning. Hum Reprod 1999;14:2499–505. PMID:10527977 https://doi.org/10.1093/humrep/14.10.2499
49. Gai L, Zhang J, Zhang H, Gai P, Zhou L, Liu Y. The effect of depot medroxyprogesterone acetate (DMPA) on bone mineral density (BMD) and evaluating changes in BMD after discontinuation of DMPA in Chinese women of reproductive age. Contraception 2011;83:218–22. PMID:21310282 https://doi.org/10.1016/j.contraception.2010.07.027
50. Bahamondes L, Espejo-Arce X, Hidalgo MM, Hidalgo-Regina C, Teatin-Juliato C, Petta CA. A cross-sectional study of the forearm bone density of long-term users of levonorgestrel-releasing intrauterine system. Hum Reprod 2006;21:1316–9. PMID:16373404 https://doi.org/10.1093/humrep/dei457
51. Bahamondes L, Monteiro-Dantas C, Espejo-Arce X, et al. A prospective study of the forearm bone density of users of etonorgestrel- and levonorgestrel-releasing contraceptive implants. Hum Reprod 2006;21:466–70. PMID:16253974 https://doi.org/10.1093/humrep/dei358
52. Pitts SA, Feldman HA, Dorale A, Gordon CM. Bone mineral density, fracture, and vitamin D in adolescents and young women using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2012;25:23–6. PMID:22078997 https://doi.org/10.1016/j.jpag.2011.07.014
53. US Department of Agriculture; US Department of Health and Human Services. Dietary guidelines for Americans, 2020–2025. 9th ed. Washington, DC: US Department of Agriculture and US Department of Health and Human Services; 2020. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf
54. Meek JY, Noble L; Section on Breastfeeding. Policy statement: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057988. PMID:35921640 https://doi.org/10.1542/peds.2022-057988
55. Braga GC, Ferriolli E, Quintana SM, Ferriani RA, Pfrimer K, Vieira CS. Immediate postpartum initiation of etonogestrel-releasing implant: A randomized controlled trial on breastfeeding impact. Contraception 2015;92:536–42. PMID:26209863 https://doi.org/10.1016/j.contraception.2015.07.009
56. Phillips SJ, Tepper NK, Kapp N, Nanda K, Temmerman M, Curtis KM. Progestogen-only contraceptive use among breastfeeding women: a systematic review. Contraception 2016;94:226–52. PMID:26410174 https://doi.org/10.1016/j.contraception.2015.09.010
57. Raymond EG, Weaver MA, Louie KS, et al. Effects of depot medroxyprogesterone acetate injection timing on medical abortion efficacy and repeat pregnancy: a randomized controlled trial. Obstet Gynecol 2016;128:739–45. PMID:27607859 https://doi.org/10.1097/AOG.0000000000001627
58. Kim C, Nguyen AT, Berry-Bibee E, Ermias Y, Gaffield ME, Kapp N. Systemic hormonal contraception initiation after abortion: a systematic review and meta-analysis. Contraception 2021;103:291–304. PMID:33548267 https://doi.org/10.1016/j.contraception.2021.01.017
59. Beksinska ME, Smit JA, Kleinschmidt I, Milford C, Farley TM. Prospective study of weight change in new adolescent users of DMPA, NET-EN, COCs, nonusers and discontinuers of hormonal contraception. Contraception 2010;81:30–4. PMID:20004270 https://doi.org/10.1016/j.contraception.2009.07.007
60. Bender NM, Segall-Gutierrez P, Najera SO, Stanczyk FZ, Montoro M, Mishell DR Jr. Effects of progestin-only long-acting contraception on metabolic markers in obese women. Contraception 2013;88:418–25. PMID:23410714 https://doi.org/10.1016/j.contraception.2012.12.007
61. Berenson AB, Rahman M. Changes in weight, total fat, percent body fat, and central-to-peripheral fat ratio associated with injectable and oral contraceptive use. Am J Obstet Gynecol 2009;200:329.e1–8. PMID:19254592 https://doi.org/10.1016/j.ajog.2008.12.052
62. Bonny AE, Secic M, Cromer B. Early weight gain related to later weight gain in adolescents on depot medroxyprogesterone acetate. Obstet Gynecol 2011;117:793–7. PMID:21422849 https://doi.org/10.1097/AOG.0b013e31820f387c
63. Bonny AE, Ziegler J, Harvey R, Debanne SM, Secic M, Cromer BA. Weight gain in obese and nonobese adolescent girls initiating depot medroxyprogesterone, oral contraceptive pills, or no hormonal contraceptive method. Arch Pediatr Adolesc Med 2006;160:40–5. PMID:16389209 https://doi.org/10.1001/archpedi.160.1.40
64. Clark MK, Dillon JS, Sowers M, Nichols S. Weight, fat mass, and central distribution of fat increase when women use depot-medroxyprogesterone acetate for contraception. Int J Obes 2005;29:1252–8. PMID:15997247 https://doi.org/10.1038/sj.ijo.0803023
65. Gerlach LS, Saldaña SN, Wang Y, Nick TG, Spigarelli MG. Retrospective review of the relationship between weight change and demographic factors following initial depot medroxyprogesterone acetate injection in adolescents. Clin Ther 2011;33:182–7. PMID:21397330 https://doi.org/10.1016/j.clinthera.2011.02.008
66. Jain J, Jakimiuk AJ, Bode FR, Ross D, Kaunitz AM. Contraceptive efficacy and safety of DMPA-SC. Contraception 2004;70:269–75. PMID:15451329 https://doi.org/10.1016/j.contraception.2004.06.011
67. Kozlowski KJ, Rickert VI, Hendon A, Davis P. Adolescents and Norplant: preliminary findings of side effects. J Adolesc Health 1995;16:373–8. PMID:7662687 https://doi.org/10.1016/S1054-139X(94)00029-E

68. Le YL, Rahman M, Berenson AB. Early weight gain predicting later weight gain among depot medroxyprogesterone acetate users. Obstet Gynecol 2009;114:279–84. PMID:19622988 https://doi.org/10.1097/AOG.0b013e3181af68b2
69. Leiman G. Depo-medroxyprogesterone acetate as a contraceptive agent: its effect on weight and blood pressure. Am J Obstet Gynecol 1972;114:97–102. PMID:4637044 https://doi.org/10.1016/0002-9378(72)90296-7
70. Lopez LM, Grimes DA, Chen M, et al. Hormonal contraceptives for contraception in overweight or obese women. Cochrane Database Syst Rev 2013;4:CD008452. PMID:23633356
71. Mangan SA, Larsen PG, Hudson S. Overweight teens at increased risk for weight gain while using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2002;15:79–82. PMID:12057528 https://doi.org/10.1016/S1083-3188(01)00147-4
72. Nyirati CM, Habash DL, Shaffer LE. Weight and body fat changes in postpartum depot-medroxyprogesterone acetate users. Contraception 2013;88:169–76. PMID:23177262 https://doi.org/10.1016/j.contraception.2012.10.016
73. Pantoja M, Medeiros T, Baccarin MC, Morais SS, Bahamondes L, dos Santos Fernandes AM. Variations in body mass index of users of depot-medroxyprogesterone acetate as a contraceptive. Contraception 2010;81:107–11. PMID:20103446 https://doi.org/10.1016/j.contraception.2009.07.008
74. Risser WL, Gefter LR, Barratt MS, Risser JM. Weight change in adolescents who used hormonal contraception. J Adolesc Health 1999;24:433–6. PMID:10401972 https://doi.org/10.1016/S1054-139X(98)00151-7
75. Segall-Gutierrez P, Xiang AH, Watanabe RM, et al. Deterioration in cardiometabolic risk markers in obese women during depot medroxyprogesterone acetate use. Contraception 2012;85:36–41. PMID:22067800 https://doi.org/10.1016/j.contraception.2011.04.016
76. Westhoff C, Jain JK, Milsom I, Ray A. Changes in weight with depot medroxyprogesterone acetate subcutaneous injection 104 mg/0.65 mL. Contraception 2007;75:261–7. PMID:17362703 https://doi.org/10.1016/j.contraception.2006.12.009
77. Paulen ME, Zapata LB, Cansino C, Curtis KM, Jamieson DJ. Contraceptive use among women with a history of bariatric surgery: a systematic review. Contraception 2010;82:86–94. PMID:20682146 https://doi.org/10.1016/j.contraception.2010.02.008
78. World Health Organization Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Cardiovascular disease and use of oral and injectable progestogen-only contraceptives and combined injectable contraceptives. Results of an international, multicenter, case-control study. Contraception 1998;57:315–24. PMID:9673838 https://doi.org/10.1016/S0010-7824(98)00041-9
79. Martinelli I, Lensing AW, Middeldorp S, et al. Recurrent venous thromboembolism and abnormal uterine bleeding with anticoagulant and hormone therapy use. Blood 2016;127:1417–25. PMID:26696010 https://doi.org/10.1182/blood-2015-08-665927
80. Sönmezer M, Atabekoğlu C, Cengiz B, Dökmeci F, Cengiz SD. Depot-medroxyprogesterone acetate in anticoagulated patients with previous hemorrhagic corpus luteum. Eur J Contracept Reprod Health Care 2005;10:9–14. PMID:16036292 https://doi.org/10.1080/13625180400020952
81. Barbhaiya M, Zuily S, Naden R, et al.; ACR/EULAR APS Classification Criteria Collaborators. The 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria. Arthritis Rheumatol 2023;75:1687–702. PMID:37635643 https://doi.org/10.1002/art.42624
82. Bergendal A, Persson I, Odeberg J, et al. Association of venous thromboembolism with hormonal contraception and thrombophilic genotypes. Obstet Gynecol 2014;124:600–9. PMID:25162263 https://doi.org/10.1097/AOG.0000000000000411
83. Leon L, Giannoukas AD, Dodd D, Chan P, Labropoulos N. Clinical significance of superficial vein thrombosis. Eur J Vasc Endovasc Surg 2005;29:10–7. PMID:15570265 https://doi.org/10.1016/j.ejvs.2004.09.021
84. Tepper NK, Paulen ME, Marchbanks PA, Curtis KM. Safety of contraceptive use among women with peripartum cardiomyopathy: a systematic review. Contraception 2010;82:95–101. PMID:20682147 https://doi.org/10.1016/j.contraception.2010.02.004
85. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
86. Folsom AR, Lutsey PL, Astor BC, Wattanakit K, Heckbert SR, Cushman M; Atherosclerosis Risk in Communities Study. Chronic kidney disease and venous thromboembolism: a prospective study. Nephrol Dial Transplant 2010;25:3296–301. PMID:20353958 https://doi.org/10.1093/ndt/gfq179
87. Kayali F, Najjar R, Aswad F, Matta F, Stein PD. Venous thromboembolism in patients hospitalized with nephrotic syndrome. Am J Med 2008;121:226–30. PMID:18328307 https://doi.org/10.1016/j.amjmed.2007.08.042
88. Mahmoodi BK, ten Kate MK, Waanders F, et al. High absolute risks and predictors of venous and arterial thromboembolic events in patients with nephrotic syndrome: results from a large retrospective cohort study. Circulation 2008;117:224–30. PMID:18158362 https://doi.org/10.1161/CIRCULATIONAHA.107.716951
89. Singhal R, Brimble KS. Thromboembolic complications in the nephrotic syndrome: pathophysiology and clinical management. Thromb Res 2006;118:397–407. PMID:15990160 https://doi.org/10.1016/j.thromres.2005.03.030
90. Wattanakit K, Cushman M, Stehman-Breen C, Heckbert SR, Folsom AR. Chronic kidney disease increases risk for venous thromboembolism. J Am Soc Nephrol 2008;19:135–40. PMID:18032796 https://doi.org/10.1681/ASN.2007030308
91. Naylor KL, McArthur E, Leslie WD, et al. The three-year incidence of fracture in chronic kidney disease. Kidney Int 2014;86:810–8. PMID:24429401 https://doi.org/10.1038/ki.2013.547
92. Pimentel A, Ureña-Torres P, Zillikens MC, Bover J, Cohen-Solal M. Fractures in patients with CKD-diagnosis, treatment, and prevention: a review by members of the European Calcified Tissue Society and the European Renal Association of Nephrology Dialysis and Transplantation. Kidney Int 2017;92:1343–55. PMID:28964571 https://doi.org/10.1016/j.kint.2017.07.021
93. Vilaca T, Salam S, Schini M, et al. Risks of hip and nonvertebral fractures in patients with CKD G3a-G5D: a systematic review and meta-analysis. Am J Kidney Dis 2020;76:521–32. PMID:32654892 https://doi.org/10.1053/j.ajkd.2020.02.450
94. Molnar AO, Bota SE, McArthur E, et al. Risk and complications of venous thromboembolism in dialysis patients. Nephrol Dial Transplant 2018;33:874–80. PMID:28992258

95. Tveit DP, Hypolite IO, Hshieh P, et al. Chronic dialysis patients have high risk for pulmonary embolism. Am J Kidney Dis 2002;39:1011–7. PMID:11979344 https://doi.org/10.1053/ajkd.2002.32774
96. Wang IK, Shen TC, Muo CH, Yen TH, Sung FC. Risk of pulmonary embolism in patients with end-stage renal disease receiving long-term dialysis. Nephrol Dial Transplant 2017;32:1386–93. PMID:27448674
97. Alem AM, Sherrard DJ, Gillen DL, et al. Increased risk of hip fracture among patients with end-stage renal disease. Kidney Int 2000;58:396–9. PMID:10886587 https://doi.org/10.1046/j.1523-1755.2000.00178.x
98. Ball AM, Gillen DL, Sherrard D, et al. Risk of hip fracture among dialysis and renal transplant recipients. JAMA 2002;288:3014–8. PMID:12479766 https://doi.org/10.1001/jama.288.23.3014
99. Jadoul M, Albert JM, Akiba T, et al. Incidence and risk factors for hip or other bone fractures among hemodialysis patients in the Dialysis Outcomes and Practice Patterns Study. Kidney Int 2006;70:1358–66. PMID:16929251 https://doi.org/10.1038/sj.ki.5001754
100. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81. PMID:15226516 https://doi.org/10.1093/rheumatology/keh282
101. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9. PMID:15280571 https://doi.org/10.1093/rheumatology/keh331
102. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8. PMID:12180730
103. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7. PMID:11665973 https://doi.org/10.1002/1529-0131(200110)44:10<2331::AID-ART395>3.0.CO;2-I
104. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33. PMID:1771400 https://doi.org/10.3109/03009749109096822
105. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30. PMID:8448613 https://doi.org/10.1093/rheumatology/32.3.227
106. Jungers P, Dougados M, Pélissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23. PMID:7092961 https://doi.org/10.1002/art.1780250603
107. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15. PMID:9048514 https://doi.org/10.1093/oxfordjournals.aje.a009122
108. McAlindon T, Giannotta L, Taub N, D'Cruz D, Hughes G. Environmental factors predicting nephritis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4. PMID:8257208 https://doi.org/10.1136/ard.52.10.720
109. McDonald J, Stewart J, Urowitz MB, Gladman DD. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60. PMID:1540039 https://doi.org/10.1136/ard.51.1.56
110. Mintz G, Gutiérrez G, Delezé M, Rodríguez E. Contraception with progestagens in systemic lupus erythematosus. Contraception 1984;30:29–38. PMID:6434228 https://doi.org/10.1016/0010-7824(84)90076-3
111. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45. PMID:7654797 https://doi.org/10.1002/art.1790080305
112. Petri M. Lupus in Baltimore: evidence-based 'clinical pearls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3. PMID:16425579 https://doi.org/10.1191/0961203305lu2230xx
113. Petri M, Kim MY, Kalunian KC, et al.; OC-SELENA Trial. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8. PMID:16354891 https://doi.org/10.1056/NEJMoa051135
114. Sánchez-Guerrero J, Uribe AG, Jiménez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49. PMID:16354890 https://doi.org/10.1056/NEJMoa050817
115. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12. PMID:16082635 https://doi.org/10.1002/art.21314
116. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3. PMID:16202726 https://doi.org/10.1016/j.ajog.2005.05.002
117. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6. PMID:12465147
118. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5. PMID:1251849 https://doi.org/10.1016/0002-9343(76)90431-9
119. Culwell KR, Curtis KM, Del Carmen Cravioto M. Safety of contraceptive method use among women with systemic lupus erythematosus: a systematic review. Obstet Gynecol 2009;114:341–53. PMID:19622996 https://doi.org/10.1097/AOG.0b013e3181ae9c64
120. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, Nantiruj K, Sumethkul V, Janwityanujit S. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51. PMID:17805483 https://doi.org/10.1007/s10067-007-0721-z
121. Wahl DG, Guillemin F, de Maistre E, Perret C, Lecompte T, Thibaut G. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73. PMID:9229367 https://doi.org/10.1177/096120339700600510
122. Farr SL, Folger SG, Paulen ME, Curtis KM. Safety of contraceptive methods for women with rheumatoid arthritis: a systematic review. Contraception 2010;82:64–71. PMID:20682144 https://doi.org/10.1016/j.contraception.2010.02.003
123. Tepper NK, Whiteman MK, Zapata LB, Marchbanks PA, Curtis KM. Safety of hormonal contraceptives among women with migraine: a systematic review. Contraception 2016;94:630–40. PMID:27153744 https://doi.org/10.1016/j.contraception.2016.04.016
124. Tepper NK, Whiteman MK, Marchbanks PA, James AH, Curtis KM. Progestin-only contraception and thromboembolism: a systematic review. Contraception 2016;94:678–700. PMID:27153743 https://doi.org/10.1016/j.contraception.2016.04.014

125. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders. 3rd edition. Cephalalgia 2018;38:1–211. https://www.ichd-3.org/wp-content/uploads/2018/01/The-International-Classification-of-Headache-Disorders-3rd-Edition-2018.pdf
126. Zapata LB, Oduyebo T, Whiteman MK, Houtchens MK, Marchbanks PA, Curtis KM. Contraceptive use among women with multiple sclerosis: a systematic review. Contraception 2016;94:612–20. PMID:27452316 https://doi.org/10.1016/j.contraception.2016.07.013
127. Pagano HP, Zapata LB, Berry-Bibee EN, Nanda K, Curtis KM. Safety of hormonal contraception and intrauterine devices among women with depressive and bipolar disorders: a systematic review. Contraception 2016;94:641–9. PMID:27364100 https://doi.org/10.1016/j.contraception.2016.06.012
128. Smith JS, Green J, Berrington de Gonzalez A, et al. Cervical cancer and use of hormonal contraceptives: a systematic review. Lancet 2003;361:1159–67. PMID:12686037 https://doi.org/10.1016/S0140-6736(03)12949-2
129. Ahmed K, Baeten JM, Beksinska M, et al.; Evidence for Contraceptive Options and HIV Outcomes (ECHO) Trial Consortium. HIV incidence among women using intramuscular depot medroxyprogesterone acetate, a copper intrauterine device, or a levonorgestrel implant for contraception: a randomized, multicentre, open-label trial. Lancet 2019;394:303–13. PMID:31204114 https://doi.org/10.1016/S0140-6736(19)31288-7
130. Curtis KM, Hannaford PC, Rodriguez MI, Chipato T, Steyn PS, Kiarie JN. Hormonal contraception and HIV acquisition among women: an updated systematic review. BMJ Sex Reprod Health 2020;46:8–16. PMID:31919239 https://doi.org/10.1136/bmjsrh-2019-200509
131. Tepper NK, Curtis KM, Cox S, Whiteman MK. Update to U.S. medical eligibility criteria for contraceptive use, 2016: updated recommendations for the use of contraception among women at high risk for HIV infection. MMWR Morb Mortal Wkly Rep 2020;69:405–10. PMID:32271729 https://doi.org/10.15585/mmwr.mm6914a3
132. Phillips SJ, Curtis KM, Polis CB. Effect of hormonal contraceptive methods on HIV disease progression: a systematic review. AIDS 2013;27:787–94. PMID:23135169 https://doi.org/10.1097/QAD.0b013e32835bb672
133. Polis CB, Phillips SJ, Curtis KM. Hormonal contraceptive use and female-to-male HIV transmission: a systematic review of the epidemiologic evidence. AIDS 2013;27:493–505. PMID:23079808 https://doi.org/10.1097/QAD.0b013e32835ad539
134. Phillips SJ, Polis CB, Curtis KM. The safety of hormonal contraceptives for women living with HIV and their sexual partners. Contraception 2016;93:11–6. PMID:26515194 https://doi.org/10.1016/j.contraception.2015.10.002
135. Tagy AH, Saker ME, Moussa AA, Kolgah A. The effect of low-dose combined oral contraceptive pills versus injectable contraceptive (Depot Provera) on liver function tests of women with compensated bilharzial liver fibrosis. Contraception 2001;64:173–6. PMID:11704097 https://doi.org/10.1016/S0010-7824(01)00248-7
136. Pyörälä T, Vähäpassi J, Huhtala M. The effect of lynestrenol and norethindrone on the carbohydrate and lipid metabolism in subjects with gestational diabetes. Ann Chir Gynaecol 1979;68:69–74. PMID:507743
137. Rådberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in gestational diabetic women during contraceptive treatment: effects on glucose tolerance and fatty acid composition of serum lipids. Gynecol Obstet Invest 1982;13:17–29. PMID:7035304 https://doi.org/10.1159/000299480
138. Kjos SL, Peters RK, Xiang A, Thomas D, Schaefer U, Buchanan TA. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280:533–8. PMID:9707143 https://doi.org/10.1001/jama.280.6.533
139. Nelson AL, Le MH, Musherraf Z, Vanberckelaer A. Intermediate-term glucose tolerance in women with a history of gestational diabetes: natural history and potential associations with breastfeeding and contraception. Am J Obstet Gynecol 2008;198:699.e1–8.PMID:18439553 https://doi.org/10.1016/j.ajog.2008.03.029
140. Xiang AH, Kawakubo M, Buchanan TA, Kjos SL. A longitudinal study of lipids and blood pressure in relation to method of contraception in Latino women with prior gestational diabetes mellitus. Diabetes Care 2007;30:1952–8. PMID:17519432 https://doi.org/10.2337/dc07-0180
141. Xiang AH, Kawakubo M, Kjos SL, Buchanan TA. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006;29:613–7. PMID:16505515 https://doi.org/10.2337/diacare.29.03.06.dc05-1940
142. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of Norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynaecol Res 2000;26:17–26. PMID:10761326 https://doi.org/10.1111/j.1447-0756.2000.tb01195.x
143. Lunt H, Brown LJ. Self-reported changes in capillary glucose and insulin requirements during the menstrual cycle. Diabet Med 1996;13:525–30. PMID:8799655 https://doi.org/10.1002/(SICI)1096-9136(199606)13:6<525::AID-DIA123>3.0.CO;2-D
144. Rådberg T, Gustafson A, Skryten A, Karlsson K. Oral contraception in diabetic women. A cross-over study on serum and high density lipoprotein (HDL) lipids and diabetes control during progestogen and combined estrogen/progestogen contraception. Horm Metab Res 1982;14:61–5. PMID:7040192
145. Skouby SO, Mølsted-Pedersen L, Kühl C, Bennet P. Oral contraceptives in diabetic women: metabolic effects of four compounds with different estrogen/progestogen profiles. Fertil Steril 1986;46:858–64. PMID:3781003 https://doi.org/10.1016/S0015-0282(16)49825-0
146. Zapata LB, Paulen ME, Cansino C, Marchbanks PA, Curtis KM. Contraceptive use among women with inflammatory bowel disease: a systematic review. Contraception 2010;82:72–85. PMID:20682145 https://doi.org/10.1016/j.contraception.2010.02.012
147. Whiteman MK, Oduyebo T, Zapata LB, Walker S, Curtis KM. Contraceptive safety among women with cystic fibrosis: a systematic review. Contraception 2016;94:621–9. PMID:27287694 https://doi.org/10.1016/j.contraception.2016.05.016
148. Brunson A, Keegan T, Mahajan A, White R, Wun T. High incidence of venous thromboembolism recurrence in patients with sickle cell disease. Am J Hematol 2019;94:862–70. PMID:31074115 https://doi.org/10.1002/ajh.25508
149. Naik RP, Streiff MB, Haywood C Jr, Segal JB, Lanzkron S. Venous thromboembolism incidence in the Cooperative Study of Sickle Cell Disease. J Thromb Haemost 2014;12:2010–6. PMID:25280124 https://doi.org/10.1111/jth.12744

150. Noubiap JJ, Temgoua MN, Tankeu R, Tochie JN, Wonkam A, Bigna JJ. Sickle cell disease, sickle trait and the risk for venous thromboembolism: a systematic review and meta-analysis. Thromb J 2018;16:27. PMID:30305805 https://doi.org/10.1186/s12959-018-0179-z
151. Ohene-Frempong K, Weiner SJ, Sleeper LA, et al. Cerebrovascular accidents in sickle cell disease: rates and risk factors. Blood 1998;91:288–94. PMID:9414296
152. Anastasilakis AD, Tsourdi E, Makras P, et al. Bone disease following solid organ transplantation: a narrative review and recommendations for management from The European Calcified Tissue Society. Bone 2019;127:401–18. PMID:31299385 https://doi.org/10.1016/j.bone.2019.07.006
153. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Recommendations for the use of antiretroviral drugs during pregnancy and interventions to reduce perinatal HIV transmission in the United States. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/en/guidelines/perinatal/recommendations-arv-drugs-pregnancy-overview
154. Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in adults and adolescents with HIV. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/sites/default/files/guidelines/documents/adult-adolescent-arv/guidelines-adult-adolescent-arv.pdf
155. Aweeka FT, Rosenkranz SL, Segal Y, et al.; NIAID AIDS Clinical Trials Group. The impact of sex and contraceptive therapy on the plasma and intracellular pharmacokinetics of zidovudine. AIDS 2006;20:1833–41. PMID:16954724 https://doi.org/10.1097/01.aids.0000244202.18629.36
156. Kearney BP, Mathias A. Lack of effect of tenofovir disoproxil fumarate on pharmacokinetics of hormonal contraceptives. Pharmacotherapy 2009;29:924–9. PMID:19637945 https://doi.org/10.1592/phco.29.8.924
157. Todd CS, Deese J, Wang M, et al.; FEM-PrEP Study Group. Sino-implant (II) continuation and effect of concomitant tenofovir disoproxil fumarate-emtricitabine use on plasma levonorgestrel concentrations among women in Bondo, Kenya. Contraception 2015;91:248–52. PMID:25459097 https://doi.org/10.1016/j.contraception.2014.10.008
158. Murnane PM, Heffron R, Ronald A, et al.; Partners PrEP Study Team. Pre-exposure prophylaxis for HIV-1 prevention does not diminish the pregnancy prevention effectiveness of hormonal contraception. AIDS 2014;28:1825–30. PMID:24785951 https://doi.org/10.1097/QAD.0000000000000290
159. Kasonde M, Niska RW, Rose C, et al. Bone mineral density changes among HIV-uninfected young adults in a randomised trial of pre-exposure prophylaxis with tenofovir-emtricitabine or placebo in Botswana. PLoS One 2014;9:e90111. PMID:24625530 https://doi.org/10.1371/journal.pone.0090111
160. Callahan R, Nanda K, Kapiga S, et al.; FEM-PrEP Study Group. Pregnancy and contraceptive use among women participating in the FEM-PrEP trial. J Acquir Immune Defic Syndr 2015;68:196–203. PMID:25590272 https://doi.org/10.1097/QAI.0000000000000413
161. Vieira CS, Bahamondes MV, de Souza RM, et al. Effect of antiretroviral therapy including lopinavir/ritonavir or efavirenz on etonogestrel-releasing implant pharmacokinetics in HIV-positive women. J Acquir Immune Defic Syndr 2014;66:378–85. PMID:24798768 https://doi.org/10.1097/QAI.0000000000000189
162. Patel RC, Onono M, Gandhi M, et al. Pregnancy rates in HIV-positive women using contraceptives and efavirenz-based or nevirapine-based antiretroviral therapy in Kenya: a retrospective cohort study. Lancet HIV 2015;2:e474–82. PMID:26520927 https://doi.org/10.1016/S2352-3018(15)00184-8
163. Perry SH, Swamy P, Preidis GA, Mwanyumba A, Motsa N, Sarero HN. Implementing the Jadelle implant for women living with HIV in a resource-limited setting: concerns for drug interactions leading to unintended pregnancies. AIDS 2014;28:791–3. PMID:24401645 https://doi.org/10.1097/QAD.0000000000000177
164. Scarsi KK, Darin KM, Nakalema S, et al. Unintended pregnancies observed with combined use of the levonorgestrel contraceptive implant and efavirenz-based antiretroviral therapy: a three-arm pharmacokinetic evaluation over 48 weeks. Clin Infect Dis 2016;62:675–82. PMID:26646680 https://doi.org/10.1093/cid/civ1001
165. Pyra M, Heffron R, Mugo NR, et al.; Partners in Prevention HSVHIV Transmission Study and Partners PrEP Study Teams. Effectiveness of hormonal contraception in HIV-infected women using antiretroviral therapy. AIDS 2015;29:2353–9. PMID:26544706 https://doi.org/10.1097/QAD.0000000000000827
166. Cohn SE, Park JG, Watts DH, et al.; ACTG A5093 Protocol Team. Depo-medroxyprogesterone in women on antiretroviral therapy: effective contraception and lack of clinically significant interactions. Clin Pharmacol Ther 2007;81:222–7. PMID:17192768 https://doi.org/10.1038/sj.clpt.6100040
167. Nanda K, Amaral E, Hays M, Viscola MA, Mehta N, Bahamondes L. Pharmacokinetic interactions between depot medroxyprogesterone acetate and combination antiretroviral therapy. Fertil Steril 2008;90:965–71. PMID:17880953 https://doi.org/10.1016/j.fertnstert.2007.07.1348
168. Watts DH, Park JG, Cohn SE, et al. Safety and tolerability of depot medroxyprogesterone acetate among HIV-infected women on antiretroviral therapy: ACTG A5093. Contraception 2008;77:84–90. PMID:18226670 https://doi.org/10.1016/j.contraception.2007.10.002
169. Polis CB, Nakigozi G, Ssempijja V, et al. Effect of injectable contraceptive use on response to antiretroviral therapy among women in Rakai, Uganda. Contraception 2012;86:725–30. PMID:22717186 https://doi.org/10.1016/j.contraception.2012.05.001
170. Hubacher D, Liku J, Kiarie J, et al. Effect of concurrent use of anti-retroviral therapy and levonorgestrel sub-dermal implant for contraception on CD4 counts: a prospective cohort study in Kenya. J Int AIDS Soc 2013;16:18448. PMID:23458102 https://doi.org/10.7448/IAS.16.1.18448
171. Myer L, Carter RJ, Katyal M, Toro P, El-Sadr WM, Abrams EJ. Impact of antiretroviral therapy on incidence of pregnancy among HIV-infected women in Sub-Saharan Africa: a cohort study. PLoS Med 2010;7:e1000229. PMID:20161723 https://doi.org/10.1371/journal.pmed.1000229
172. Day S, Graham SM, Masese LN, et al. A prospective cohort study of the effect of depot medroxyprogesterone acetate on detection of plasma and cervical HIV-1 in women initiating and continuing antiretroviral therapy. J Acquir Immune Defic Syndr 2014;66:452–6. PMID:24798764 https://doi.org/10.1097/QAI.0000000000000187
173. DuBois BN, Atrio J, Stanczyk FZ, Cherala G. Increased exposure of norethindrone in HIV+ women treated with ritonavir-boosted atazanavir therapy. Contraception 2015;91:71–5. PMID:25245190 https://doi.org/10.1016/j.contraception.2014.08.009

174. Luque AE, Cohn SE, Park JG, et al. Depot medroxyprogesterone acetate in combination with a twice-daily lopinavir-ritonavir-based regimen in HIV-infected women showed effective contraception and a lack of clinically significant interactions, with good safety and tolerability: results of the ACTG 5283 study. Antimicrob Agents Chemother 2015;59:2094–101. PMID:25624326 https://doi.org/10.1128/AAC.04701-14
175. Odlind V, Olsson SE. Enhanced metabolism of levonorgestrel during phenytoin treatment in a woman with Norplant implants. Contraception 1986;33:257–61. PMID:3087695 https://doi.org/10.1016/0010-7824(86)90018-1
176. Schindlbeck C, Janni W, Friese K. Failure of Implanon contraception in a patient taking carbamazepin for epilepsia. Arch Gynecol Obstet 2006;273:255–6. PMID:16208481 https://doi.org/10.1007/s00404-005-0064-4
177. Shane-McWhorter L, Cerveny JD, MacFarlane LL, Osborn C. Enhanced metabolism of levonorgestrel during phenobarbital treatment and resultant pregnancy. Pharmacotherapy 1998;18:1360–4. PMID:9855340 https://doi.org/10.1002/j.1875-9114.1998.tb03161.x
178. Gaffield ME, Culwell KR, Lee CR. The use of hormonal contraception among women taking anticonvulsant therapy. Contraception 2011;83:16–29. PMID:21134499 https://doi.org/10.1016/j.contraception.2010.06.013
179. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7. PMID:16146436 https://doi.org/10.1111/j.1528-1167.2005.10105.x
180. Berry-Bibee ENKM, Kim MJ, Simmons KB, et al. Drug interactions between hormonal contraceptives and psychotropic drugs: a systematic review. Contraception 2016;94:650–67. PMID:27444984 https://doi.org/10.1016/j.contraception.2016.07.011
181. Berry-Bibee ENKM, Kim MJ, Tepper NK, Riley HE, Curtis KM. Co-administration of St. John's wort and hormonal contraceptives: a systematic review. Contraception 2016;94:668–77. PMID:27444983 https://doi.org/10.1016/j.contraception.2016.07.010

Appendix D: Classifications for Combined Hormonal Contraceptives

Combined hormonal contraceptives (CHCs) include combined oral contraceptives (COCs) (containing a progestin plus ethinyl estradiol [EE] ≤35 μg, estradiol valerate, or estetrol); combined transdermal patches (levonorgestrel/EE or norelgestromin/EE); and combined vaginal rings (etonogestrel/EE or segesterone acetate/EE) (Box D1) (Table D1). Limited information is available about the safety of COCs with estradiol valerate or estetrol, combined transdermal patches, and combined vaginal rings among users with specific medical conditions. Evidence indicates that estradiol valerate and estetrol COCs, combined transdermal patches, and combined vaginal rings provide comparable safety and pharmacokinetic profiles to EE-containing COCs with similar hormone formulations (*1*–*33*). Pending further studies, the evidence available for recommendations about EE-containing COCs applies to the recommendations for estradiol valerate and estetrol COCs, the combined transdermal patch, and vaginal rings. Therefore, the estradiol valerate and estetrol COCs, the patches, and the rings should have the same categories as EE-containing COCs, except where noted. The assigned categories should be considered a preliminary best judgment, which will be reevaluated as new data become available.

COCs, patches, and rings do not protect against sexually transmitted infections (STIs), including HIV infection, and patients using CHCs should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*34*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*34*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*35*).

BOX D1. Categories for classifying combined hormonal contraceptives

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE D1. Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
Personal Characteristics and Reproductive History		
Pregnancy	NA	**Clarification:** Use of CHCs is not required. No known harm to the patient, the course of pregnancy, or the fetus occurs if CHCs are inadvertently used during pregnancy.
Age		
a. Menarche to <40 years	1	**Evidence:** Evidence is inconsistent about whether CHC use affects fracture risk (*36–47*), although three recent studies demonstrate no effect (*36,37,47*). CHC use might decrease BMD in adolescents, especially in those choosing very low-dose formulations (COCs containing <30 μg ethinyl estradiol) (*48–61*). CHC use has little to no effect on BMD in premenopausal women (*62–76*) and might preserve bone mass in those who are perimenopausal (*77–85*). BMD is a surrogate marker for fracture risk that might not be valid for premenopausal women and therefore might not accurately predict current or future (postmenopausal) fracture risk (*86–88*). **Comment:** The risk for cardiovascular disease increases with age and might increase with CHC use. In the absence of other adverse clinical conditions, CHCs can be used until menopause.
b. ≥40 years	2	
Parity		
a. Nulliparous	1	—
b. Parous	1	—
Breastfeeding		
a. <21 days postpartum	4	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*89*) or up to age 2 years or longer (*90*). **Evidence (breastfeeding):** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*91*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without risk factors for breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
b. 21 to <30 days postpartum		
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	3	**Clarification:** For persons with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*89*) or up to age 2 years or longer (*90*). **Evidence (breastfeeding):** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*91*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without risk factors for breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
ii. Without other risk factors for VTE	3	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*89*) or up to age 2 years or longer (*90*). **Evidence (breastfeeding):** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*91*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
c. 30–42 days postpartum		
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	3	**Clarification:** For persons with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*89*) or up to age 2 years or longer (*90*). **Evidence (breastfeeding):** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*91*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
ii. Without other risk factors for VTE	2	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*89*) or up to age 2 years or longer (*90*). **Evidence (breastfeeding):** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*91*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
d. >42 days postpartum	2	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for breastfeeding parent and infant. The U.S. Dietary Guidelines for Americans and American Academy of Pediatrics recommend that infants be exclusively breastfed for about the first 6 months with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*89*) or up to age 2 years or longer (*90*). **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*91*). **Comment:** Risk factors for breastfeeding difficulties include previous breastfeeding difficulties, certain medical conditions, certain perinatal complications, and preterm birth. For all breastfeeding persons, with or without breastfeeding difficulties, discussions about contraception should include information about risks, benefits, and alternatives.
Postpartum (nonbreastfeeding)		
a. <21 days postpartum	4	**Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*). Risk for pregnancy during the first 21 days postpartum is very low but increases after that point; ovulation before first menses is common (*98*).
b. 21–42 days postpartum		
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2 postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	3	**Clarification:** For persons with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*).
ii. Without other risk factors for VTE	2	**Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*92*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*93–97*).
c. >42 days postpartum	1	—
Postabortion (spontaneous or induced)		
a. First trimester abortion		**Clarification:** CHCs may be started immediately after abortion completion or at time of medication abortion initiation. **Evidence:** Evidence suggests that there is no increased risk for adverse events when CHCs are initiated after first trimester procedural or medication abortion (immediately or delayed) (*99*). Immediate initiation of COCs after first trimester procedural or medication abortion did not cause clinically significant changes in coagulation parameters compared with placebo, a hormonal IUD, a nonhormonal contraceptive method, or delayed COC initiation (*100*).
i. Procedural (surgical)	1	
ii. Medication	1	
iii. Spontaneous abortion with no intervention	1	
b. Second trimester abortion		**Clarification:** CHCs may be started immediately after abortion completion or at time of medication abortion initiation. **Evidence:** Limited evidence suggests that there is no increased risk for adverse events when CHCs are initiated after second trimester procedural abortion (immediately or delayed) (*99*).
i. Procedural (surgical)	1	
ii. Medication	1	
iii. Spontaneous abortion with no intervention	1	
c. Immediate postseptic abortion	1	**Clarification:** CHCs may be started immediately after abortion completion or at time of medication abortion initiation.
Past ectopic pregnancy	1	**Comment:** The risk for future ectopic pregnancy is increased among those who have had an ectopic pregnancy in the past. CHCs protect against pregnancy in general, including ectopic gestation.
History of pelvic surgery	1	—
Smoking		**Evidence:** COC users who smoked were at increased risk for cardiovascular diseases, especially myocardial infarction, compared with those who did not smoke. Studies also demonstrated an increased risk for myocardial infarction with increasing number of cigarettes smoked per day (*101–113*).
a. Age <35 years	2	
b. Age ≥35 years		
i. <15 cigarettes per day	3	
ii. ≥15 cigarettes per day	4	

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
Obesity		**Clarification:** Risk for thrombosis increases with multiple risk factors, such as obesity, older age (e.g., ≥40 years), diabetes, smoking, family history of thrombosis, and dyslipidemia. When a person has multiple risk factors, any of which alone would increase risk for thrombosis, use of CHCs might increase thrombosis risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two category 2 risk factors might not necessarily warrant a higher category. **Evidence:** Although the absolute risk for VTE in healthy women of reproductive age is small, COC use and higher BMI independently increase risk for VTE, with the greatest relative risks among those with both risk factors. From a systematic review, COC users with obesity consistently had a relative risk for VTE of 5–8 times that of nonusers with obesity (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Research examining the interaction between COCs and BMI on VTE risk is limited, particularly for those in the highest BMI categories (BMI ≥35 kg/m^2). Comparative studies on the risk for VTE among contraceptive patch or ring users by weight or BMI were not identified (*114–116*). Limited evidence suggests that COC users with obesity do not have a higher risk for acute myocardial infarction or stroke than do nonusers with obesity (*114*) (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Limited evidence suggests that effectiveness of certain COC formulations might decrease with increasing BMI; however the observed reductions in effectiveness are minimal and evidence is conflicting (*117–124*). Effectiveness of the patch might be reduced in women with BMI ≥30 kg/m^2 or weight >90 kg (*125*).
a. BMI ≥30 kg/m^2	2	
b. Menarche to <18 years and BMI ≥30 kg/m^2	2	
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent laparoscopic placement of an adjustable gastric band (*126*).
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	COCs: 3 Patch and ring: 1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent a biliopancreatic diversion; however, evidence from pharmacokinetic studies reported conflicting results of oral contraceptive effectiveness among women who underwent a jejunoileal bypass (*126*). **Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications, such as long-term diarrhea or vomiting.
Surgery		
a. Minor surgery without immobilization	1	—
b. Major surgery		
ii. Without prolonged immobilization	2	—
i. With prolonged immobilization	4	—
Cardiovascular Disease		
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	3/4	**Clarification:** When a person has multiple major risk factors, any of which alone would substantially increase risk for cardiovascular disease, use of CHCs might increase risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two category 2 risk factors might not necessarily warrant a higher category. **Clarification:** The recommendations apply to known pre-existing medical conditions or characteristics. Few if any screening tests are needed before initiation of contraception. See U.S. *SPR* (https://www.cdc.gov/contraception/hcp/usspr/) (*127*).
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Adequately controlled hypertension	3	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive. **Clarification:** Persons adequately treated for hypertension are at reduced risk for acute myocardial infarction and stroke compared with untreated persons. Although no data exist, CHC users with adequately controlled and monitored hypertension should be at reduced risk for acute myocardial infarction and stroke compared with untreated hypertensive CHC users. **Evidence:** Among women with hypertension, COC users were at higher risk than nonusers for stroke, acute myocardial infarction, and peripheral arterial disease (*101,103,110–113,128–142*). Discontinuation of COCs in women with hypertension might improve blood pressure control (*143*).
b. Elevated blood pressure levels (properly taken measurements)		**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a person as hypertensive. **Evidence:** Among women with hypertension, COC users were at higher risk than nonusers for stroke, acute myocardial infarction, and peripheral arterial disease (*101,103,110–113,128–142*). Discontinuation of COCs in women with hypertension might improve blood pressure control (*143*).
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	3	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	4	
c. Vascular disease	4	

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	2	**Evidence:** Women with a history of high blood pressure in pregnancy who also used COCs had a higher risk for myocardial infarction and VTE than did COC users who did not have a history of high blood pressure during pregnancy. The absolute risks for acute myocardial infarction and VTE in this population remained small (*112,129,141,142,144–150*).
Deep venous thrombosis/Pulmonary embolism This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Current or history of DVT/PE, receiving anticoagulant therapy (therapeutic dose) (e.g., acute DVT/PE or long-term therapeutic dose)	3	**Clarification**: Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. CHCs can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis. **Clarification:** When a patient discontinues therapeutic dose of anticoagulant therapy, careful consideration should be given to transitioning from CHCs to a progestin-only or nonhormonal method, if acceptable to the patient. **Evidence**: Limited evidence was identified on use of CHCs among women with DVT/PE receiving anticoagulant therapy (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). In one study among women with a history of acute VTE currently receiving therapeutic anticoagulant therapy (i.e., rivaroxaban or enoxaparin/vitamin K antagonist [warfarin or acenocoumarol]), the incidence of recurrent VTE was similar among estrogen users (CHC or estrogen-only pills), POC users, and women not on hormonal therapy (*151*).
b. History of DVT/PE, receiving anticoagulant therapy (prophylactic dose)		**Clarification**: Persons using anticoagulant therapy are at risk for gynecologic complications of therapy, such as heavy or prolonged bleeding and hemorrhagic ovarian cysts. CHCs can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	4	
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	
c. History of DVT/PE, not receiving anticoagulant therapy		
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	4	—
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	—
d. Family history (first-degree relatives)	2	**Comment:** Certain conditions that increase the risk for DVT/PE are heritable.
Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	4	**Clarification**: Routine screening in the general population before contraceptive initiation is not recommended. **Clarification**: If a person has current or history of DVT/PE, see recommendations for DVT/PE. **Clarification**: Classification of antiphospholipid syndrome includes presence of a clinical feature (e.g., thrombosis or obstetric morbidity) and persistently abnormal antiphospholipid antibody test on two or more occasions at least 12 weeks apart (*152*). **Evidence:** Among women with factor V Leiden mutation, prothrombin gene mutation, antithrombin deficiency, and protein C deficiency, COC users had an increased risk for venous and arterial thrombosis compared with nonusers. Evidence was inconsistent on risk for thrombosis among women with protein S deficiency using COCs. No evidence was identified on COC use among persons with antiphospholipid syndrome (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Superficial venous disorders		
a. Varicose veins	1	**Evidence:** One study suggested that among women with varicose veins, the rate of VTE and superficial venous thrombosis was higher in oral contraceptive users compared with nonusers; however, statistical significance was not reported and the number of events was small (*153*).
b. Superficial venous thrombosis (acute or history)	3	**Clarification:** Superficial venous thrombosis might be associated with an increased risk for VTE. If a person has risk factors for concurrent DVT (e.g., thrombophilia or cancer) or has current or history of DVT, see recommendations for DVT/PE. Superficial venous thrombosis associated with a peripheral intravenous catheter is less likely to be associated with additional thrombosis and use of CHCs may be considered. **Evidence:** One study demonstrated that among women with superficial venous thrombosis, the risk for VTE was higher in oral contraceptive users compared with nonusers (*153*).

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TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	4	—
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	4	—
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Uncomplicated	2	—
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	4	**Comment:** Among persons with valvular heart disease, CHC use might further increase the risk for arterial thrombosis; persons with complicated valvular heart disease are at greatest risk.
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		**Evidence:** No direct evidence exists about the safety of CHCs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension and transient ischemic attack in women with cardiac disease using COCs. No cases of heart failure were reported (*154*). **Comment:** COCs might increase fluid retention in healthy persons; fluid retention might worsen heart failure in persons with peripartum cardiomyopathy. COCs might induce cardiac arrhythmias in healthy persons; persons with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: no limitation of activities or slight, mild limitation of activity) (*155*)		
i. <6 months	4	
ii. ≥6 months	3	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: marked limitation of activity or should be at complete rest) (*155*)	4	
Renal Disease		
Chronic kidney disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Current nephrotic syndrome	4	**Evidence:** No direct evidence was identified on CHC use among persons with CKD with current nephrotic syndrome (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with severe CKD or nephrotic syndrome are at higher risk for thrombosis than the general population (*156–158*). Use of CHCs might further elevate risk for thrombosis among those with CKD with current nephrotic syndrome. **Comment:** A person might have CKD without current nephrotic syndrome but might have other conditions often associated with CKD (e.g., diabetes, hypertension, and SLE). See recommendations for other conditions if they apply.
b. Hemodialysis	4	**Evidence:** No direct evidence was identified on CHC use among persons with CKD on hemodialysis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with CKD on dialysis are at higher risk for thrombosis than the general population (*156–158*). Use of CHCs might further elevate risk for thrombosis among those with CKD on dialysis. **Comment:** A person might have CKD without hemodialysis, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, and SLE). See recommendations for other conditions if they apply.
c. Peritoneal dialysis	4	**Evidence:** No direct evidence was identified on CHC use among persons with CKD on peritoneal dialysis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). Persons with CKD on dialysis are at higher risk for thrombosis than the general population (*156–158*). Use of CHCs might further elevate risk for thrombosis among those with CKD. **Comment:** A person might have CKD without peritoneal dialysis, but might have other conditions often associated with CKD (e.g., diabetes, hypertension, and SLE). See recommendations for other conditions if they apply.
Rheumatic Diseases		
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Positive (or unknown) antiphospholipid antibodies	4	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*159–177*). **Evidence:** Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*178,179*).
b. Severe thrombocytopenia	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*159–177*).

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
c. Immunosuppressive therapy	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*159–177*).
d. None of the above	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for persons with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors (*159–177*).
Rheumatoid arthritis		**Evidence:** Limited evidence demonstrates no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives, progesterone, or estrogen (*180*).
a. Not receiving immunosuppressive therapy	2	
b. Receiving immunosuppressive therapy	2	
Neurologic Conditions		
Headaches		
a. Nonmigraine (mild or severe)	1	**Clarification:** Classification depends on accurate diagnosis of those severe headaches that are migraines and those headaches that are not, as well as diagnosis of ever experiencing aura. Aura is a specific focal neurologic symptom. For more information about headache classification see the International Headache Society's *International Classification of Headache Disorders, 3rd ed.* (https://ichd-3.org) (*181*). Any new headaches or marked changes in headaches should be evaluated.
b. Migraine		**Clarification:** Classification depends on accurate diagnosis of those severe headaches that are migraines and those headaches that are not, as well as diagnosis of ever experiencing aura. Aura is a specific focal neurologic symptom. For more information about headache classification see the International Headache Society's *International Classification of Headache Disorders, 3rd ed.* (https://ichd-3.org) (*181*). Any new headaches or marked changes in headaches should be evaluated. **Clarification:** Classification is for persons without any other risk factors for stroke (e.g., age, hypertension, and smoking). **Evidence:** Among women with migraine, oral contraceptive use is associated with about a threefold increased risk for ischemic stroke compared with nonuse, although most studies did not specify migraine type or oral contraceptive formulation. The only study to examine migraine type found that the risk for ischemic stroke among women with migraine with aura was increased to a similar level among both oral contraceptive users and nonusers, compared with women without migraine (*182*). The risk for ischemic stroke is increased among women using COCs, compared with women not using COCs (*101*,*183*). The risk for ischemic stroke is also increased among women with migraine with aura, compared with women without migraine (*184–186*). One older meta-analysis found that migraine without aura was associated with an increased risk for ischemic stroke, while two more recent meta-analyses did not find such an association (*184–186*). **Comment:** Menstrual migraine is a subtype of migraine without aura. For more information, see the International Headache Society's *International Classification of Headache Disorders, 3rd ed.* (https://ichd-3.org) (*181*).
i. Without aura (includes menstrual migraine)	2	
ii. With aura	4	
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	**Clarification:** If a person is taking anticonvulsants, see recommendations for Drug Interactions. Certain anticonvulsants lower COC effectiveness. The extent to which patch or ring use is similar to COC use in this regard remains unclear.
Multiple sclerosis		**Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with multiple sclerosis does not worsen the clinical course of disease (*187*). **Comment:** No data exist that evaluate the increased risk for VTE among persons with multiple sclerosis using CHCs. However, persons with multiple sclerosis are at higher risk for VTE than those without multiple sclerosis.
a. Without prolonged immobility	1	
b. With prolonged immobility	3	
Depressive Disorders		
Depressive disorders	1	**Clarification**: If a person is receiving psychotropic medications or St. John's wort, see recommendations for Drug Interactions. **Evidence**: COC use was not associated with increased depressive symptoms in women with depression or scoring above threshold levels on a validated depression screening instrument compared with baseline or with nonusers with depression. One small study of women with bipolar disorder found that oral contraceptives did not significantly change mood across the menstrual cycle (*188*).
Reproductive Tract Infections and Disorders		
Vaginal bleeding patterns		
a. Irregular pattern without heavy bleeding	1	**Comment:** Irregular menstrual bleeding patterns are common among healthy persons.
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	1	**Clarification:** Unusually heavy bleeding should raise the suspicion of a serious underlying condition. **Evidence:** A Cochrane Collaboration Review identified one RCT evaluating the effectiveness of COC use compared with naproxen and danazol in treating menorrhagia. Women with menorrhagia did not report worsening of the condition or any adverse events related to COC use (*189*).
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	2	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. **Comment:** No conditions that cause vaginal bleeding will be worsened in the short-term by use of CHCs.

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
Endometriosis	1	**Evidence:** A Cochrane Collaboration Review identified one RCT evaluating the effectiveness of COC use compared with a gonadotropin-releasing hormone analog in treating the symptoms of endometriosis. Women with endometriosis did not report worsening of the condition or any adverse events related to COC use (*190*).
Benign ovarian tumors (including cysts)	1	—
Severe dysmenorrhea	1	**Evidence:** Risk for side effects with COC use was not higher among women with dysmenorrhea than among women not using COCs. Certain COC users had a reduction in pain and bleeding (*191,192*).
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that persons are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** After molar pregnancy evacuation, the balance of evidence found COC use did not increase the risk for postmolar trophoblastic disease, and β–hCG levels regressed more rapidly in certain COC users than in nonusers (193). Limited evidence suggests that use of COCs during chemotherapy does not significantly affect the regression or treatment of postmolar trophoblastic disease compared with women who used a nonhormonal contraceptive method or DMPA during chemotherapy (*193*).
a. Suspected gestational trophoblastic disease (immediate postevacuation)		
i. Uterine size first trimester	1	
ii. Uterine size second trimester	1	
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)		
i. Undetectable or nonpregnant β-hCG levels	1	
ii. Decreasing β-hCG levels	1	
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	1	
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	1	
Cervical ectropion	1	**Comment:** Cervical ectropion is not a risk factor for cervical cancer, and restriction of CHC use is unnecessary.
Cervical intraepithelial neoplasia	2	**Evidence:** Among women with persistent human papillomavirus infection, long-term COC use (≥5 years) might increase the risk for carcinoma in situ and invasive carcinoma (*194*). Limited evidence on women with low-grade squamous intraepithelial lesions found use of the vaginal ring did not worsen the condition (*9*).
Cervical cancer (awaiting treatment)	2	**Comment:** Theoretical concern exists that CHC use might affect prognosis of the existing disease. While awaiting treatment, persons may use CHCs. In general, treatment of this condition can render a person infertile.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Undiagnosed mass	2	**Clarification:** Evaluation of mass should be pursued as early as possible.
b. Benign breast disease	1	—
c. Family history of cancer	1	**Evidence:** Women with breast cancer susceptibility genes (e.g., *BRCA1* and *BRCA2*) have a higher baseline risk for breast cancer than women without these genes. The baseline risk for breast cancer also is higher among women with a family history of breast cancer than among those who do not have such a history. However, evidence does not suggest that the increased risk for breast cancer among women with either a family history of breast cancer or breast cancer susceptibility genes is modified by the use of COCs (*195–212*).
d. Breast cancer		**Comment:** Breast cancer is a hormonally sensitive tumor, and the prognosis for persons with current or recent breast cancer might worsen with CHC use.
i. Current	4	
ii. Past and no evidence of current disease for 5 years	3	
Endometrial hyperplasia	1	—
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	**Comment:** COC use reduces the risk for endometrial cancer; whether patch or ring use reduces the risk for endometrial cancer is not known. While awaiting treatment, patients may use CHCs. In general, treatment of this condition can render a person infertile.
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	**Comment:** COC use reduces the risk for ovarian cancer; whether patch or ring use reduces the risk for ovarian cancer is not known. While awaiting treatment, patients may use CHCs. In general, treatment of this condition can render a person infertile.
Uterine fibroids	1	**Comment:** COCs do not appear to cause growth of uterine fibroids, and patch and ring also are not expected to cause growth.
Pelvic inflammatory disease		**Comment:** COCs might reduce the risk for PID among persons with STIs but do not protect against HIV infection or lower genital tract STIs. Whether use of patch or ring reduces the risk for PID among persons with STIs is unknown; however, they do not protect against HIV infection or lower genital tract STIs.
a. Current PID	1	
b. Past PID		
i. With subsequent pregnancy	1	
ii. Without subsequent pregnancy	1	
Sexually transmitted infections		
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	1	—
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	—
c. Other factors related to STIs	1	—

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
HIV		
High risk for HIV infection	1	**Evidence:** Low-to-moderate-quality evidence from 11 observational studies suggested no association between COC use (it was assumed that studies that did not specify oral contraceptive type examined mostly, if not exclusively, COC use) and HIV acquisition. No studies of patch or ring were identified (*213,214*).
HIV infection For persons with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	**Clarification:** Drug interactions might exist between hormonal contraceptives and ARV drugs; see recommendations for Drug Interactions. **Evidence:** Overall, evidence does not support an association between COC use and progression of HIV. Limited direct evidence does not support an association between COC use and transmission of HIV to noninfected partners; studies measuring genital viral shedding as a proxy for infectivity have had mixed results. Studies measuring whether hormonal contraceptive methods affect plasma HIV viral load generally have found no effect (*215–217*).
Other Infections		
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. Uncomplicated	1	**Evidence:** Among women with uncomplicated schistosomiasis, COC use had no adverse effects on liver function (*218–224*).
b. Fibrosis of the liver (if severe, see recommendations for Cirrhosis)	1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		**Clarification:** If a person is taking rifampin, see recommendations for Drug Interactions. Rifampin is likely to decrease COC effectiveness. The extent to which patch or ring use is similar to COC use in this regard remains unclear.
a. Nonpelvic	1	
b. Pelvic	1	
Malaria	1	—
Endocrine Conditions		
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. History of gestational disease	1	**Evidence:** The development of non–insulin-dependent diabetes in women with a history of gestational diabetes is not increased by use of COCs (*225–232*). Likewise, lipid levels appear to be unaffected by COC use (*233–235*).
b. Nonvascular disease		**Evidence:** Among women with insulin-dependent or non–insulin-dependent diabetes, COC use had limited effect on daily insulin requirements and no effect on long-term diabetes control (e.g., glycosylated hemoglobin levels) or progression to retinopathy. Changes in lipid profile and hemostatic markers were limited, and most changes remained within normal values (*236–245*).
i. Non-insulin dependent	2	—
ii. Insulin dependent	2	—
c. Nephropathy, retinopathy, or neuropathy	3/4	**Clarification:** The category should be assessed according to the severity of the condition.
d. Other vascular disease or diabetes of >20 years' duration	3/4	**Clarification:** The category should be assessed according to the severity of the condition.
Thyroid disorders		
a. Simple goiter	1	—
b. Hyperthyroid	1	—
c. Hypothyroid	1	—
Gastrointestinal Conditions		
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	2/3	**Clarification:** For persons with mild IBD and with no other risk factor for VTE, the benefits of CHC use generally outweigh the risks (category 2). However, for persons with IBD who are at increased risk for VTE (e.g., those with active or extensive disease, surgery, immobilization, corticosteroid use, vitamin deficiencies, or fluid depletion), the risks of CHC use generally outweigh the benefits (category 3). **Evidence:** Risk for disease relapse was not significantly higher among women with IBD using oral contraceptives (most studies did not specify type) than among nonusers (*246*). Absorption of COCs among women with mild ulcerative colitis and no or small ileal resections was similar to the absorption among healthy women (*246*). Findings might not apply to women with Crohn's disease or more extensive bowel resections. No data exist that evaluate the increased risk for VTE among women with IBD using CHCs. However, women with IBD are at higher risk than unaffected women for VTE (*246*).
Gallbladder disease		**Comment:** CHCs might cause a small increased risk for gallbladder disease. CHCs might worsen existing gallbladder disease.
a. Asymptomatic	2	
b. Symptomatic		
i. Current	3	
ii. Treated by cholecystectomy	2	
iii. Medically treated	3	

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC		Clarification/Evidence/Comment
History of cholestasis			
a. Pregnancy related		2	**Comment:** History of pregnancy-related cholestasis might predict an increased risk for COC-related cholestasis.
b. Past COC related		3	**Comment:** History of COC-related cholestasis predicts an increased risk with subsequent COC use.
Viral hepatitis	Initiation	Continuation	—
a. Acute or flare	3/4	2	**Clarification (initiation):** The category should be assessed according to the severity of the condition. **Evidence:** Limited evidence was identified on COC use among persons with acute viral hepatitis. Data suggest that in women with chronic viral hepatitis, COC use does not increase the risk or severity of fibrosis, nor does it increase the risk for hepatocellular carcinoma. For women with chronic viral hepatitis, COC use does not appear to trigger severe liver dysfunction (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Hepatic metabolism of exogenous hormones might be impaired in persons with liver dysfunction, which could lead to increased estrogen levels in circulation and estrogen-related side effects and adverse events (e.g., thrombosis).
b. Chronic	1	1	**Evidence:** Data suggest that in women with chronic viral hepatitis, COC use does not increase the risk or severity of fibrosis, nor does it increase the risk for hepatocellular carcinoma. For women with chronic viral hepatitis, COC use does not appear to trigger severe liver dysfunction (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Cirrhosis Decompensated cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Compensated (normal liver function)		1	**Evidence**: No direct evidence was identified on CHC use among persons with compensated cirrhosis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Decompensated (impaired liver function)		4	**Evidence**: No direct evidence was identified on CHC use among persons with decompensated cirrhosis (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). **Comment:** Hepatic metabolism of exogenous hormones might be impaired in persons with liver dysfunction, which could lead to increased estrogen levels in circulation and estrogen-related side effects and adverse events (e.g., thrombosis). Any estrogen-related hepatotoxicity might be less tolerated in persons with existing liver dysfunction.
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 3).			
a. Benign			
i. Focal nodular hyperplasia		2	**Evidence:** Limited evidence suggests that COC use does not influence either progression or regression of focal nodular hyperplasia (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
ii. Hepatocellular adenoma		4	**Evidence:** Evidence suggests that COC use is associated with progression of hepatocellular adenoma growth, while COC discontinuation is associated with stability or regression (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Malignant (hepatocellular carcinoma)		4	**Evidence:** No direct evidence was identified on CHC use among persons with hepatocellular carcinoma (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Respiratory Conditions			
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		1	**Clarification:** Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for persons with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions. **Clarification:** Certain drugs to treat cystic fibrosis (e.g., lumacaftor) might reduce effectiveness of hormonal contraceptives, including oral, injectable, transdermal, and implantable contraceptives. **Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with cystic fibrosis is not associated with worsening of disease severity. Very limited evidence suggests that cystic fibrosis does not impair the effectiveness of hormonal contraception (*247*).
Hematologic Conditions			
Thalassemia		1	**Comment:** Anecdotal evidence from countries where thalassemia is prevalent indicates that COC use does not worsen the condition.
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		4	**Evidence**: Persons with sickle cell disease are at higher risk for stroke and venous thrombosis than the general population (*248–251*). CHC use might further elevate risk for thrombosis among persons with sickle cell disease, but evidence is limited (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Iron deficiency anemia		1	**Comment:** CHC use might decrease menstrual blood loss.

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
Solid Organ Transplantation		
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).		
a. No graft failure	2	**Clarification:** Persons with transplant due to Budd-Chiari syndrome should not use CHCs because of the increased risk for thrombosis. **Evidence:** Limited evidence among CHC users indicated no adverse events and no overall changes in biochemical parameters (e.g., blood pressure, cholesterol) and no pregnancies (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). However, one study reported discontinuations of COC use in two (8%) of 26 women as a result of serious medical complications, including acute graft rejection (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
b. Graft failure	4	**Evidence:** Limited evidence among CHC users indicated no adverse events and no overall changes in biochemical parameters (e.g., blood pressure, cholesterol) and no pregnancies (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516). However, one study reported discontinuations of COC use in two (8%) of 26 women as a result of serious medical complications, including acute graft rejection (Supplementary Appendix, https://stacks.cdc.gov/view/cdc/156516).
Drug Interactions		
Antiretrovirals used for prevention (PrEP) or treatment of HIV infection		**Comment:** These recommendations generally are for ARV agents used alone. However, most persons receiving ARV therapy are using multiple drugs in combination. In general, whether interactions between ARVs and hormonal contraceptives differ when ARVs are given alone or in combination is unknown.
See the following guidelines for the most up-to-date recommendations on drug-drug interactions between hormonal contraception and ARVs: 1) Recommendations for the Use of Antiretroviral Drugs During Pregnancy and Interventions to Reduce Perinatal HIV Transmission in the United States (https://clinicalinfo.hiv.gov/en/guidelines/perinatal/prepregnancy-counseling-childbearing-age-overview?view=full#table-3) (*252*) and 2) Guidelines for the Use of Antiretroviral Agents in Adults and Adolescents with HIV (https://clinicalinfo.hiv.gov/en/guidelines/hiv-clinical-guidelines-adult-and-adolescent-arv/drug-interactions-overview?view=full) (*253*).		
a. Nucleoside reverse transcriptase inhibitors (NRTIs)		
i. Abacavir (ABC)	1	**Evidence:** NRTIs do not appear to have significant risk for interactions with hormonal contraceptive methods (*254–259*).
ii. Tenofovir (TDF)	1	
iii. Zidovudine (AZT)	1	
iv. Lamivudine (3TC)	1	
v. Didanosine (DDI)	1	
vi. Emtricitabine (FTC)	1	
vii. Stavudine (D4T)	1	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)		
i. Efavirenz (EFV)	2	**Clarification:** Evidence suggests drug interactions between EFV and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive. **Evidence:** Two studies suggested that pregnancy rates might be higher among women using COCs and EFV compared with COCs alone, although one study found no difference in pregnancy rates (*260–262*). Two studies found conflicting results on ovulations in women receiving COCs and EFV compared with EFV alone (*263,264*). Two pharmacokinetic studies demonstrated decreases in ethinyl estradiol and progestin concentrations in women receiving COCs and EFV compared with COCs alone (*264,265*). Pharmacokinetic studies demonstrated generally no changes in EFV concentrations with concomitant COC use (*264–266*).
ii. Etravirine (ETR)	1	**Evidence:** One study demonstrated no clinically relevant pharmacokinetic or pharmacodynamic changes in women using COCs and ETR compared with COCs alone (*267*).
iii. Nevirapine (NVP)	1	**Evidence:** Five studies found no significant differences in pregnancy rates among women using COCs and NVP compared with women using COCs alone (*260–262,266,268*). Three studies reported no ovulations among women receiving COCs and NVP (*263,268,269*). Two pharmacokinetic studies demonstrated decreased concentrations of ethinyl estradiol and progestin among women using COCs and NVP compared with COCs alone, and one study found no change in contraceptive hormone concentrations (*263,269,270*). Pharmacokinetic studies demonstrated generally no changes in NVP concentrations with concomitant COC use (*263,270,271*).
iv. Rilpivirine (RPV)	1	**Evidence:** One study demonstrated no clinical significant pharmacokinetic changes or adverse events in women using COCs and RPV compared with COCs alone (*272*).
c. Ritonavir-boosted protease inhibitors		
i. Ritonavir-boosted atazanavir (ATV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One pharmacokinetic study demonstrated decreased estrogen but increased progestin concentrations in women using COCs and ATV/r compared with COCs alone (*273*).
ii. Ritonavir-boosted darunavir (DRV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One pharmacokinetic study demonstrated no change in follicle-stimulating hormone or luteinizing hormone but decreases in ethinyl estradiol and norethindrone in women using COCs with DRV/r compared with COCs alone (*274*).

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
iii. Ritonavir-boosted fosamprenavir (FPV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** Information from the package label states that both ethinyl estradiol and norethindrone concentrations decreased with concurrent administration of COCs and FPV/r (*275*).
iv. Ritonavir-boosted lopinavir (LPV/r)	1	**Evidence:** One study demonstrated a nonsignificant increase in pregnancy rates among women using COCs and LPV/r compared with COCs alone (*260*). One study demonstrated no ovulations in women using the combined hormonal patch and LPV/r compared with combined hormonal patch alone; ethinyl estradiol concentrations for COC and patch users decreased but norelgestromin concentrations increased with use of the patch (*276*).
v. Ritonavir-boosted saquinavir (SQV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One pharmacokinetic study demonstrated no change in SQV concentrations in women using COC and SQV compared with COCs alone (*277*).
iv. Ritonavir-boosted tipranavir (TPV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** Information from the package label states that ethinyl estradiol concentrations decrease but norethindrone concentrations increased with concurrent administration of COCs and TPV/r (*278*).
d. Protease inhibitors without ritonavir		
i. Atazanavir (ATV)	2	**Clarification:** Theoretical concern exists that increased levels of ethinyl estradiol because of interactions with ATV might increase the risk for adverse events. **Evidence:** Information from the package label states that there are inconsistent changes in ethinyl estradiol concentrations and increases in progestin concentrations with concurrent administration of two different COCs and ATV (*279*). **Comment:** When ATV is administered with cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels might vary when combined with other ARVs.
ii. Fosamprenavir (FPV)	3	**Clarification:** Concern exists that interactions between FPV and hormonal contraceptives leading to decreased levels of FPV might diminish effectiveness of the ARV drug. **Evidence:** Information from the package label states that amprenavir concentrations decreased with concurrent administration of COCs and amprenavir. Norethindrone concentrations increased and ethinyl estradiol concentrations did not change (*275*).
iii. Indinavir (IDV)	1	**Evidence:** One small study found no pregnancies in women using COCs and IDV (*262*).
iv. Nelfinavir (NFV)	2	**Clarification:** Evidence suggests drug interactions between certain protease inhibitors and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One small study suggested that women using COCs and NFV might have had higher pregnancy rates than those using COCs alone (*262*).
e. CCR5 co-receptor antagonists		
i. Maraviroc (MVC)	1	**Evidence:** COC concentrations were not altered by co-administration with MVC (*280*).
f. HIV integrase strand transfer inhibitors		
i. Raltegravir (RAL)	1	**Evidence:** One pharmacokinetic study demonstrated increased concentrations of norgestimate and no change in ethinyl estradiol among women using COCs and RAL compared with COCs alone (*281*).
ii. Dolutegravir (DTG)	1	**Evidence:** One study demonstrated no clinically relevant pharmacokinetic or pharmacodynamic changes in women using COCs and DTG compared with COCs alone (*282*).
iii. Elvitegravir (EVG)	1	**Evidence:** Information from the package label states that ethinyl estradiol concentrations decreased and norgestimate concentrations increased with concurrent administration of COCs and EVG (*283*). **Comment:** When EVG is administered with cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels might vary when combined with other ARVs.
g. Fusion inhibitors		
i. Enfuvirtide	1	—
Anticonvulsant therapy		
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	3	**Clarification:** Although the interaction of certain anticonvulsants with CHCs is not harmful, it is likely to reduce the effectiveness of CHCs. Use of other contraceptives should be encouraged for persons who are long-term users of any of these drugs. When a COC is chosen, a preparation containing a minimum of 30 μg ethinyl estradiol should be used. **Evidence:** Use of certain anticonvulsants might decrease the effectiveness of COCs (*284–288*).
b. Lamotrigine	3	**Clarification:** The recommendation for lamotrigine applies only for situations where lamotrigine monotherapy is taken concurrently with COCs. Anticonvulsant treatment regimens that combine lamotrigine and non–enzyme-inducing antiepileptic drugs (e.g., sodium valproate) do not interact with COCs. **Evidence:** Pharmacokinetic studies demonstrate levels of lamotrigine decrease significantly during COC use (*288–293*). Certain women who used both COCs and lamotrigine experienced increased seizure activity in one trial (*289*).

See table footnotes on page 84.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	CHC	Clarification/Evidence/Comment
Antimicrobial therapy		
a. Broad-spectrum antibiotics	1	**Evidence:** Most broad-spectrum antibiotics do not affect the contraceptive effectiveness of COCs (*294–330*), patch (*331*), or ring (*332*).
b. Antifungals	1	**Evidence:** Studies of antifungal agents have demonstrated no clinically significant pharmacokinetic interactions with COCs (*333–342*) or ring (*343*).
c. Antiparasitics	1	**Evidence:** Studies of antiparasitic agents have demonstrated no clinically significant pharmacokinetic interactions with COCs (*218,344–348*).
d. Rifampin or rifabutin therapy	3	**Clarification:** Although the interaction of rifampin or rifabutin therapy with CHCs is not harmful, it is likely to reduce the effectiveness of CHCs. Use of other contraceptives should be encouraged for persons who are long-term users of either of these drugs. When a COC is chosen, a preparation containing a minimum of 30 μg ethinyl estradiol should be used. **Evidence:** The balance of the evidence suggests that rifampin reduces the effectiveness of COCs (*349–363*). Data on rifabutin are limited, but effects on metabolism of COCs are less than with rifampin, and small studies have not demonstrated evidence of ovulation (*351,357*).
Psychotropic medications		**Comment:** For many common psychotropic agents, limited or no theoretical concern exists for clinically significant drug interactions when co-administered with hormonal contraceptives. However, either no or very limited data exist examining potential interactions for these classes of medications. For psychotropic agents that are CYP1A2 substrates (e.g., duloxetine, mirtazapine, ziprasidone, olanzapine, clomipramine, imipramine, and amitriptyline), co-administration with CHCs could theoretically yield increased concentrations of the psychotropic drug. For agents with narrow therapeutic windows (e.g., tricyclic antidepressants), increased drug concentrations might pose safety concerns that could necessitate closer monitoring.
a. Selective serotonin reuptake inhibitors (SSRIs)	1	**Evidence:** Limited clinical and pharmacokinetic data do not demonstrate concern for SSRIs decreasing the effectiveness of oral contraceptives. Limited evidence suggests that for women taking SSRIs, the use of hormonal contraceptives was not associated with differences in effectiveness of the SSRI for treatment or in adverse events when compared with women not taking hormonal contraceptives (*364*). **Comment:** Drugs that are inhibitors of CYP3A4 or CYP2C9 theoretically have the potential to increase levels of contraceptive steroids which might increase adverse events. Fluvoxamine is an SSRI known to be a moderate inhibitor of both CYP3A4 and CYP2C9; however, no clinical or pharmacokinetic studies were identified to explore potential drug-drug interactions.
St. John's wort	2	**Evidence:** Although clinical data are limited, studies with pharmacokinetic and pharmacodynamics outcomes raise concern that St. John's wort might decrease effectiveness of hormonal contraceptives, including increased risk for breakthrough bleeding and ovulation and increased metabolism of estrogen and progestins. Any interactions might be dependent on the dose of St. John's wort, and the concentration of active ingredients across types of St. John's wort preparations might vary (*365*).

Abbreviations: ARV = antiretroviral; BMD = bone mineral density; BMI = body mass index; CHC = combined hormonal contraceptive; CKD = chronic kidney disease; COC = combined oral contraceptive; CYP = cytochrome P450; DMPA = depot medroxyprogesterone acetate; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; IBD = inflammatory bowel disease; IUD = intrauterine device; LDL = low-density lipoprotein; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; POC = progestin-only contraceptive; PrEP = pre-exposure prophylaxis; RCT = randomized clinical trial; SLE = systemic lupus erythematosus; SSRI = selective serotonin reuptake inhibitor; STI = sexually transmitted infection; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*; U.S. SPR = *U.S. Selected Practice Recommendations for Contraceptive Use*; VTE = venous thromboembolism.

References

1. Abrams LS, Skee DM, Natarajan J, Wong FA, Lasseter KC. Multiple-dose pharmacokinetics of a contraceptive patch in healthy women participants. Contraception 2001;64:287–94. PMID:11777488 https://doi.org/10.1016/S0010-7824(01)00273-6
2. Ahrendt HJ, Nisand I, Bastianelli C, et al. Efficacy, acceptability and tolerability of the combined contraceptive ring, NuvaRing, compared with an oral contraceptive containing 30 microg of ethinyl estradiol and 3 mg of drospirenone. Contraception 2006;74:451–7. PMID:17157101 https://doi.org/10.1016/j.contraception.2006.07.004
3. Audet MC, Moreau M, Koltun WD, et al.; ORTHO EVRA/EVRA 004 Study Group. Evaluation of contraceptive efficacy and cycle control of a transdermal contraceptive patch vs an oral contraceptive: a randomized controlled trial. JAMA 2001;285:2347–54. PMID:11343482 https://doi.org/10.1001/jama.285.18.2347
4. Bjarnadóttir RI, Tuppurainen M, Killick SR. Comparison of cycle control with a combined contraceptive vaginal ring and oral levonorgestrel/ethinyl estradiol. Am J Obstet Gynecol 2002;186:389–95. PMID:11904596 https://doi.org/10.1067/mob.2002.121103
5. Boonyarangkul A, Taneepanichskul S. Comparison of cycle control and side effects between transdermal contraceptive patch and an oral contraceptive in women older than 35 years. J Med Assoc Thai 2007;90:1715–9. PMID:17957909
6. Burkman RT. The transdermal contraceptive patch: a new approach to hormonal contraception. Int J Fertil Womens Med 2002;47:69–76. PMID:11991433
7. Cole JA, Norman H, Doherty M, Walker AM. Venous thromboembolism, myocardial infarction, and stroke among transdermal contraceptive system users. Obstet Gynecol 2007;109:339–46. PMID:17267834 https://doi.org/10.1097/01.AOG.0000250968.82370.04
8. Devineni D, Skee D, Vaccaro N, et al. Pharmacokinetics and pharmacodynamics of a transdermal contraceptive patch and an oral contraceptive. J Clin Pharmacol 2007;47:497–509. PMID:17389559 https://doi.org/10.1177/0091270006297919
9. Dieben TOM, Roumen FJME, Apter D. Efficacy, cycle control, and user acceptability of a novel combined contraceptive vaginal ring. Obstet Gynecol 2002;100:585–93. PMID:12220783

10. Dittrich R, Parker L, Rosen JB, Shangold G, Creasy GW, Fisher AC; Ortho Evra/Evra 001 Study Group. Transdermal contraception: evaluation of three transdermal norelgestromin/ethinyl estradiol doses in a randomized, multicenter, dose-response study. Am J Obstet Gynecol 2002;186:15–20. PMID:11810078 https://doi.org/10.1067/mob.2002.118844
11. Duijkers I, Killick S, Bigrigg A, Dieben TOM. A comparative study on the effects of a contraceptive vaginal ring NuvaRing and an oral contraceptive on carbohydrate metabolism and adrenal and thyroid function. Eur J Contracept Reprod Health Care 2004;9:131–40. PMID:15697102 https://doi.org/10.1080/13625180400007199
12. Duijkers IJ, Klipping C, Verhoeven CH, Dieben TOM. Ovarian function with the contraceptive vaginal ring or an oral contraceptive: a randomized study. Hum Reprod 2004;19:2668–73. PMID:15333593 https://doi.org/10.1093/humrep/deh493
13. Elkind-Hirsch KE, Darensbourg C, Ogden B, Ogden LF, Hindelang P. Contraceptive vaginal ring use for women has less adverse metabolic effects than an oral contraceptive. Contraception 2007;76:348–56. PMID:17963858 https://doi.org/10.1016/j.contraception.2007.08.001
14. Hedon B, Helmerhorst FM, Cronje HS, et al.; The EVRA 003 Study Group. Comparison of efficacy, cycle control, compliance and safety in users of a contraceptive patch versus an oral contraceptive. Int J Gynaecol Obstet 2000;70:78. https://doi.org/10.1016/S0020-7292(00)85161-9
15. Jick S, Kaye JA, Li L, Jick H. Further results on the risk of nonfatal venous thromboembolism in users of the contraceptive transdermal patch compared to users of oral contraceptives containing norgestimate and 35 microg of ethinyl estradiol. Contraception 2007;76:4–7. PMID:17586129 https://doi.org/10.1016/j.contraception.2007.03.003
16. Jick SS, Jick H. Cerebral venous sinus thrombosis in users of four hormonal contraceptives: levonorgestrel-containing oral contraceptives, norgestimate-containing oral contraceptives, desogestrel-containing oral contraceptives and the contraceptive patch. Contraception 2006;74:290–2. PMID:16982227 https://doi.org/10.1016/j.contraception.2006.05.071
17. Jick SS, Jick H. The contraceptive patch in relation to ischemic stroke and acute myocardial infarction. Pharmacotherapy 2007;27:218–20. PMID:17253912 https://doi.org/10.1592/phco.27.2.218
18. Jick SS, Kaye JA, Russmann S, Jick H. Risk of nonfatal venous thromboembolism in women using a contraceptive transdermal patch and oral contraceptives containing norgestimate and 35 microg of ethinyl estradiol. Contraception 2006;73:223–8. PMID:16472560 https://doi.org/10.1016/j.contraception.2006.01.001
19. Magnusdóttir EM, Bjarnadóttir RI, Onundarson PT, et al. The contraceptive vaginal ring (NuvaRing) and hemostasis: a comparative study. Contraception 2004;69:461–7. PMID:15157790 https://doi.org/10.1016/j.contraception.2003.12.010
20. Massai R, Mäkäräinen L, Kuukankorpi A, Klipping C, Duijkers I, Dieben T. The combined contraceptive vaginal ring and bone mineral density in healthy pre-menopausal women. Hum Reprod 2005;20:2764–8. PMID:15980008 https://doi.org/10.1093/humrep/dei117
21. Milsom I, Lete I, Bjertnaes A, et al. Effects on cycle control and bodyweight of the combined contraceptive ring, NuvaRing, versus an oral contraceptive containing 30 microg ethinyl estradiol and 3 mg drospirenone. Hum Reprod 2006;21:2304–11. PMID:16763008 https://doi.org/10.1093/humrep/del162
22. Oddsson K, Leifels-Fischer B, de Melo NR, et al. Efficacy and safety of a contraceptive vaginal ring (NuvaRing) compared with a combined oral contraceptive: a 1-year randomized trial. Contraception 2005;71:176–82. PMID:15722066 https://doi.org/10.1016/j.contraception.2004.09.001
23. Pierson RA, Archer DF, Moreau M, Shangold GA, Fisher AC, Creasy GW. Ortho Evra/Evra versus oral contraceptives: follicular development and ovulation in normal cycles and after an intentional dosing error. Fertil Steril 2003;80:34–42. PMID:12849799 https://doi.org/10.1016/S0015-0282(03)00556-9
24. Radowicki S, Skórzewska K, Szlendak K. [Safety evaluation of a transdermal contraceptive system with an oral contraceptive]. Ginekol Pol 2005;76:884–9. PMID:16566363
25. Sabatini R, Cagiano R. Comparison profiles of cycle control, side effects and sexual satisfaction of three hormonal contraceptives. Contraception 2006;74:220–3. PMID:16904415 https://doi.org/10.1016/j.contraception.2006.03.022
26. Smallwood GH, Meador ML, Lenihan JP Jr, Shangold GA, Fisher AC, Creasy GW; ORTHO EVRA/EVRA 002 Study Group. Efficacy and safety of a transdermal contraceptive system. Obstet Gynecol 2001;98:799–805. PMID:11704172 https://doi.org/10.1097/00006250-200111000-00016
27. Timmer CJ, Mulders TM. Pharmacokinetics of etonogestrel and ethinylestradiol released from a combined contraceptive vaginal ring. Clin Pharmacokinet 2000;39:233–42. PMID:11020137 https://doi.org/10.2165/00003088-200039030-00005
28. Tuppurainen M, Klimscheffskij R, Venhola M, Dieben TOM. The combined contraceptive vaginal ring (NuvaRing) and lipid metabolism: a comparative study. Contraception 2004;69:389–94. PMID:15105061 https://doi.org/10.1016/j.contraception.2004.01.004
29. Urdl W, Apter D, Alperstein A, et al.; ORTHO EVRA/EVRA 003 Study Group. Contraceptive efficacy, compliance and beyond: factors related to satisfaction with once-weekly transdermal compared with oral contraception. Eur J Obstet Gynecol Reprod Biol 2005;121:202–10. PMID:16054963 https://doi.org/10.1016/j.ejogrb.2005.01.021
30. van den Heuvel MW, van Bragt AJM, Alnabawy AKM, Kaptein MCJ. Comparison of ethinylestradiol pharmacokinetics in three hormonal contraceptive formulations: the vaginal ring, the transdermal patch and an oral contraceptive. Contraception 2005;72:168–74. PMID:16102549 https://doi.org/10.1016/j.contraception.2005.03.005
31. Veres S, Miller L, Burington B. A comparison between the vaginal ring and oral contraceptives. Obstet Gynecol 2004;104:555–63. PMID:15339769 https://doi.org/10.1097/01.AOG.0000136082.59644.13
32. White T, Ozel B, Jain JK, Stanczyk FZ. Effects of transdermal and oral contraceptives on estrogen-sensitive hepatic proteins. Contraception 2006;74:293–6. PMID:16982228 https://doi.org/10.1016/j.contraception.2006.04.005
33. Zieman M, Guillebaud J, Weisberg E, Shangold GA, Fisher AC, Creasy GW. Contraceptive efficacy and cycle control with the Ortho Evra/Evra transdermal system: the analysis of pooled data. Fertil Steril 2002;77(Suppl 2):S13–8. PMID:11849631 https://doi.org/10.1016/S0015-0282(01)03275-7
34. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
35. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
36. Memon S, Iversen L, Hannaford PC. Is the oral contraceptive pill associated with fracture in later life? New evidence from the Royal College of General Practitioners Oral Contraception Study. Contraception 2011;84:40–7. PMID:21664509 https://doi.org/10.1016/j.contraception.2010.11.019

37. Vestergaard P, Rejnmark L, Mosekilde L. Fracture risk in very young women using combined oral contraceptives. Contraception 2008;78:358–64. PMID:18929731 https://doi.org/10.1016/j.contraception.2008.06.010
38. Vestergaard P, Rejnmark L, Mosekilde L. Oral contraceptive use and risk of fractures. Contraception 2006;73:571–6. PMID:16730486 https://doi.org/10.1016/j.contraception.2006.01.006
39. Barad D, Kooperberg C, Wactawski-Wende J, Liu J, Hendrix SL, Watts NB. Prior oral contraception and postmenopausal fracture: a Women's Health Initiative observational cohort study. Fertil Steril 2005;84:374–83. PMID:16084878 https://doi.org/10.1016/j.fertnstert.2005.01.132
40. Michaëlsson K, Baron JA, Farahmand BY, Ljunghall S. Influence of parity and lactation on hip fracture risk. Am J Epidemiol 2001;153:1166–72. PMID:11415951 https://doi.org/10.1093/aje/153.12.1166
41. Michaëlsson K, Baron JA, Farahmand BY, Persson I, Ljunghall S. Oral-contraceptive use and risk of hip fracture: a case-control study. Lancet 1999;353:1481–4. PMID:10232314 https://doi.org/10.1016/S0140-6736(98)09044-8
42. La Vecchia C, Tavani A, Gallus S. Oral contraceptives and risk of hip fractures. Lancet 1999;354:335–6. PMID:10440332 https://doi.org/10.1016/S0140-6736(05)75239-9
43. Vessey M, Mant J, Painter R. Oral contraception and other factors in relation to hospital referral for fracture. Findings in a large cohort study. Contraception 1998;57:231–5. PMID:9649913 https://doi.org/10.1016/S0010-7824(98)00026-2
44. O'Neill TW, Marsden D, Adams JE, Silman AJ. Risk factors, falls, and fracture of the distal forearm in Manchester, UK. J Epidemiol Community Health 1996;50:288–92. PMID:8935460 https://doi.org/10.1136/jech.50.3.288
45. Mallmin H, Ljunghall S, Persson I, Bergström R. Risk factors for fractures of the distal forearm: a population-based case-control study. Osteoporos Int 1994;4:298–304. PMID:7696821 https://doi.org/10.1007/BF01622186
46. Cooper C, Hannaford P, Croft P, Kay CR. Oral contraceptive pill use and fractures in women: a prospective study. Bone 1993;14:41–5. PMID:8443001 https://doi.org/10.1016/8756-3282(93)90254-8
47. Meier C, Brauchli YB, Jick SS, Kraenzlin ME, Meier CR. Use of depot medroxyprogesterone acetate and fracture risk. J Clin Endocrinol Metab 2010;95:4909–16. PMID:20685865 https://doi.org/10.1210/jc.2010-0032
48. Cibula D, Skrenkova J, Hill M, Stepan JJ. Low-dose estrogen combined oral contraceptives may negatively influence physiological bone mineral density acquisition during adolescence. Eur J Endocrinol 2012;166:1003–11. PMID:22436400 https://doi.org/10.1530/EJE-11-1047
49. Gai L, Jia Y, Zhang M, et al. Effect of two kinds of different combined oral contraceptives use on bone mineral density in adolescent women. Contraception 2012;86:332–6. PMID:22364818 https://doi.org/10.1016/j.contraception.2012.01.009
50. Scholes D, Hubbard RA, Ichikawa LE, et al. Oral contraceptive use and bone density change in adolescent and young adult women: a prospective study of age, hormone dose, and discontinuation. J Clin Endocrinol Metab 2011;96:E1380–7. PMID:21752879 https://doi.org/10.1210/jc.2010-3027
51. Lattakova M, Borovsky M, Payer J, Killinger Z. Oral contraception usage in relation to bone mineral density and bone turnover in adolescent girls. Eur J Contracept Reprod Health Care 2009;14:207–14. PMID:19565418 https://doi.org/10.1080/13625180902838828
52. Beksinska ME, Kleinschmidt I, Smit JA, Farley TMM. Bone mineral density in a cohort of adolescents during use of norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives and after discontinuation of norethisterone enanthate. Contraception 2009;79:345–9. PMID:19341845 https://doi.org/10.1016/j.contraception.2008.11.009
53. Pikkarainen E, Lehtonen-Veromaa M, Möttönen T, Kautiainen H, Viikari J. Estrogen-progestin contraceptive use during adolescence prevents bone mass acquisition: a 4-year follow-up study. Contraception 2008;78:226–31. PMID:18692613 https://doi.org/10.1016/j.contraception.2008.05.002
54. Cromer BA, Bonny AE, Stager M, et al. Bone mineral density in adolescent females using injectable or oral contraceptives: a 24-month prospective study. Fertil Steril 2008;90:2060–7. PMID:18222431 https://doi.org/10.1016/j.fertnstert.2007.10.070
55. Berenson AB, Rahman M, Breitkopf CR, Bi LX. Effects of depot medroxyprogesterone acetate and 20-microgram oral contraceptives on bone mineral density. Obstet Gynecol 2008;112:788–99. PMID:18827121 https://doi.org/10.1097/AOG.0b013e3181875b78
56. Harel Z, Riggs S, Vaz R, Flanagan P, Harel D, Machan JT. Bone accretion in adolescents using the combined estrogen and progestin transdermal contraceptive method Ortho Evra: a pilot study. J Pediatr Adolesc Gynecol 2010;23:23–31. PMID:19647454 https://doi.org/10.1016/j.jpag.2009.04.008
57. Cobb KL, Bachrach LK, Sowers M, et al. The effect of oral contraceptives on bone mass and stress fractures in female runners. Med Sci Sports Exerc 2007;39:1464–73. PMID:17805075 https://doi.org/10.1249/mss.0b013e318074e532
58. Beksinska ME, Kleinschmidt I, Smit JA, Farley TMM. Bone mineral density in adolescents using norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives for contraception. Contraception 2007;75:438–43. PMID:17519149 https://doi.org/10.1016/j.contraception.2007.02.001
59. Lara-Torre E, Edwards CP, Perlman S, Hertweck SP. Bone mineral density in adolescent females using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2004;17:17–21. PMID:15010034 https://doi.org/10.1016/j.jpag.2003.11.017
60. Cromer BA, Blair JM, Mahan JD, Zibners L, Naumovski Z. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. J Pediatr 1996;129:671–6. PMID:8917232 https://doi.org/10.1016/S0022-3476(96)70148-8
61. Polatti F, Perotti F, Filippa N, Gallina D, Nappi RE. Bone mass and long-term monophasic oral contraceptive treatment in young women. Contraception 1995;51:221–4. PMID:7796586 https://doi.org/10.1016/0010-7824(95)00036-A
62. Sørdal T, Grob P, Verhoeven C. Effects on bone mineral density of a monophasic combined oral contraceptive containing nomegestrol acetate/17β-estradiol in comparison to levonorgestrel/ethinylestradiol. Acta Obstet Gynecol Scand 2012;91:1279–85. PMID:22762147 https://doi.org/10.1111/j.1600-0412.2012.01498.x
63. Gargano V, Massaro M, Morra I, Formisano C, Di Carlo C, Nappi C. Effects of two low-dose combined oral contraceptives containing drospirenone on bone turnover and bone mineral density in young fertile women: a prospective controlled randomized study. Contraception 2008;78:10–5. PMID:18555812 https://doi.org/10.1016/j.contraception.2008.01.016
64. Nappi C, Di Spiezio Sardo A, Greco E, Tommaselli GA, Giordano E, Guida M. Effects of an oral contraceptive containing drospirenone on bone turnover and bone mineral density. Obstet Gynecol 2005;105:53–60. PMID:15625142 https://doi.org/10.1097/01.AOG.0000148344.26475.fc

65. Berenson AB, Radecki CM, Grady JJ, Rickert VI, Thomas A. A prospective, controlled study of the effects of hormonal contraception on bone mineral density. Obstet Gynecol 2001;98:576–82. PMID:11576570 https://doi.org/10.1016/S0029-7844(01)01495-8
66. Berenson AB, Breitkopf CR, Grady JJ, Rickert VI, Thomas A. Effects of hormonal contraception on bone mineral density after 24 months of use. Obstet Gynecol 2004;103:899–906. PMID:15121563 https://doi.org/10.1097/01.AOG.0000117082.49490.d5
67. Elgán C, Samsioe G, Dykes AK. Influence of smoking and oral contraceptives on bone mineral density and bone remodeling in young women: a 2-year study. Contraception 2003;67:439–47. PMID:12814812 https://doi.org/10.1016/S0010-7824(03)00048-9
68. Elgán C, Dykes AK, Samsioe G. Bone mineral density changes in young women: a two year study. Gynecol Endocrinol 2004;19:169–77. PMID:15724798 https://doi.org/10.1080/09513590400012119
69. Endrikat J, Mih E, Düsterberg B, et al. A 3-year double-blind, randomized, controlled study on the influence of two oral contraceptives containing either 20 microg or 30 microg ethinylestradiol in combination with levonorgestrel on bone mineral density. Contraception 2004;69:179–87. PMID:14969664 https://doi.org/10.1016/j.contraception.2003.10.002
70. Paoletti AM, Orrù M, Lello S, et al. Short-term variations in bone remodeling markers of an oral contraception formulation containing 3 mg of drospirenone plus 30 microg of ethinyl estradiol: observational study in young postadolescent women. Contraception 2004;70:293–8. PMID:15451333 https://doi.org/10.1016/j.contraception.2004.04.004
71. Nappi C, Di Spiezio Sardo A, Acunzo G, et al. Effects of a low-dose and ultra-low-dose combined oral contraceptive use on bone turnover and bone mineral density in young fertile women: a prospective controlled randomized study. Contraception 2003;67:355–9. PMID:12742557 https://doi.org/10.1016/S0010-7824(03)00025-8
72. Reed SD, Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Longitudinal changes in bone density in relation to oral contraceptive use. Contraception 2003;68:177–82. PMID:14561537 https://doi.org/10.1016/S0010-7824(03)00147-1
73. Cobb KL, Kelsey JL, Sidney S, Ettinger B, Lewis CE. Oral contraceptives and bone mineral density in white and black women in CARDIA. Coronary Risk Development in Young Adults. Osteoporos Int 2002;13:893–900. PMID:12415437 https://doi.org/10.1007/s001980200123
74. Burr DB, Yoshikawa T, Teegarden D, et al. Exercise and oral contraceptive use suppress the normal age-related increase in bone mass and strength of the femoral neck in women 18–31 years of age. Bone 2000;27:855–63. PMID:11113398 https://doi.org/10.1016/S8756-3282(00)00403-8
75. Recker RR, Davies KM, Hinders SM, Heaney RP, Stegman MR, Kimmel DB. Bone gain in young adult women. JAMA 1992;268:2403–8. PMID:1404797 https://doi.org/10.1001/jama.1992.03490170075028
76. Mazess RB, Barden HS. Bone density in premenopausal women: effects of age, dietary intake, physical activity, smoking, and birth-control pills. Am J Clin Nutr 1991;53:132–42. PMID:1984338 https://doi.org/10.1093/ajcn/53.1.132
77. Gambacciani M, Cappagli B, Lazzarini V, Ciaponi M, Fruzzetti F, Genazzani AR. Longitudinal evaluation of perimenopausal bone loss: effects of different low dose oral contraceptive preparations on bone mineral density. Maturitas 2006;54:176–80. PMID:16332417 https://doi.org/10.1016/j.maturitas.2005.10.007
78. Gambacciani M, Spinetti A, Taponeco F, Cappagli B, Piaggesi L, Fioretti P. Longitudinal evaluation of perimenopausal vertebral bone loss: effects of a low-dose oral contraceptive preparation on bone mineral density and metabolism. Obstet Gynecol 1994;83:392–6. PMID:8127531
79. Gambacciani M, Spinetti A, Cappagli B, et al. Hormone replacement therapy in perimenopausal women with a low dose oral contraceptive preparation: effects on bone mineral density and metabolism. Maturitas 1994;19:125–31. PMID:7968645 https://doi.org/10.1016/0378-5122(94)90062-0
80. Gambacciani M, Cappagli B, Ciaponi M, Benussi C, Genazzani AR. Hormone replacement therapy in perimenopause: effect of a low dose oral contraceptive preparation on bone quantitative ultrasound characteristics. Menopause 1999;6:43–8. PMID:10100179 https://doi.org/10.1097/00042192-199906010-00009
81. Volpe A, Malmusi S, Zanni AL, Landi S, Cagnacci A. Oral contraceptives and bone metabolism. Eur J Contracept Reprod Health Care 1997;2:225–8. PMID:9678077 https://doi.org/10.3109/13625189709165298
82. Hansen MA, Overgaard K, Riis BJ, Christiansen C. Potential risk factors for development of postmenopausal osteoporosis—examined over a 12-year period. Osteoporos Int 1991;1:95–102. PMID:1790399 https://doi.org/10.1007/BF01880450
83. Shargil AA. Hormone replacement therapy in perimenopausal women with a triphasic contraceptive compound: a three-year prospective study. Int J Fertil 1985;30:15–18–28, 18–28. PMID:2862116
84. Taechakraichana N, Limpaphayom K, Ninlagarn T, Panyakhamlerd K, Chaikittisilpa S, Dusitsin N. A randomized trial of oral contraceptive and hormone replacement therapy on bone mineral density and coronary heart disease risk factors in postmenopausal women. Obstet Gynecol 2000;95:87–94. PMID:10636509
85. Taechakraichana N, Jaisamrarn U, Panyakhamlerd K, Chaikittisilpa S, Limpaphayom K. Difference in bone acquisition among hormonally treated postmenopausal women with normal and low bone mass. J Med Assoc Thai 2001;84(Suppl 2):S586–92. PMID:11853286
86. Grimes DA, Schulz KF. Surrogate end points in clinical research: hazardous to your health. Obstet Gynecol 2005;105:1114–8. PMID:15863552 https://doi.org/10.1097/01.AOG.0000157445.67309.19
87. Schönau E. The peak bone mass concept: is it still relevant? Pediatr Nephrol 2004;19:825–31. PMID:15197638 https://doi.org/10.1007/s00467-004-1465-5
88. Cohen A, Shane E. Treatment of premenopausal women with low bone mineral density. Curr Osteoporos Rep 2008;6:39–46. PMID:18430399 https://doi.org/10.1007/s11914-008-0007-7
89. US Department of Agriculture; US Department of Health and Human Services. Dietary guidelines for Americans, 2020–2025. 9th ed. Washington, DC: US Department of Agriculture and US Department of Health and Human Services; 2020. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf
90. Meek JY, Noble L; Section on Breastfeeding. Policy statement: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057988. PMID:35921640 https://doi.org/10.1542/peds.2022-057988
91. Tepper NK, Phillips SJ, Kapp N, Gaffield ME, Curtis KM. Combined hormonal contraceptive use among breastfeeding women: an updated systematic review. Contraception 2016;94:262–74. PMID:26002804 https://doi.org/10.1016/j.contraception.2015.05.006
92. Petersen JF, Bergholt T, Nielsen AK, Paidas MJ, Løkkegaard EC. Combined hormonal contraception and risk of venous thromboembolism within the first year following pregnancy. Danish nationwide historical cohort 1995–2009. Thromb Haemost 2014;112:73–8. PMID:24499991 https://doi.org/10.1160/TH13-09-0797
93. Jackson E, Curtis KM, Gaffield ME. Risk of venous thromboembolism during the postpartum period: a systematic review. Obstet Gynecol 2011;117:691–703. PMID:21343773 https://doi.org/10.1097/AOG.0b013e31820ce2db

94. Kamel H, Navi BB, Sriram N, Hovsepian DA, Devereux RB, Elkind MS. Risk of a thrombotic event after the 6-week postpartum period. N Engl J Med 2014;370:1307–15. PMID:24524551 https://doi.org/10.1056/NEJMoa1311485
95. Sultan AA, Tata LJ, West J, et al. Risk factors for first venous thromboembolism around pregnancy: a population-based cohort study from the United Kingdom. Blood 2013;121:3953–61. PMID:23550034 https://doi.org/10.1182/blood-2012-11-469551
96. Sultan AA, West J, Tata LJ, Fleming KM, Nelson-Piercy C, Grainge MJ. Risk of first venous thromboembolism in and around pregnancy: a population-based cohort study. Br J Haematol 2012;156:366–73. PMID:22145820 https://doi.org/10.1111/j.1365-2141.2011.08956.x
97. Tepper NK, Boulet SL, Whiteman MK, et al. Postpartum venous thromboembolism: incidence and risk factors. Obstet Gynecol 2014;123:987–96. PMID:24785851 https://doi.org/10.1097/AOG.0000000000000230
98. Jackson E, Glasier A. Return of ovulation and menses in postpartum nonlactating women: a systematic review. Obstet Gynecol 2011;117:657–62. PMID:21343770 https://doi.org/10.1097/AOG.0b013e31820ce18c
99. Kim C, Nguyen AT, Berry-Bibee E, Ermias Y, Gaffield ME, Kapp N. Systemic hormonal contraception initiation after abortion: a systematic review and meta-analysis. Contraception 2021;103:291–304. PMID:33548267 https://doi.org/10.1016/j.contraception.2021.01.017
100. Gaffield ME, Kapp N, Ravi A. Use of combined oral contraceptives post abortion. Contraception 2009;80:355–62. PMID:19751858 https://doi.org/10.1016/j.contraception.2009.04.005
101. Gillum LA, Mamidipudi SK, Johnston SC. Ischemic stroke risk with oral contraceptives: A meta-analysis. JAMA 2000;284:72–8. PMID:10872016 https://doi.org/10.1001/jama.284.1.72
102. Jick SS, Walker AM, Stergachis A, Jick H. Oral contraceptives and breast cancer. Br J Cancer 1989;59:618–21. PMID:2713248 https://doi.org/10.1038/bjc.1989.125
103. Khader YS, Rice J, John L, Abueita O. Oral contraceptives use and the risk of myocardial infarction: a meta-analysis. Contraception 2003;68:11–7. PMID:12878281 https://doi.org/10.1016/S0010-7824(03)00073-8
104. Lawson DH, Davidson JF, Jick H. Oral contraceptive use and venous thromboembolism: absence of an effect of smoking. BMJ 1977;2:729–30. PMID:334332 https://doi.org/10.1136/bmj.2.6089.729
105. Lidegaard O, Edström B, Kreiner S. Oral contraceptives and venous thromboembolism. A case-control study. Contraception 1998;57:291–301. PMID:9673836 https://doi.org/10.1016/S0010-7824(98)00033-X
106. Nightingale AL, Lawrenson RA, Simpson EL, Williams TJ, MacRae KD, Farmer RD. The effects of age, body mass index, smoking and general health on the risk of venous thromboembolism in users of combined oral contraceptives. Eur J Contracept Reprod Health Care 2000;5:265–74. PMID:11245554 https://doi.org/10.1080/13625180008500402
107. Petitti DB, Wingerd J, Pellegrin F, Ramcharan S. Risk of vascular disease in women. Smoking, oral contraceptives, noncontraceptive estrogens, and other factors. JAMA 1979;242:1150–4. PMID:470067 https://doi.org/10.1001/jama.1979.03300110022020
108. Rosenberg L, Palmer JR, Rao RS, Shapiro S. Low-dose oral contraceptive use and the risk of myocardial infarction. Arch Intern Med 2001;161:1065–70. PMID:11322840 https://doi.org/10.1001/archinte.161.8.1065
109. Straneva P, Hinderliter A, Wells E, Lenahan H, Girdler S. Smoking, oral contraceptives, and cardiovascular reactivity to stress. Obstet Gynecol 2000;95:78–83. PMID:10636507 https://doi.org/10.1016/S0029-7844(99)00497-4
110. Tanis BC, van den Bosch MA, Kemmeren JM, et al. Oral contraceptives and the risk of myocardial infarction. N Engl J Med 2001;345:1787–93. PMID:11752354 https://doi.org/10.1056/NEJMoa003216
111. Van den bosch MAAJ, Kemmeren JM, Tanis BC, et al. The RATIO study: oral contraceptives and the risk of peripheral arterial disease in young women. J Thromb Hemost 2003;1:439–44. PMID:12871447 https://doi.org/10.1046/j.1538-7836.2003.00079.x
112. World Health Organization Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Venous thromboembolic disease and combined oral contraceptives: results of international multicentre case-control study. Lancet 1995;346:1575–82. PMID:7500748 https://doi.org/10.1016/S0140-6736(95)91926-0
113. Collaborative Group for the Study of Stroke in Young Women. Oral contraceptives and stroke in young women. Associated risk factors. JAMA 1975;231:718–22. PMID:1172861 https://doi.org/10.1001/jama.1975.03240190022010
114. Horton LG, Simmons KB, Curtis KM. Combined hormonal contraceptive use among obese women and risk for cardiovascular events: a systematic review. Contraception 2016;94:590–604. PMID:27263039 https://doi.org/10.1016/j.contraception.2016.05.014
115. Schink T, Princk C, Braitmaier M, Haug U. Use of combined oral contraceptives and risk of venous thromboembolism in young women: a nested case-control analysis using German claims data. BJOG 2022;129:2107–16. PMID:35876787 https://doi.org/10.1111/1471-0528.17268
116. Traven SA, McGurk KM, Althoff AD, et al. Resident level involvement affects operative time and surgical complications in lower extremity fracture care. J Surg Educ 2021;78:1755–61. PMID:33903063 https://doi.org/10.1016/j.jsurg.2021.03.004
117. Brunner Huber LR, Hogue CJ, Stein AD, Drews C, Zieman M. Body mass index and risk for oral contraceptive failure: a case-cohort study in South Carolina. Ann Epidemiol 2006;16:637–43. PMID:16516489 https://doi.org/10.1016/j.annepidem.2006.01.001
118. Brunner Huber LR, Toth JL. Obesity and oral contraceptive failure: findings from the 2002 National Survey of Family Growth. Am J Epidemiol 2007;166:1306–11. PMID:17785712 https://doi.org/10.1093/aje/kwm221
119. Brunner LR, Hogue CJ. The role of body weight in oral contraceptive failure: results from the 1995 national survey of family growth. Ann Epidemiol 2005;15:492–9. PMID:16029841 https://doi.org/10.1016/j.annepidem.2004.10.009
120. Holt VL, Cushing-Haugen KL, Daling JR. Body weight and risk of oral contraceptive failure. Obstet Gynecol 2002;99:820–7. PMID:11978293 https://doi.org/10.1016/S0029-7844(02)01939-7
121. Holt VL, Scholes D, Wicklund KG, Cushing-Haugen KL, Daling JR. Body mass index, weight, and oral contraceptive failure risk. Obstet Gynecol 2005;105:46–52. PMID:15625141 https://doi.org/10.1097/01.AOG.0000149155.11912.52
122. Trussell J, Schwarz EB, Guthrie K. Obesity and oral contraceptive pill failure. Contraception 2009;79:334–8. PMID:19341843 https://doi.org/10.1016/j.contraception.2008.11.017
123. Vessey M. Oral contraceptive failures and body weight: findings in a large cohort study. J Fam Plann Reprod Health Care 2001;27:90–1. PMID:12457519 https://doi.org/10.1783/147118901101195092
124. Yamazaki M, Dwyer K, Sobhan M, et al. Effect of obesity on the effectiveness of hormonal contraceptives: an individual participant data meta-analysis. Contraception 2015;92:445–52. PMID:26247330 https://doi.org/10.1016/j.contraception.2015.07.016
125. Dragoman MV, Simmons KB, Paulen ME, Curtis KM. Combined hormonal contraceptive (CHC) use among obese women and contraceptive effectiveness: a systematic review. Contraception 2017;95:117–29. PMID:27823942 https://doi.org/10.1016/j.contraception.2016.10.010
126. Paulen ME, Zapata LB, Cansino C, Curtis KM, Jamieson DJ. Contraceptive use among women with a history of bariatric surgery: a systematic review. Contraception 2010;82:86–94. PMID:20682146 https://doi.org/10.1016/j.contraception.2010.02.008

127. Curtis KM, Nguyen AT, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2024. MMWR Recomm Rep 24;73(No. RR-3):1–77.
128. Lidegaard Ø, Edström B, Kreiner S. Oral contraceptives and venous thromboembolism: a five-year national case-control study. Contraception 2002;65:187–96. PMID:11929640 https://doi.org/10.1016/S0010-7824(01)00307-9
129. Croft P, Hannaford PC. Risk factors for acute myocardial infarction in women: evidence from the Royal College of General Practitioners' oral contraception study. BMJ 1989;298:165–8. PMID:2493841 https://doi.org/10.1136/bmj.298.6667.165
130. D'Avanzo B, La Vecchia C, Negri E, Parazzini F, Franceschi S. Oral contraceptive use and risk of myocardial infarction: an Italian case-control study. J Epidemiol Community Health 1994;48:324–5. PMID:8051537 https://doi.org/10.1136/jech.48.3.324
131. Dunn NR, Faragher B, Thorogood M, et al. Risk of myocardial infarction in young female smokers. Heart 1999;82:581–3. PMID:10525513 https://doi.org/10.1136/hrt.82.5.581
132. Hannaford PC, Croft PR, Kay CR. Oral contraception and stroke. Evidence from the Royal College of General Practitioners' Oral Contraception Study. Stroke 1994;25:935–42. PMID:8165687 https://doi.org/10.1161/01.STR.25.5.935
133. Heinemann LA, Lewis MA, Spitzer WO, Thorogood M, Guggenmoos-Holzmann I, Bruppacher R; Transnational Research Group on Oral Contraceptives and the Health of Young Women. Thromboembolic stroke in young women. A European case-control study on oral contraceptives. Contraception 1998;57:29–37. PMID:9554248 https://doi.org/10.1016/S0010-7824(97)00204-7
134. Kemmeren JM, Tanis BC, van den Bosch MA, et al. Risk of Arterial Thrombosis in Relation to Oral Contraceptives (RATIO) study: oral contraceptives and the risk of ischemic stroke. Stroke 2002;33:1202–8. PMID:11988591 https://doi.org/10.1161/01.STR.0000015345.61324.3F
135. Lewis MA, Heinemann LA, Spitzer WO, MacRae KD, Bruppacher R. The use of oral contraceptives and the occurrence of acute myocardial infarction in young women. Results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Contraception 1997;56:129–40. PMID:9347202 https://doi.org/10.1016/S0010-7824(97)00118-2
136. Lidegaard O. Oral contraception and risk of a cerebral thromboembolic attack: results of a case-control study. BMJ 1993;306:956–63. PMID:8490470 https://doi.org/10.1136/bmj.306.6883.956
137. Lidegaard O. Oral contraceptives, pregnancy and the risk of cerebral thromboembolism: the influence of diabetes, hypertension, migraine and previous thrombotic disease. Br J Obstet Gynaecol 1995;102:153–9. PMID:7756208 https://doi.org/10.1111/j.1471-0528.1995.tb09070.x
138. Lubianca JN, Faccin CS, Fuchs FD. Oral contraceptives: a risk factor for uncontrolled blood pressure among hypertensive women. Contraception 2003;67:19–24. PMID:12521653 https://doi.org/10.1016/S0010-7824(02)00429-8
139. Narkiewicz K, Graniero GR, D'Este D, Mattarei M, Zonzin P, Palatini P. Ambulatory blood pressure in mild hypertensive women taking oral contraceptives. A case-control study. Am J Hypertens 1995;8:249–53. PMID:7794573 https://doi.org/10.1016/0895-7061(95)96212-3
140. Siritho S, Thrift AG, McNeil JJ, You RX, Davis SM, Donnan GA; Melbourne Risk Factor Study (MERFS) Group. Risk of ischemic stroke among users of the oral contraceptive pill: The Melbourne Risk Factor Study (MERFS) Group. Stroke 2003;34:1575–80. PMID:12805499 https://doi.org/10.1161/01.STR.0000077925.16041.6B
141. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Ischaemic stroke and combined oral contraceptives: results of an international, multicentre, case-control study. Lancet 1996;348:498–505. PMID:8757151 https://doi.org/10.1016/S0140-6736(95)12393-8
142. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Acute myocardial infarction and combined oral contraceptives: results of an international multicentre case-control study. Lancet 1997;349:1202–9. PMID:9130941 https://doi.org/10.1016/S0140-6736(97)02358-1
143. Lubianca JN, Moreira LB, Gus M, Fuchs FD. Stopping oral contraceptives: an effective blood pressure-lowering intervention in women with hypertension. J Hum Hypertens 2005;19:451–5. PMID:15759027 https://doi.org/10.1038/sj.jhh.1001841
144. Aberg H, Karlsson L, Melander S. Studies on toxaemia of pregnancy with special reference to blood pressure. II. Results after 6-11 years' follow-up. Ups J Med Sci 1978;83:97–102. PMID:664120 https://doi.org/10.3109/03009737809179119
145. Carmichael SM, Taylor MM, Ayers CR. Oral contraceptives, hypertension, and toxemia. Obstet Gynecol 1970;35:371–6. PMID:4190423
146. Meinel H, Ihle R, Laschinski M. [Effect of hormonal contraceptives on blood pressure following pregnancy-induced hypertension]. Zentralbl Gynakol 1987;109:527–31. PMID:3604495
147. Pritchard JA, Pritchard SA. Blood pressure response to estrogen-progestin oral contraceptive after pregnancy-induced hypertension. Am J Obstet Gynecol 1977;129:733–9. PMID:607805 https://doi.org/10.1016/0002-9378(77)90390-8
148. Sibai BM, Taslimi MM, el-Nazer A, Amon E, Mabie BC, Ryan GM. Maternal-perinatal outcome associated with the syndrome of hemolysis, elevated liver enzymes, and low platelets in severe preeclampsia-eclampsia. Am J Obstet Gynecol 1986;155:501–9. PMID:3529964 https://doi.org/10.1016/0002-9378(86)90266-8
149. Sibai BM, Ramadan MK, Chari RS, Friedman SA. Pregnancies complicated by HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): subsequent pregnancy outcome and long-term prognosis. Am J Obstet Gynecol 1995;172:125–9. PMID:7847520 https://doi.org/10.1016/0002-9378(95)90099-3
150. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Haemorrhagic stroke, overall stroke risk, and combined oral contraceptives: results of an international, multicentre, case-control study. Lancet 1996;348:505–10. PMID:8757152 https://doi.org/10.1016/S0140-6736(95)12394-6
151. Martinelli I, Lensing AW, Middeldorp S, et al. Recurrent venous thromboembolism and abnormal uterine bleeding with anticoagulant and hormone therapy use. Blood 2016;127:1417–25. PMID:26696010 https://doi.org/10.1182/blood-2015-08-665927
152. Barbhaiya M, Zuily S, Naden R, et al.; ACR/EULAR APS Classification Criteria Collaborators. The 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria. Arthritis Rheumatol 2023;75:1687–702. PMID:37635643 https://doi.org/10.1002/art.42624
153. Tepper NK, Marchbanks PA, Curtis KM. Superficial venous disease and combined hormonal contraceptives: a systematic review. Contraception 2016;94:275–9. PMID:25835269 https://doi.org/10.1016/j.contraception.2015.03.010
154. Tepper NK, Paulen ME, Marchbanks PA, Curtis KM. Safety of contraceptive use among women with peripartum cardiomyopathy: a systematic review. Contraception 2010;82:95–101. PMID:20682147 https://doi.org/10.1016/j.contraception.2010.02.004
155. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
156. Molnar AO, Bota SE, McArthur E, et al. Risk and complications of venous thromboembolism in dialysis patients. Nephrol Dial Transplant 2018;33:874–80. PMID:28992258 https://doi.org/10.1093/ndt/gfx212
157. Tveit DP, Hypolite IO, Hshieh P, et al. Chronic dialysis patients have high risk for pulmonary embolism. Am J Kidney Dis 2002;39:1011–7. PMID:11979344 https://doi.org/10.1053/ajkd.2002.32774

158. Wang IK, Shen TC, Muo CH, Yen TH, Sung FC. Risk of pulmonary embolism in patients with end-stage renal disease receiving long-term dialysis. Nephrol Dial Transplant 2017;32:1386–93. PMID:27448674
159. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81. PMID:15226516 https://doi.org/10.1093/rheumatology/keh282
160. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9. PMID:15280571 https://doi.org/10.1093/rheumatology/keh331
161. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8. PMID:12180730
162. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7. PMID:11665973 https://doi.org/10.1002/1529-0131(200110)44:10<2331::AID-ART395>3.0.CO;2-I
163. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33. PMID:1771400 https://doi.org/10.3109/03009749109096822
164. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30. PMID:8448613 https://doi.org/10.1093/rheumatology/32.3.227
165. Jungers P, Dougados M, Pélissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23. PMID:7092961 https://doi.org/10.1002/art.1780250603
166. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15. PMID:9048514 https://doi.org/10.1093/oxfordjournals.aje.a009122
167. McAlindon T, Giannotta L, Taub N, D'Cruz D, Hughes G. Environmental factors predicting nephritis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4. PMID:8257208 https://doi.org/10.1136/ard.52.10.720
168. McDonald J, Stewart J, Urowitz MB, Gladman DD. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60. PMID:1540039 https://doi.org/10.1136/ard.51.1.56
169. Mintz G, Gutiérrez G, Delezé M, Rodríguez E. Contraception with progestagens in systemic lupus erythematosus. Contraception 1984;30:29–38. PMID:6434228 https://doi.org/10.1016/0010-7824(84)90076-3
170. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45. PMID:7654797 https://doi.org/10.1002/art.1790080305
171. Petri M. Lupus in Baltimore: evidence-based 'clinical pearls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3. PMID:16425579 https://doi.org/10.1191/0961203305lu2230xx
172. Petri M, Kim MY, Kalunian KC, et al.; OC-SELENA Trial. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8. PMID:16354891 https://doi.org/10.1056/NEJMoa051135
173. Sánchez-Guerrero J, Uribe AG, Jiménez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49. PMID:16354890 https://doi.org/10.1056/NEJMoa050817
174. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12. PMID:16082635 https://doi.org/10.1002/art.21314
175. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3. PMID:16202726 https://doi.org/10.1016/j.ajog.2005.05.002
176. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6. PMID:12465147
177. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5. PMID:1251849 https://doi.org/10.1016/0002-9343(76)90431-9
178. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, Nantiruj K, Sumethkul V, Janwityanujit S. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51. PMID:17805483 https://doi.org/10.1007/s10067-007-0721-z
179. Wahl DG, Guillemin F, de Maistre E, Perret C, Lecompte T, Thibaut G. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73. PMID:9229367 https://doi.org/10.1177/096120339700600510
180. Farr SL, Folger SG, Paulen ME, Curtis KM. Safety of contraceptive methods for women with rheumatoid arthritis: a systematic review. Contraception 2010;82:64–71. PMID:20682144 https://doi.org/10.1016/j.contraception.2010.02.003
181. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition. Cephalalgia 2018;38:1–211. https://www.ichd-3.org/wp-content/uploads/2018/01/The-International-Classification-of-Headache-Disorders-3rd-Edition-2018.pdf
182. Tepper NK, Whiteman MK, Zapata LB, Marchbanks PA, Curtis KM. Safety of hormonal contraceptives among women with migraine: a systematic review. Contraception 2016;94:630–40. PMID:27153744 https://doi.org/10.1016/j.contraception.2016.04.016
183. Xu Z, Li Y, Tang S, Huang X, Chen T. Current use of oral contraceptives and the risk of first-ever ischemic stroke: a meta-analysis of observational studies. Thromb Res 2015;136:52–60. PMID:25936231 https://doi.org/10.1016/j.thromres.2015.04.021
184. Etminan M, Takkouche B, Isorna FC, Samii A. Risk of ischaemic stroke in people with migraine: systematic review and meta-analysis of observational studies. BMJ 2005;330:63. PMID:15596418 https://doi.org/10.1136/bmj.38302.504063.8F
185. Schürks M, Rist PM, Bigal ME, Buring JE, Lipton RB, Kurth T. Migraine and cardiovascular disease: systematic review and meta-analysis. BMJ 2009;339(oct27 1):b3914. PMID:19861375 https://doi.org/10.1136/bmj.b3914
186. Spector JT, Kahn SR, Jones MR, Jayakumar M, Dalal D, Nazarian S. Migraine headache and ischemic stroke risk: an updated meta-analysis. Am J Med 2010;123:612–24. PMID:20493462 https://doi.org/10.1016/j.amjmed.2009.12.021
187. Zapata LB, Oduyebo T, Whiteman MK, Houtchens MK, Marchbanks PA, Curtis KM. Contraceptive use among women with multiple sclerosis: a systematic review. Contraception 2016;94:612–20. PMID:27452316 https://doi.org/10.1016/j.contraception.2016.07.013
188. Pagano HP, Zapata LB, Berry-Bibee EN, Nanda K, Curtis KM. Safety of hormonal contraception and intrauterine devices among women with depressive and bipolar disorders: a systematic review. Contraception 2016;94:641–9. PMID:27364100 https://doi.org/10.1016/j.contraception.2016.06.012
189. Iyer V, Farquhar C, Jepson R. Oral contraceptive pills for heavy menstrual bleeding. Cochrane Database Syst Rev 2000;(2):CD000154. PMID:10796696 https://doi.org/10.1002/14651858.CD000154

190. Davis L, Kennedy SS, Moore J, Prentice A. Oral contraceptives for pain associated with endometriosis. Cochrane Database Syst Rev 2007;(3):CD001019. PMID:17636650 https://doi.org/10.1002/14651858.CD001019.pub2
191. Hendrix SL, Alexander NJ. Primary dysmenorrhea treatment with a desogestrel-containing low-dose oral contraceptive. Contraception 2002;66:393–9. PMID:12499030 https://doi.org/10.1016/S0010-7824(02)00414-6
192. Wong CL, Farquhar C, Roberts H. Oral contraceptive pills for primary dysmenorrhoea. Cochrane Database Syst Rev 2001;(2). http://dx.doi.org/10.1002/14651858.CD002120.pub3
193. Gaffield ME, Kapp N, Curtis KM. Combined oral contraceptive and intrauterine device use among women with gestational trophoblastic disease. Contraception 2009;80:363–71. PMID:19751859 https://doi.org/10.1016/j.contraception.2009.03.022
194. Smith JS, Green J, Berrington de Gonzalez A, et al. Cervical cancer and use of hormonal contraceptives: a systematic review. Lancet 2003;361:1159–67. PMID:12686037 https://doi.org/10.1016/S0140-6736(03)12949-2
195. Black MM, Barclay THC, Polednak A, Kwon CS, Leis HP Jr, Pilnik S. Family history, oral contraceptive usage, and breast cancer. Cancer 1983;51:2147–51. PMID:6839302 https://doi.org/10.1002/1097-0142(19830601)51:11<2147::AID-CNCR2820511133>3.0.CO;2-X
196. Brinton LA, Hoover R, Szklo M, Fraumeni JF Jr. Oral contraceptives and breast cancer. Int J Epidemiol 1982;11:316–22. PMID:7152784 https://doi.org/10.1093/ije/11.4.316
197. Brohet RM, Goldgar DE, Easton DF, et al. Oral contraceptives and breast cancer risk in the international BRCA1/2 carrier cohort study: a report from EMBRACE, GENEPSO, GEO-HEBON, and the IBCCS Collaborating Group. J Clin Oncol 2007;25:3831–6. PMID:17635951 https://doi.org/10.1200/JCO.2007.11.1179
198. Claus EB, Stowe M, Carter D. Oral contraceptives and the risk of ductal breast carcinoma in situ. Breast Cancer Res Treat 2003;81:129–36. PMID:14572155 https://doi.org/10.1023/A:1025728524310
199. Collaborative Group on Hormonal Factors in Breast Cancer. Familial breast cancer: collaborative reanalysis of individual data from 52 epidemiological studies including 58,209 women with breast cancer and 101,986 women without the disease. Lancet 2001;358:1389–99. PMID:11705483 https://doi.org/10.1016/S0140-6736(01)06524-2
200. Grabrick DM, Hartmann LC, Cerhan JR, et al. Risk of breast cancer with oral contraceptive use in women with a family history of breast cancer. JAMA 2000;284:1791–8. PMID:11025831 https://doi.org/10.1001/jama.284.14.1791
201. Gronwald J, Byrski T, Huzarski T, et al. Influence of selected lifestyle factors on breast and ovarian cancer risk in BRCA1 mutation carriers from Poland. Breast Cancer Res Treat 2006;95:105–9. PMID:16261399 https://doi.org/10.1007/s10549-005-9051-5
202. Haile RW, Thomas DC, McGuire V, et al.; kConFab Investigators; Ontario Cancer Genetics Network Investigators. BRCA1 and BRCA2 mutation carriers, oral contraceptive use, and breast cancer before age 50. Cancer Epidemiol Biomarkers Prev 2006;15:1863–70. PMID:17021353 https://doi.org/10.1158/1055-9965.EPI-06-0258
203. Harris NV, Weiss NS, Francis AM, Polissar L. Breast cancer in relation to patterns of oral contraceptive use. Am J Epidemiol 1982;116:643–51. PMID:7137151 https://doi.org/10.1093/oxfordjournals.aje.a113447
204. Hennekens CH, Speizer FE, Lipnick RJ, et al. A case-control study of oral contraceptive use and breast cancer. J Natl Cancer Inst 1984;72:39–42. PMID:6363789 https://doi.org/10.1093/jnci/72.1.39
205. Jernström H, Loman N, Johannsson OT, Borg A, Olsson H. Impact of teenage oral contraceptive use in a population-based series of early-onset breast cancer cases who have undergone BRCA mutation testing. Eur J Cancer 2005;41:2312–20. PMID:16118051 https://doi.org/10.1016/j.ejca.2005.03.035
206. Marchbanks PA, McDonald JA, Wilson HG, et al. Oral contraceptives and the risk of breast cancer. N Engl J Med 2002;346:2025–32. PMID:12087137 https://doi.org/10.1056/NEJMoa013202
207. Milne RL, Knight JA, John EM, et al. Oral contraceptive use and risk of early-onset breast cancer in carriers and noncarriers of BRCA1 and BRCA2 mutations. Cancer Epidemiol Biomarkers Prev 2005;14:350–6. PMID:15734957 https://doi.org/10.1158/1055-9965.EPI-04-0376
208. Narod SA, Dubé MP, Klijn J, et al. Oral contraceptives and the risk of breast cancer in BRCA1 and BRCA2 mutation carriers. J Natl Cancer Inst 2002;94:1773–9. PMID:12464649 https://doi.org/10.1093/jnci/94.23.1773
209. Rosenberg L, Palmer JR, Rao RS, et al. Case-control study of oral contraceptive use and risk of breast cancer. Am J Epidemiol 1996;143:25–37. PMID:8533744 https://doi.org/10.1093/oxfordjournals.aje.a008654
210. Silvera SAN, Miller AB, Rohan TE. Oral contraceptive use and risk of breast cancer among women with a family history of breast cancer: a prospective cohort study. Cancer Causes Control 2005;16:1059–63. PMID:16184471 https://doi.org/10.1007/s10552-005-0343-1
211. Ursin G, Henderson BE, Haile RW, et al. Does oral contraceptive use increase the risk of breast cancer in women with BRCA1/BRCA2 mutations more than in other women? Cancer Res 1997;57:3678–81. PMID:9288771
212. Ursin G, Ross RK, Sullivan-Halley J, Hanisch R, Henderson B, Bernstein L. Use of oral contraceptives and risk of breast cancer in young women. Breast Cancer Res Treat 1998;50:175–84. PMID:9822222 https://doi.org/10.1023/A:1006037823178
213. Curtis KM, Hannaford PC, Rodriguez MI, Chipato T, Steyn PS, Kiarie JN. Hormonal contraception and HIV acquisition among women: an updated systematic review. BMJ Sex Reprod Health 2020;46:8–16. PMID:31919239 https://doi.org/10.1136/bmjsrh-2019-200509
214. Tepper NK, Curtis KM, Cox S, Whiteman MK. Update to U.S. medical eligibility criteria for contraceptive use, 2016: updated recommendations for the use of contraception among women at high risk for HIV infection. MMWR Morb Mortal Wkly Rep 2020;69:405–10. PMID:32271729 https://doi.org/10.15585/mmwr.mm6914a3
215. Polis CB, Phillips SJ, Curtis KM. Hormonal contraceptive use and female-to-male HIV transmission: a systematic review of the epidemiologic evidence. AIDS 2013;27:493–505. PMID:23079808 https://doi.org/10.1097/QAD.0b013e32835ad539
216. Phillips SJ, Polis CB, Curtis KM. The safety of hormonal contraceptives for women living with HIV and their sexual partners. Contraception 2016;93:11–6. PMID:26515194 https://doi.org/10.1016/j.contraception.2015.10.002
217. Phillips SJ, Curtis KM, Polis CB. Effect of hormonal contraceptive methods on HIV disease progression: a systematic review. AIDS 2013;27:787–94. PMID:23135169 https://doi.org/10.1097/QAD.0b013e32835bb672
218. el-Raghy I, Back DJ, Osman F, Orme ML, Fathalla M. Contraceptive steroid concentrations in women with early active schistosomiasis: lack of effect of antischistosomal drugs. Contraception 1986;33:373–7. PMID:3089682 https://doi.org/10.1016/0010-7824(86)90099-5
219. Gad-el-Mawla N, Abdallah A. Liver function in bilharzial females receiving contraceptive pills. Acta Hepatosplenol 1969;16:308–10. PMID:5358869
220. Gad-el-Mawla N, el-Roubi O, Sabet S, Abdallah A. Plasma lipids and lipoproteins in bilharzial females during oral contraceptive therapy. J Egypt Med Assoc 1972;55:137–47. PMID:5051373
221. Shaaban MM, Hammad WA, Falthalla MF, et al. Effects of oral contraception on liver function tests and serum proteins in women with active schistosomiasis. Contraception 1982;26:75–82. PMID:7128137 https://doi.org/10.1016/0010-7824(82)90174-3

222. Shaaban MM, Ghaneimah SA, Mohamed MA, Abdel-Chani S, Mostafa SA. Effect of oral contraception on serum bile acid. Int J Gynaecol Obstet 1984;22:111–5. PMID:6145634 https://doi.org/10.1016/0020-7292(84)90023-7
223. Sy FS, Osteria TS, Opiniano V, Gler S. Effect of oral contraceptive on liver function tests of women with schistosomiasis in the Philippines. Contraception 1986;34:283–94. PMID:3098499 https://doi.org/10.1016/0010-7824(86)90009-0
224. Tagy AH, Saker ME, Moussa AA, Kolgah A. The effect of low-dose combined oral contraceptive pills versus injectable contraceptive (Depot Provera) on liver function tests of women with compensated bilharzial liver fibrosis. Contraception 2001;64:173–6. PMID:11704097 https://doi.org/10.1016/S0010-7824(01)00248-7
225. Beck P, Wells SA. Comparison of the mechanisms underlying carbohydrate intolerance in subclinical diabetic women during pregnancy and during post-partum oral contraceptive steroid treatment. J Clin Endocrinol Metab 1969;29:807–18. PMID:4978229 https://doi.org/10.1210/jcem-29-6-807
226. Kjos SL, Peters RK, Xiang A, Thomas D, Schaefer U, Buchanan TA. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280:533–8. PMID:9707143 https://doi.org/10.1001/jama.280.6.533
227. Kung AW, Ma JT, Wong VC, et al. Glucose and lipid metabolism with triphasic oral contraceptives in women with history of gestational diabetes. Contraception 1987;35:257–69. PMID:3111786 https://doi.org/10.1016/0010-7824(87)90027-8
228. Rådberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in gestational diabetic women during contraceptive treatment: effects on glucose tolerance and fatty acid composition of serum lipids. Gynecol Obstet Invest 1982;13:17–29. PMID:7035304 https://doi.org/10.1159/000299480
229. Skouby SO, Andersen O, Kühl C. Oral contraceptives and insulin receptor binding in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1986;155:802–7. PMID:3766633 https://doi.org/10.1016/S0002-9378(86)80024-2
230. Skouby SO, Andersen O, Saurbrey N, Kühl C. Oral contraception and insulin sensitivity: in vivo assessment in normal women and women with previous gestational diabetes. J Clin Endocrinol Metab 1987;64:519–23. PMID:3102539 https://doi.org/10.1210/jcem-64-3-519
231. Skouby SO, Mølsted-Pedersen L, Kühl C. Low dosage oral contraception in women with previous gestational diabetes. Obstet Gynecol 1982;59:325–8. PMID:6804901
232. Xiang AH, Kawakubo M, Kjos SL, Buchanan TA. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006;29:613–7. PMID:16505515 https://doi.org/10.2337/diacare.29.03.06.dc05-1940
233. Kjos SL, Shoupe D, Douyan S, et al. Effect of low-dose oral contraceptives on carbohydrate and lipid metabolism in women with recent gestational diabetes: results of a controlled, randomized, prospective study. Am J Obstet Gynecol 1990;163:1822–7. PMID:2256489 https://doi.org/10.1016/0002-9378(90)90757-X
234. Rådberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in women with previous gestational diabetes during contraceptive treatment: effects on serum lipids and high density lipoproteins. Acta Endocrinol (Copenh) 1982;101:134–9. PMID:7124287 https://doi.org/10.1530/acta.0.1010134
235. Skouby SO, Kühl C, Mølsted-Pedersen L, Petersen K, Christensen MS. Triphasic oral contraception: metabolic effects in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1985;153:495–500. PMID:3933351 https://doi.org/10.1016/0002-9378(85)90460-0
236. Beck P, Arnett DM, Alsever RN, Eaton RP. Effect of contraceptive steroids on arginine-stimulated glucagon and insulin secretion in women. II. Carbohydrate and lipid physiology in insulin-dependent diabetics. Metabolism 1976;25:23–31. PMID:1246206 https://doi.org/10.1016/0026-0495(76)90156-6
237. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of Norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynaecol Res 2000;26:17–26. PMID:10761326 https://doi.org/10.1111/j.1447-0756.2000.tb01195.x
238. Garg SK, Chase HP, Marshall G, Hoops SL, Holmes DL, Jackson WE. Oral contraceptives and renal and retinal complications in young women with insulin-dependent diabetes mellitus. JAMA 1994;271:1099–102. PMID:8151852 https://doi.org/10.1001/jama.1994.03510380055037
239. Grigoryan OR, Grodnitskaya EE, Andreeva EN, Shestakova MV, Melnichenko GA, Dedov II. Contraception in perimenopausal women with diabetes mellitus. Gynecol Endocrinol 2006;22:198–206. PMID:16723306 https://doi.org/10.1080/09513590600624317
240. Margolis KL, Adami H-O, Luo J, Ye W, Weiderpass E. A prospective study of oral contraceptive use and risk of myocardial infarction among Swedish women. Fertil Steril 2007;88:310–6. PMID:17624338 https://doi.org/10.1016/j.fertnstert.2006.11.206
241. Petersen KR, Skouby SO, Jespersen J. Balance of coagulation activity with fibrinolysis during use of oral contraceptives in women with insulin-dependent diabetes mellitus. Int J Fertil Menopausal Stud 1995;40(Suppl 2):105–11. PMID:8574252
242. Petersen KR, Skouby SO, Sidelmann J, Jespersen J. Assessment of endothelial function during oral contraception in women with insulin-dependent diabetes mellitus. Metabolism 1994;43:1379–83. PMID:7968593 https://doi.org/10.1016/0026-0495(94)90031-0
243. Rådberg T, Gustafson A, Skryten A, Karlsson K. Oral contraception in diabetic women. A cross-over study on serum and high density lipoprotein (HDL) lipids and diabetes control during progestogen and combined estrogen/progestogen contraception. Horm Metab Res 1982;14:61–5. PMID:7040192
244. Skouby SO, Jensen BM, Kühl C, Mølsted-Pedersen L, Svenstrup B, Nielsen J. Hormonal contraception in diabetic women: acceptability and influence on diabetes control and ovarian function of a nonalkylated estrogen/progestogen compound. Contraception 1985;32:23–31. PMID:4053603 https://doi.org/10.1016/0010-7824(85)90113-1
245. Skouby SO, Mølsted-Pedersen L, Kühl C, Bennet P. Oral contraceptives in diabetic women: metabolic effects of four compounds with different estrogen/progestogen profiles. Fertil Steril 1986;46:858–64. PMID:3781003 https://doi.org/10.1016/S0015-0282(16)49825-0
246. Zapata LB, Paulen ME, Cansino C, Marchbanks PA, Curtis KM. Contraceptive use among women with inflammatory bowel disease: a systematic review. Contraception 2010;82:72–85. PMID:20682145 https://doi.org/10.1016/j.contraception.2010.02.012
247. Whiteman MK, Oduyebo T, Zapata LB, Walker S, Curtis KM. Contraceptive safety among women with cystic fibrosis: a systematic review. Contraception 2016;94:621–9. PMID:27287694 https://doi.org/10.1016/j.contraception.2016.05.016
248. Brunson A, Keegan T, Mahajan A, White R, Wun T. High incidence of venous thromboembolism recurrence in patients with sickle cell disease. Am J Hematol 2019;94:862–70. PMID:31074115 https://doi.org/10.1002/ajh.25508
249. Naik RP, Streiff MB, Haywood C Jr, Segal JB, Lanzkron S. Venous thromboembolism incidence in the Cooperative Study of Sickle Cell Disease. J Thromb Haemost 2014;12:2010–6. PMID:25280124 https://doi.org/10.1111/jth.12744
250. Noubiap JJ, Temgoua MN, Tankeu R, Tochie JN, Wonkam A, Bigna JJ. Sickle cell disease, sickle trait and the risk for venous thromboembolism: a systematic review and meta-analysis. Thromb J 2018;16:27. PMID:30305805 https://doi.org/10.1186/s12959-018-0179-z

251. Ohene-Frempong K, Weiner SJ, Sleeper LA, et al. Cerebrovascular accidents in sickle cell disease: rates and risk factors. Blood 1998;91:288–94. PMID:9414296
252. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Recommendations for the use of antiretroviral drugs during pregnancy and interventions to reduce perinatal HIV transmission in the United States. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/en/guidelines/perinatal/recommendations-arv-drugs-pregnancy-overview
253. Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in adults and adolescents with HIV. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/sites/default/files/guidelines/documents/adult-adolescent-arv/guidelines-adult-adolescent-arv.pdf
254. Aweeka FT, Rosenkranz SL, Segal Y, et al.; NIAID AIDS Clinical Trials Group. The impact of sex and contraceptive therapy on the plasma and intracellular pharmacokinetics of zidovudine. AIDS 2006;20:1833–41. PMID:16954724 https://doi.org/10.1097/01.aids.0000244202.18629.36
255. Kearney BP, Mathias A. Lack of effect of tenofovir disoproxil fumarate on pharmacokinetics of hormonal contraceptives. Pharmacotherapy 2009;29:924–9. PMID:19637945 https://doi.org/10.1592/phco.29.8.924
256. Todd CS, Deese J, Wang M, et al.; FEM-PrEP Study Group. Sino-implant (II) continuation and effect of concomitant tenofovir disoproxil fumarate-emtricitabine use on plasma levonorgestrel concentrations among women in Bondo, Kenya. Contraception 2015;91:248–52. PMID:25459097 https://doi.org/10.1016/j.contraception.2014.10.008
257. Murnane PM, Heffron R, Ronald A, et al.; Partners PrEP Study Team. Pre-exposure prophylaxis for HIV-1 prevention does not diminish the pregnancy prevention effectiveness of hormonal contraception. AIDS 2014;28:1825–30. PMID:24785951 https://doi.org/10.1097/QAD.0000000000000290
258. Kasonde M, Niska RW, Rose C, et al. Bone mineral density changes among HIV-uninfected young adults in a randomised trial of pre-exposure prophylaxis with tenofovir-emtricitabine or placebo in Botswana. PLoS One 2014;9:e90111. PMID:24625530 https://doi.org/10.1371/journal.pone.0090111
259. Callahan R, Nanda K, Kapiga S, et al.; FEM-PrEP Study Group. Pregnancy and contraceptive use among women participating in the FEM-PrEP trial. J Acquir Immune Defic Syndr 2015;68:196–203. PMID:25590272 https://doi.org/10.1097/QAI.0000000000000413
260. Patel RC, Onono M, Gandhi M, et al. Pregnancy rates in HIV-positive women using contraceptives and efavirenz-based or nevirapine-based antiretroviral therapy in Kenya: a retrospective cohort study. Lancet HIV 2015;2:e474–82. PMID:26520927 https://doi.org/10.1016/S2352-3018(15)00184-8
261. Pyra M, Heffron R, Mugo NR, et al.; Partners in Prevention HSVHIV Transmission Study and Partners PrEP Study Teams. Effectiveness of hormonal contraception in HIV-infected women using antiretroviral therapy. AIDS 2015;29:2353–9. PMID:26544706 https://doi.org/10.1097/QAD.0000000000000827
262. Clark RA, Theall K. Population-based study evaluating association between selected antiretroviral therapies and potential oral contraceptive failure. J Acquir Immune Defic Syndr 2004;37:1219–20. PMID:15319685 https://doi.org/10.1097/01.qai.0000136724.15758.ae
263. Landolt NK, Phanuphak N, Ubolyam S, et al. Efavirenz, in contrast to nevirapine, is associated with unfavorable progesterone and antiretroviral levels when coadministered with combined oral contraceptives. J Acquir Immune Defic Syndr 2013;62:534–9. PMID:23187949 https://doi.org/10.1097/QAI.0b013e31827e8f98
264. Sevinsky H, Eley T, Persson A, et al. The effect of efavirenz on the pharmacokinetics of an oral contraceptive containing ethinyl estradiol and norgestimate in healthy HIV-negative women. Antivir Ther 2011;16:149–56. PMID:21447863 https://doi.org/10.3851/IMP1725
265. Landolt NK, Phanuphak N, Ubolyam S, et al. Significant decrease of ethinylestradiol with nevirapine, and of etonogestrel with efavirenz in HIV-positive women. J Acquir Immune Defic Syndr 2014;66:e50–2. PMID:24608892 https://doi.org/10.1097/QAI.0000000000000134
266. Myer L, Carter RJ, Katyal M, Toro P, El-Sadr WM, Abrams EJ. Impact of antiretroviral therapy on incidence of pregnancy among HIV-infected women in Sub-Saharan Africa: a cohort study. PLoS Med 2010;7:e1000229. PMID:20161723 https://doi.org/10.1371/journal.pmed.1000229
267. Schöller-Gyüre M, Kakuda TN, Woodfall B, et al. Effect of steady-state etravirine on the pharmacokinetics and pharmacodynamics of ethinylestradiol and norethindrone. Contraception 2009;80:44–52. PMID:19501215 https://doi.org/10.1016/j.contraception.2009.01.009
268. Nanda K, Delany-Moretlwe S, Dubé K, et al. Nevirapine-based antiretroviral therapy does not reduce oral contraceptive effectiveness. AIDS 2013;27(Suppl 1):S17–25. PMID:24088680 https://doi.org/10.1097/QAD.0000000000000050
269. Stuart GS, Moses A, Corbett A, et al. Combined oral contraceptives and antiretroviral PK/PD in Malawian women: pharmacokinetics and pharmacodynamics of a combined oral contraceptive and a generic combined formulation antiretroviral in Malawi. J Acquir Immune Defic Syndr 2011;58:e40–3. PMID:21921726 https://doi.org/10.1097/QAI.0b013e31822b8bf8
270. Mildvan D, Yarrish R, Marshak A, et al. Pharmacokinetic interaction between nevirapine and ethinyl estradiol/norethindrone when administered concurrently to HIV-infected women. J Acquir Immune Defic Syndr 2002;29:471–7. PMID:11981363 https://doi.org/10.1097/00042560-200204150-00007
271. Muro E, Droste JAH, Hofstede HT, Bosch M, Dolmans W, Burger DM. Nevirapine plasma concentrations are still detectable after more than 2 weeks in the majority of women receiving single-dose nevirapine: implications for intervention studies. J Acquir Immune Defic Syndr 2005;39:419–21. PMID:16010163 https://doi.org/10.1097/01.qai.0000167154.37357.f9
272. Crauwels HM, van Heeswijk RP, Buelens A, Stevens M, Hoetelmans RM. Lack of an effect of rilpivirine on the pharmacokinetics of ethinylestradiol and norethindrone in healthy volunteers. Int J Clin Pharmacol Ther 2014;52:118–28. PMID:24161160 https://doi.org/10.5414/CP201943
273. Zhang J, Chung E, Yones C, et al. The effect of atazanavir/ritonavir on the pharmacokinetics of an oral contraceptive containing ethinyl estradiol and norgestimate in healthy women. Antivir Ther 2011;16:157–64. PMID:21447864 https://doi.org/10.3851/IMP1724
274. Sekar VJ, Lefebvre E, Guzman SS, et al. Pharmacokinetic interaction between ethinyl estradiol, norethindrone and darunavir with low-dose ritonavir in healthy women. Antivir Ther 2008;13:563–9. PMID:18672535 https://doi.org/10.1177/135965350801300415
275. Glaxo Smith Kline. Lexiva (fosamprenavir calcium) [Package insert]. Research Triangle Park, NC:Glaxo Smith Kline; 2015.
276. Vogler MA, Patterson K, Kamemoto L, et al. Contraceptive efficacy of oral and transdermal hormones when co-administered with protease inhibitors in HIV-1-infected women: pharmacokinetic results of ACTG trial A5188. J Acquir Immune Defic Syndr 2010;55:473–82. PMID:20842042 https://doi.org/10.1097/QAI.0b013e3181eb5ff5
277. Fröhlich M, Burhenne J, Martin-Facklam M, et al. Oral contraception does not alter single dose saquinavir pharmacokinetics in women. Br J Clin Pharmacol 2004;57:244–52. PMID:14998420 https://doi.org/10.1111/j.1365-2125.2003.01983.x
278. Boehringer Ingelheim Pharmaceuticals. Aptivus (tipranavir) [Package insert]. Ridgefield, CT: Boehringer Ingelheim Pharmaceuticals; 2005.

279. Bristol-Myers Squibb. Reyataz (atazanavir sulfate) [Package insert]. Princeton, NJ: Bristol-Myers Squibb; 2003.
280. Abel S, Russell D, Whitlock LA, Ridgway CE, Muirhead GJ. Effect of maraviroc on the pharmacokinetics of midazolam, lamivudine/zidovudine, and ethinyloestradiol/levonorgestrel in healthy volunteers. Br J Clin Pharmacol 2008;65(Suppl 1):19–26. PMID:18333862 https://doi.org/10.1111/j.1365-2125.2008.03132.x
281. Anderson MS, Hanley WD, Moreau AR, et al. Effect of raltegravir on estradiol and norgestimate plasma pharmacokinetics following oral contraceptive administration in healthy women. Br J Clin Pharmacol 2011;71:616–20. PMID:21395656 https://doi.org/10.1111/j.1365-2125.2010.03885.x
282. Song IH, Borland J, Chen S, Wajima T, Peppercorn AF, Piscitelli SC. Dolutegravir has no effect on the pharmacokinetics of oral contraceptives with norgestimate and ethinyl estradiol. Ann Pharmacother 2015;49:784–9. PMID:25862012 https://doi.org/10.1177/1060028015580637
283. Gilead Sciences. Vitekta (elvitegravir) [Package insert]. Foster City, CA: Gilead Sciences; 2012.
284. Back DJ, Bates M, Bowden A, et al. The interaction of phenobarbital and other anticonvulsants with oral contraceptive steroid therapy. Contraception 1980;22:495–503. PMID:7471739 https://doi.org/10.1016/0010-7824(80)90102-X
285. Doose DR, Wang SS, Padmanabhan M, Schwabe S, Jacobs D, Bialer M. Effect of topiramate or carbamazepine on the pharmacokinetics of an oral contraceptive containing norethindrone and ethinyl estradiol in healthy obese and nonobese female subjects. Epilepsia 2003;44:540–9. PMID:12681003 https://doi.org/10.1046/j.1528-1157.2003.55602.x
286. Fattore C, Cipolla G, Gatti G, et al. Induction of ethinylestradiol and levonorgestrel metabolism by oxcarbazepine in healthy women. Epilepsia 1999;40:783–7. PMID:10368079 https://doi.org/10.1111/j.1528-1157.1999.tb00779.x
287. Rosenfeld WE, Doose DR, Walker SA, Nayak RK. Effect of topiramate on the pharmacokinetics of an oral contraceptive containing norethindrone and ethinyl estradiol in patients with epilepsy. Epilepsia 1997;38:317–23. PMID:9070594 https://doi.org/10.1111/j.1528-1157.1997.tb01123.x
288. Gaffield ME, Culwell KR, Lee CR. The use of hormonal contraception among women taking anticonvulsant therapy. Contraception 2011;83:16–29. PMID:21134499 https://doi.org/10.1016/j.contraception.2010.06.013
289. Christensen J, Petrenaite V, Atterman J, et al. Oral contraceptives induce lamotrigine metabolism: evidence from a double-blind, placebo-controlled trial. Epilepsia 2007;48:484–9. PMID:17346247 https://doi.org/10.1111/j.1528-1167.2007.00997.x
290. Contin M, Albani F, Ambrosetto G, et al. Variation in lamotrigine plasma concentrations with hormonal contraceptive monthly cycles in patients with epilepsy. Epilepsia 2006;47:1573–5. PMID:16981875 https://doi.org/10.1111/j.1528-1167.2006.00558.x
291. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7. PMID:16146436 https://doi.org/10.1111/j.1528-1167.2005.10105.x
292. Sabers A, Buchholt JM, Uldall P, Hansen EL. Lamotrigine plasma levels reduced by oral contraceptives. Epilepsy Res 2001;47:151–4. PMID:11673029 https://doi.org/10.1016/S0920-1211(01)00305-9
293. Sabers A, Ohman I, Christensen J, Tomson T. Oral contraceptives reduce lamotrigine plasma levels. Neurology 2003;61:570–1. PMID:12939444 https://doi.org/10.1212/01.WNL.0000076485.09353.7A
294. Back DJ, Breckenridge AM, MacIver M, et al. The effects of ampicillin on oral contraceptive steroids in women. Br J Clin Pharmacol 1982;14:43–8. PMID:6809025 https://doi.org/10.1111/j.1365-2125.1982.tb04932.x
295. Back DJ, Grimmer SF, Orme ML, Proudlove C, Mann RD, Breckenridge AM. Evaluation of Committee on Safety of Medicines yellow card reports on oral contraceptive-drug interactions with anticonvulsants and antibiotics. Br J Clin Pharmacol 1988;25:527–32. PMID:3408633 https://doi.org/10.1111/j.1365-2125.1988.tb03341.x
296. Back DJ, Tjia J, Martin C, et al. The lack of interaction between temafloxacin and combined oral contraceptive steroids. Contraception 1991;43:317–23. PMID:1906791 https://doi.org/10.1016/0010-7824(91)90070-V
297. Bacon JF, Shenfield GM. Pregnancy attributable to interaction between tetracycline and oral contraceptives. BMJ 1980;280:293. PMID:7357347 https://doi.org/10.1136/bmj.280.6210.293
298. Bainton R. Interaction between antibiotic therapy and contraceptive medication. Oral Surg Oral Med Oral Pathol 1986;61:453–5. PMID:3459119 https://doi.org/10.1016/0030-4220(86)90385-3
299. Bollen M. Use of antibiotics when taking the oral contraceptive pill. [comment]. Aust Fam Physician 1995;24:928–9. PMID:7794163
300. Bromham DR, Cartmill RS. Knowledge and use of secondary contraception among patients requesting termination of pregnancy. BMJ 1993;306:556–7. PMID:8461770 https://doi.org/10.1136/bmj.306.6877.556
301. Côté J. Interaction of griseofulvin and oral contraceptives. [Comment]. J Am Acad Dermatol 1990;22:124–5. PMID:2298948 https://doi.org/10.1016/S0190-9622(08)80010-2
302. Csemiczky G, Alvendal C, Landgren BM. Risk for ovulation in women taking a low-dose oral contraceptive (Microgynon) when receiving antibacterial treatment with a fluoroquinolone (ofloxacin). Adv Contracept 1996;12:101–9. PMID:8863905 https://doi.org/10.1007/BF01849631
303. de Groot AC, Eshuis H, Stricker BH. [Inefficacy of oral contraception during use of minocycline]. Ned Tijdschr Geneeskd 1990;134:1227–9. PMID:2143563
304. DeSano EA Jr, Hurley SC. Possible interactions of antihistamines and antibiotics with oral contraceptive effectiveness. Fertil Steril 1982;37:853–4. PMID:6123451 https://doi.org/10.1016/S0015-0282(16)46350-8
305. Donley TG, Smith RF, Roy B. Reduced oral contraceptive effectiveness with concurrent antibiotic use: a protocol for prescribing antibiotics to women of childbearing age. Compendium 1990;11:392–6. PMID:2083416
306. Friedman CI, Huneke AL, Kim MH, Powell J. The effect of ampicillin on oral contraceptive effectiveness. Obstet Gynecol 1980;55:33–7. PMID:7188714
307. Grimmer SF, Allen WL, Back DJ, Breckenridge AM, Orme M, Tjia J. The effect of cotrimoxazole on oral contraceptive steroids in women. Contraception 1983;28:53–9. PMID:6414761 https://doi.org/10.1016/S0010-7824(83)80005-5
308. Helms SE, Bredle DL, Zajic J, Jarjoura D, Brodell RT, Krishnarao I. Oral contraceptive failure rates and oral antibiotics. J Am Acad Dermatol 1997;36:705–10. PMID:9146531 https://doi.org/10.1016/S0190-9622(97)80322-2
309. Hempel E, Böhm W, Carol W, Klinger G. [Enzyme induction by drugs and hormonal contraception]. Zentralbl Gynakol 1973;95:1451–7. PMID:4129505
310. Hempel E, Zorn C, Graf K. [Effect of chemotherapy agents and antibiotics on hormonal contraception]. Z Arztl Fortbild (Jena) 1978;72:924–6. PMID:726527
311. Hetényi G. Possible interactions between antibiotics and oral contraceptives. Ther Hung 1989;37:86–9. PMID:2686086
312. Hughes BR, Cunliffe WJ. Interactions between the oral contraceptive pill and antibiotics. [Comment]. Br J Dermatol 1990;122:717–8. PMID:2141276 https://doi.org/10.1111/j.1365-2133.1990.tb07299.x

313. Joshi JV, Joshi UM, Sankholi GM, et al. A study of interaction of low-dose combination oral contraceptive with ampicillin and metronidazole. Contraception 1980;22:643–52. PMID:7214911 https://doi.org/10.1016/0010-7824(80)90089-X
314. Kakouris H, Kovacs GT. Pill failure and nonuse of secondary precautions. Br J Fam Plann 1992;18:41–4.
315. Kakouris H, Kovacs GT. How common are predisposing factors to pill failure among pill users? Br J Fam Plann 1994;20:33–5.
316. Kovacs GT, Riddoch G, Duncombe P, et al. Inadvertent pregnancies in oral contraceptive users. Med J Aust 1989;150:549–51. PMID:2716563 https://doi.org/10.5694/j.1326-5377.1989.tb136691.x
317. Lequeux A. [Pregnancy under oral contraceptives after treatment with tetracycline]. Louv Med 1980;99:413–4. PMID:12336602
318. London BM, Lookingbill DP. Frequency of pregnancy in acne patients taking oral antibiotics and oral contraceptives. Arch Dermatol 1994;130:392–3. PMID:8129425 https://doi.org/10.1001/archderm.1994.01690030128027
319. Maggiolo F, Puricelli G, Dottorini M, Caprioli S, Bianchi W, Suter F. The effect of ciprofloxacin on oral contraceptive steroid treatments. Drugs Exp Clin Res 1991;17:451–4. PMID:1822438
320. Murphy AA, Zacur HA, Charache P, Burkman RT. The effect of tetracycline on levels of oral contraceptives. Am J Obstet Gynecol 1991;164:28–33. PMID:1986620 https://doi.org/10.1016/0002-9378(91)90617-Z
321. Neely JL, Abate M, Swinker M, D'Angio R. The effect of doxycycline on serum levels of ethinyl estradiol, norethindrone, and endogenous progesterone. Obstet Gynecol 1991;77:416–20. PMID:1992409
322. Pillans PI, Sparrow MJ. Pregnancy associated with a combined oral contraceptive and itraconazole. [Comment]. N Z Med J 1993;106:436. PMID:8414287
323. Scholten PC, Droppert RM, Zwinkels MG, Moesker HL, Nauta JJ, Hoepelman IM. No interaction between ciprofloxacin and an oral contraceptive. Antimicrob Agents Chemother 1998;42:3266–8. PMID:9835524 https://doi.org/10.1128/AAC.42.12.3266
324. Silber TJ. Apparent oral contraceptive failure associated with antibiotic administration. J Adolesc Health Care 1983;4:287–9. PMID:6643209 https://doi.org/10.1016/S0197-0070(83)80014-X
325. Sparrow MJ. Pill method failures. N Z Med J 1987;100:102–5. PMID:3470667
326. Sparrow MJ. Pregnancies in reliable pill takers. N Z Med J 1989;102:575–7. PMID:2812591
327. Sparrow MJ. Pill method failures in women seeking abortion: fourteen years experience. N Z Med J 1998;111:386–8. PMID:9830420
328. van Dijke CP, Weber JC. Interaction between oral contraceptives and griseofulvin. Br Med J (Clin Res Ed) 1984;288:1125–6. PMID:6424759 https://doi.org/10.1136/bmj.288.6424.1125-a
329. Wermeling DP, Chandler MH, Sides GD, Collins D, Muse KN. Dirithromycin increases ethinyl estradiol clearance without allowing ovulation. Obstet Gynecol 1995;86:78–84. PMID:7784027 https://doi.org/10.1016/0029-7844(95)00075-3
330. Young LK, Farquhar CM, McCowan LM, Roberts HE, Taylor J. The contraceptive practices of women seeking termination of pregnancy in an Auckland clinic. N Z Med J 1994;107:189–92. PMID:8196861
331. Abrams LS, Skee D, Natarajan J, Wong FA. Pharmacokinetic overview of Ortho Evra/Evra. Fertil Steril 2002;77(Suppl 2):S3–12. PMID:11849630 https://doi.org/10.1016/S0015-0282(01)03261-7
332. Dogterom P, van den Heuvel MW, Thomsen T. Absence of pharmacokinetic interactions of the combined contraceptive vaginal ring NuvaRing with oral amoxicillin or doxycycline in two randomised trials. Clin Pharmacokinet 2005;44:429–38. PMID:15828855 https://doi.org/10.2165/00003088-200544040-00007
333. Devenport MH, Crook D, Wynn V, Lees LJ. Metabolic effects of low-dose fluconazole in healthy female users and non-users of oral contraceptives. Br J Clin Pharmacol 1989;27:851–9. PMID:2547410 https://doi.org/10.1111/j.1365-2125.1989.tb03449.x
334. Hilbert J, Messig M, Kuye O, Friedman H. Evaluation of interaction between fluconazole and an oral contraceptive in healthy women. Obstet Gynecol 2001;98:218–23. PMID:11506836
335. Kovács I, Somos P, Hámori M. Examination of the potential interaction between ketoconazole (Nizoral) and oral contraceptives with special regard to products of low hormone content (Rigevidon, Anteovin). Ther Hung 1986;34:167–70. PMID:3441880
336. Lunell NO, Pschera H, Zador G, Carlström K. Evaluation of the possible interaction of the antifungal triazole SCH 39304 with oral contraceptives in normal healthy women. Gynecol Obstet Invest 1991;32:91–7. PMID:1748330 https://doi.org/10.1159/000293003
337. McDaniel PA, Caldroney RD. Oral contraceptives and griseofulvin interactions. Drug Intell Clin Pharm 1986;20:384. PMID:3709350 https://doi.org/10.1177/106002808602000511
338. Meyboom RH, van Puijenbroek EP, Vinks MH, Lastdrager CJ. Disturbance of withdrawal bleeding during concomitant use of itraconazole and oral contraceptives. N Z Med J 1997;110:300. PMID:9293288
339. Rieth H, Sauerbrey N. [Interaction studies with fluconazole, a new triazole antifungal drug]. Wien Med Wochenschr 1989;139:370–4. PMID:2556861
340. Sinofsky FE, Pasquale SA. The effect of fluconazole on circulating ethinyl estradiol levels in women taking oral contraceptives. Am J Obstet Gynecol 1998;178:300–4. PMID:9500490 https://doi.org/10.1016/S0002-9378(98)80016-1
341. van Puijenbroek EP, Feenstra J, Meyboom RH. [Pill cycle disturbance in simultaneous use of itraconazole and oral contraceptives]. Ned Tijdschr Geneeskd 1998;142:146–9. PMID:9557015
342. Van Puijenbroek EP, Egberts AC, Meyboom RH, Leufkens HG. Signalling possible drug-drug interactions in a spontaneous reporting system: delay of withdrawal bleeding during concomitant use of oral contraceptives and itraconazole. Br J Clin Pharmacol 1999;47:689–93. PMID:10383548 https://doi.org/10.1046/j.1365-2125.1999.00957.x
343. Verhoeven CH, van den Heuvel MW, Mulders TM, Dieben TO. The contraceptive vaginal ring, NuvaRing, and antimycotic co-medication. Contraception 2004;69:129–32. PMID:14759617 https://doi.org/10.1016/j.contraception.2003.10.001
344. Back DJ, Breckenridge AM, Grimmer SF, Orme ML, Purba HS. Pharmacokinetics of oral contraceptive steroids following the administration of the antimalarial drugs primaquine and chloroquine. Contraception 1984;30:289–95. PMID:6439467 https://doi.org/10.1016/0010-7824(84)90092-1
345. Croft AM, Herxheimer A. Adverse effects of the antimalaria drug, mefloquine: due to primary liver damage with secondary thyroid involvement? BMC Public Health 2002;2:6. PMID:11914150 https://doi.org/10.1186/1471-2458-2-6
346. Karbwang J, Looareesuwan S, Back DJ, Migasana S, Bunnag D, Breckenridge AM. Effect of oral contraceptive steroids on the clinical course of malaria infection and on the pharmacokinetics of mefloquine in Thai women. Bull World Health Organ 1988;66:763–7. PMID:3266115
347. McGready R, Stepniewska K, Seaton E, et al. Pregnancy and use of oral contraceptives reduces the biotransformation of proguanil to cycloguanil. Eur J Clin Pharmacol 2003;59:553–7. PMID:12955370 https://doi.org/10.1007/s00228-003-0651-x
348. Wanwimolruk S, Kaewvichit S, Tanthayaphinant O, Suwannarach C, Oranratnachai A. Lack of effect of oral contraceptive use on the pharmacokinetics of quinine. Br J Clin Pharmacol 1991;31:179–81. PMID:2049234 https://doi.org/10.1111/j.1365-2125.1991.tb05509.x
349. Back DJ, Breckenridge AM, Crawford F, et al. The effect of rifampicin on norethisterone pharmacokinetics. Eur J Clin Pharmacol 1979;15:193–7. PMID:37091 https://doi.org/10.1007/BF00563105

350. Back DJ, Breckenridge AM, Crawford FE, et al. The effect of rifampicin on the pharmacokinetics of ethynylestradiol in women. Contraception 1980;21:135–43. PMID:7189454 https://doi.org/10.1016/0010-7824(80)90125-0
351. Barditch-Crovo P, Trapnell CB, Ette E, et al. The effects of rifampin and rifabutin on the pharmacokinetics and pharmacodynamics of a combination oral contraceptive. Clin Pharmacol Ther 1999;65:428–38. PMID:10223781 https://doi.org/10.1016/S0009-9236(99)70138-4
352. Bolt HM, Bolt M, Kappus H. Interaction of rifampicin treatment with pharmacokinetics and metabolism of ethinyloestradiol in man. Acta Endocrinol (Copenh) 1977;85:189–97. PMID:577076 https://doi.org/10.1530/acta.0.0850189
353. Gupta KC, Ali MY. Failure of oral contraceptive with rifampicin. Med J Zambia 1980;15:23. PMID:7269801
354. Hirsch A, Tillement JP, Chretien J. Effets contrariants de la rifampicine sur les contraceptifs oraux: a propos de trois grossesses non desiree chez trois malades. Rev Fr Mal Respir 1975;2:174–82.
355. Joshi JV, Joshi UM, Sankolli GM, et al. A study of interaction of a low-dose combination oral contraceptive with anti-tubercular drugs. Contraception 1980;21:617–29. PMID:7428368 https://doi.org/10.1016/0010-7824(80)90034-7
356. Kropp R. [Rifampicin and oral contraceptives (author's transl)]. Prax Pneumol 1974;28:270–2. PMID:4839951
357. LeBel M, Masson E, Guilbert E, et al. Effects of rifabutin and rifampicin on the pharmacokinetics of ethinylestradiol and norethindrone. J Clin Pharmacol 1998;38:1042–50. PMID:9824786 https://doi.org/10.1177/009127009803801109
358. Meyer B, Müller F, Wessels P, Maree J. A model to detect interactions between roxithromycin and oral contraceptives. Clin Pharmacol Ther 1990;47:671–4. PMID:2113449 https://doi.org/10.1038/clpt.1990.92
359. Nocke-Finke L, Breuer H, Reimers D. [Effects of rifampicin on the menstrual cycle and on oestrogen excretion in patients taking oral contraceptives]. Dtsch Med Wochenschr 1973;98:1521–3. PMID:4580141 https://doi.org/10.1055/s-0028-1107071
360. Piguet B, Muglioni JF, Chaline G. [Letter: Oral contraception and rifampicin]. Nouv Presse Med 1975;04:115–6. PMID:1138226
361. Reimers D, Jezek A. [The simultaneous use of rifampicin and other antitubercular agents with oral contraceptives]. Prax Pneumol 1971;25:255–62. PMID:5556355
362. Skolnick JL, Stoler BS, Katz DB, Anderson WH. Rifampin, oral contraceptives, and pregnancy. JAMA 1976;236:1382. PMID:989097 https://doi.org/10.1001/jama.1976.03270130044027
363. Szoka PR, Edgren RA. Drug interactions with oral contraceptives: compilation and analysis of an adverse experience report database. Fertil Steril 1988;49(Suppl 2):31S–8S. PMID:3282933
364. Berry-Bibee EN, Kim MJ, Simmons KB, et al. Drug interactions between hormonal contraceptives and psychotropic drugs: a systematic review. Contraception 2016;94:650–67. PMID:27444984 https://doi.org/10.1016/j.contraception.2016.07.011
365. Berry-Bibee EN, Kim MJ, Tepper NK, Riley HEM, Curtis KM. Co-administration of St. John's wort and hormonal contraceptives: a systematic review. Contraception 2016;94:668–77. PMID:27444983 https://doi.org/10.1016/j.contraception.2016.07.010

Appendix E: Classifications for Barrier Methods

Classifications for barrier contraceptive methods include those for condoms, which include external (male) condoms (latex or synthetic) and internal (female) condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide (Box E1) (Table E1).

Patients should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for sexually transmitted infections (STIs), including HIV infection (*1*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*1*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*2*).

BOX E1. Categories for classifying barrier methods

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE E1. Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
Personal Characteristics and Reproductive History				
Pregnancy	NA	NA	NA	**Clarification:** None of these methods are relevant for contraception during known pregnancy. However, for persons who remain at risk for STIs or HIV infection during pregnancy, the correct and consistent use of condoms is recommended.
Age				
a. Menarche to <40 years	1	1	1	—
b. ≥40 years	1	1	1	—
Parity				
a. Nulliparous	1	1	1	—
b. Parous	1	1	2	**Clarification:** Risk for cervical cap failure is higher in parous persons than in nulliparous persons.
Postpartum (breastfeeding and nonbreastfeeding)				
a. <6 weeks postpartum	1	1	NA	**Clarification:** Diaphragm and cap are unsuitable until uterine involution is complete.
b. ≥6 weeks postpartum	1	1	1	—
Postabortion (spontaneous or induced)				
a. First trimester abortion	1	1	1	—
b. Second trimester abortion	1	1	1	**Clarification:** Diaphragm and cap are unsuitable until 6 weeks after second trimester abortion.
c. Immediate postseptic abortion	1	1	1	—
Past ectopic pregnancy	1	1	1	—
History of pelvic surgery	1	1	1	—
Smoking				
a. Age <35 years	1	1	1	—
b. Age ≥35 years				
i. <15 cigarettes per day	1	1	1	—
ii. ≥15 cigarettes per day	1	1	1	—
Obesity				
a. BMI ≥30 kg/m^2	1	1	1	—
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	1	—
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	—
Surgery				
a. Minor surgery without immobilization	1	1	1	—
b. Major surgery				
i. Without prolonged immobilization	1	1	1	—
ii. With prolonged immobilization	1	1	1	—
Cardiovascular Disease				
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	1	1	—
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Adequately controlled hypertension	1	1	1	—
b. Elevated blood pressure levels (properly taken measurements)				
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	1	1	—
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1	1	1	—
c. Vascular disease	1	1	1	—

See table footnotes on page 104.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	—
Deep venous thrombosis/Pulmonary embolism This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Current or history of DVT/PE, receiving anticoagulant therapy (therapeutic dose) (e.g., acute DVT/PE or long-term therapeutic dose)	1	1	1	—
b. History of DVT/PE, receiving anticoagulant therapy (prophylactic dose)				
i. Higher risk for recurrent DVT/PE (one or more risk factors)	1	1	1	—
• Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome)				
• Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer				
• History of recurrent DVT/PE				
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	1	1	—
c. History of DVT/PE, not receiving anticoagulant therapy				
i. Higher risk for recurrent DVT/PE (one or more risk factors)	1	1	1	—
• History of estrogen-associated DVT/PE				
• Pregnancy-associated DVT/PE				
• Idiopathic DVT/PE				
• Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome)				
• Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer				
• History of recurrent DVT/PE				
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	1	1	—
d. Family history (first-degree relatives)	1	1	1	—
Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	**Clarification:** Routine screening in the general population before contraceptive initiation is not recommended.
Superficial venous disorders				
a. Varicose veins	1	1	1	—
b. Superficial venous thrombosis (acute or history)	1	1	1	—
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	—
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	—
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Uncomplicated	1	1	1	—
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1	2	—

See table footnotes on page 104.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: no limitation of activities or slight, mild limitation of activity) (*3*)				
i. <6 months	1	1	1	—
ii. ≥6 months	1	1	1	—
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: marked limitation of activity or should be at complete rest) (*3*)	1	1	1	—
Renal Disease				
Chronic kidney disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Current nephrotic syndrome	1	1	1	—
b. Hemodialysis	1	1	1	—
c. Peritoneal dialysis	1	1	1	—
Rheumatic Diseases				
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Positive (or unknown) antiphospholipid antibodies	1	1	1	—
b. Severe thrombocytopenia	1	1	1	—
c. Immunosuppressive therapy	1	1	1	—
d. None of the above	1	1	1	—
Rheumatoid arthritis				
a. Not receiving immunosuppressive therapy	1	1	1	—
b. Receiving immunosuppressive therapy	1	1	1	—
Neurologic Conditions				
Headaches				
a. Nonmigraine (mild or severe)	1	1	1	—
b. Migraine				
i. Without aura (includes menstrual migraine)	1	1	1	**Comment:** Menstrual migraine is a subtype of migraine without aura. For more information see the International Headache Society's *International Classification of Headache Disorders, 3rd ed.* (https://ichd-3.org) (*4*).
ii. With aura	1	1	1	—
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	—
Multiple sclerosis				
a. Without prolonged immobility	1	1	1	—
b. With prolonged immobility	1	1	1	—
Depressive Disorders				
Depressive disorders	1	1	1	—
Reproductive Tract Infections and Disorders				
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	1	1	1	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation.
Endometriosis	1	1	1	—
Benign ovarian tumors (including cysts)	1	1	1	—
Severe dysmenorrhea	1	1	1	—

See table footnotes on page 104.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Suspected gestational trophoblastic disease (immediate postevacuation)				
i. Uterine size first trimester	1	1	1	—
ii. Uterine size second trimester	1	1	1	—
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)				
i. Undetectable or nonpregnant β–hCG levels	1	1	1	—
ii. Decreasing β–hCG levels	1	1	1	—
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	1	1	1	—
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	1	1	1	—
Cervical ectropion	1	1	1	—
Cervical intraepithelial neoplasia	1	1	1	**Clarification:** The cap should not be used. Diaphragm use has no restrictions.
Cervical cancer (awaiting treatment)	1	Vaginal pH modulator: 1 Spermicide: 2	1	**Clarification:** The cap should not be used. Diaphragm use has no restrictions. **Comment:** Repeated and high-dose use of the spermicide nonoxynol-9 can cause vaginal and cervical irritation or abrasions.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Undiagnosed mass	1	1	1	—
b. Benign breast disease	1	1	1	—
c. Family history of cancer	1	1	1	—
d. Breast cancer				
i. Current	1	1	1	—
ii. Past and no evidence of current disease for 5 years	1	1	1	—
Endometrial hyperplasia	1	1	1	—
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	—
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	1	1	—
Uterine fibroids	1	1	1	—
Anatomical abnormalities	1	1	NA	**Clarification:** The diaphragm cannot be used in certain cases of prolapse. Cap use is not appropriate for a person with markedly distorted cervical anatomy.
Pelvic inflammatory disease				
a. Current PID	1	1	1	—
b. Past PID				
i. With subsequent pregnancy	1	1	1	—
ii. Without subsequent pregnancy	1	1	1	—
Sexually transmitted infections				
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	1	1	1	—
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	—
c. Other factors related to STIs	1	1	1	—

See table footnotes on page 104.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
HIV				
High risk for HIV infection	1	Vaginal pH modulator: 1 Spermicide: 4	4	**Evidence:** Repeated and high-dose use of the spermicide nonoxynol-9 was associated with increased risk for genital lesions, which might increase the risk for HIV infection (*5*). **Comment:** Diaphragm and cap use is assigned category 4 because of concerns about the spermicide, not the diaphragm or cap.
HIV infection For persons with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	Vaginal pH modulator: 1 Spermicide: 3	3	**Comment:** Use of spermicides, including with diaphragms and caps, can disrupt the cervical mucosa, which might increase viral shedding and HIV transmission to noninfected sex partners.
Other Infections				
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Uncomplicated	1	1	1	—
b. Fibrosis of the liver	1	1	1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Nonpelvic	1	1	1	—
b. Pelvic	1	1	1	—
Malaria	1	1	1	—
History of toxic shock syndrome	1	1	3	**Comment:** Toxic shock syndrome has been reported in association with contraceptive sponge and diaphragm use.
Urinary tract infection	1	Vaginal pH modulator: 2 Spermicide: 1	2	**Comment:** Use of diaphragms and spermicides might increase risk for urinary tract infection.
Endocrine Conditions				
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. History of gestational disease	1	1	1	—
b. Nonvascular disease				
i. Non-insulin dependent	1	1	1	—
ii. Insulin dependent	1	1	1	—
c. Nephropathy, retinopathy, or neuropathy	1	1	1	—
d. Other vascular disease or diabetes of >20 years' duration	1	1	1	—
Thyroid disorders				
a. Simple goiter	1	1	1	—
b. Hyperthyroid	1	1	1	—
c. Hypothyroid	1	1	1	—
Gastrointestinal Conditions				
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	1	1	—
Gallbladder disease				
a. Asymptomatic	1	1	1	—
b. Symptomatic				
i. Current	1	1	1	—
ii. Treated by cholecystectomy	1	1	1	—
iii. Medically treated	1	1	1	—
History of cholestasis				
a. Pregnancy related	1	1	1	—
b. Past COC related	1	1	1	—
Viral hepatitis				
a. Acute or flare	1	1	1	—
b. Chronic	1	1	1	—

See table footnotes on page 104.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
Cirrhosis				
Decompensated cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Compensated (normal liver function)	1	1	1	—
b. Decompensated (impaired liver function)	1	1	1	—
Liver tumors				
Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. Benign				
i. Focal nodular hyperplasia	1	1	1	—
ii. Hepatocellular adenoma	1	1	1	—
b. Malignant (hepatocellular carcinoma)	1	1	1	—
Respiratory Conditions				
Cystic fibrosis	1	1	1	—
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
Hematologic Conditions				
Thalassemia	1	1	1	—
Sickle cell disease	1	1	1	—
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
Iron deficiency anemia	1	1	1	—
Solid Organ Transplantation				
Solid organ transplantation				
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).				
a. No graft failure	1	1	1	—
b. Graft failure	1	1	1	—

See table footnotes on page 104.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicide and vaginal pH modulator, and diaphragm with spermicide and cervical cap with spermicide

Condition	Category			Clarification/Evidence/Comment
	Condom	Spermicide/Vaginal pH modulator	Diaphragm/Cap (with spermicide)	
Drug Interactions				
Antiretrovirals used for prevention (PrEP) or treatment of HIV infection				**Clarification:** No drug interaction between ARV therapy and barrier method use is known. HIV infection is classified as category 1 for vaginal pH modulator and category 3 for spermicide and diaphragm and cap use (see recommendations for HIV infection). High risk for HIV infection is classified as category 1 for vaginal pH modulator and category 4 for spermicide and diaphragm or cap (see recommendations for High risk for HIV infection).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)				
i. Abacavir (ABC)	1	1/3/4	3/4	
ii. Tenofovir (TDF)	1	1/3/4	3/4	
iii. Zidovudine (AZT)	1	1/3/4	3/4	
iv. Lamivudine (3TC)	1	1/3/4	3/4	
v. Didanosine (DDI)	1	1/3/4	3/4	
vi. Emtricitabine (FTC)	1	1/3/4	3/4	
vii. Stavudine (D4T)	1	1/3/4	3/4	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)				
i. Efavirenz (EFV)	1	1/3/4	3/4	
ii. Etravirine (ETR)	1	1/3/4	3/4	
iii. Nevirapine (NVP)	1	1/3/4	3/4	
iv. Rilpivirine (RPV)	1	1/3/4	3/4	
c. Ritonavir-boosted protease inhibitors				
i. Ritonavir-boosted atazanavir (ATV/r)	1	1/3/4	3/4	
ii. Ritonavir-boosted darunavir (DRV/r)	1	1/3/4	3/4	
iii. Ritonavir-boosted fosamprenavir (FPV/r)	1	1/3/4	3/4	
iv. Ritonavir-boosted lopinavir (LPV/r)	1	1/3/4	3/4	
v. Ritonavir-boosted saquinavir (SQV/r)	1	1/3/4	3/4	
vi. Ritonavir-boosted tipranavir (TPV/r)	1	1/3/4	3/4	
d. Protease inhibitors without ritonavir				
i. Atazanavir (ATV)	1	1/3/4	3/4	
ii. Fosamprenavir (FPV)	1	1/3/4	3/4	
iii. Indinavir (IDV)	1	1/3/4	3/4	
iv. Nelfinavir (NFV)	1	1/3/4	3/4	
e. CCR5 co-receptor antagonists				
i. Maraviroc (MVC)	1	1/3/4	3/4	
f. HIV integrase strand transfer inhibitors				
i. Raltegravir (RAL)	1	1/3/4	3/4	
ii. Dolutegravir (DTG)	1	1/3/4	3/4	
iii. Elvitegravir (EVG)	1	1/3/4	3/4	
g. Fusion inhibitors				
i. Enfuvirtide	1	1/3/4	3/4	
Anticonvulsant therapy				
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, or oxcarbazepine)	1	1	1	—
b. Lamotrigine	1	1	1	—
Antimicrobial therapy				
a. Broad-spectrum antibiotics	1	1	1	—
b. Antifungals	1	1	1	—
c. Antiparasitics	1	1	1	—
d. Rifampin or rifabutin therapy	1	1	1	—
Psychotropic medications				
a. Selective serotonin reuptake inhibitors (SSRIs)	1	1	1	—
St. John's wort	1	1	1	—
Allergy to latex	3	1	3	**Clarification:** The condition of allergy to latex does not apply to plastic condoms or diaphragms.

Abbreviations: ARV = antiretroviral; BMI = body mass index; COC = combined oral contraceptive; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; LDL = low-density lipoprotein; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; PrEP = pre-exposure prophylaxis; STI = sexually transmitted infection.

References

1. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
2. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
3. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
4. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders. 3rd ed. Cephalalgia 2018;38:1–211. https://www.ichd-3.org/wp-content/uploads/2018/01/The-International-Classification-of-Headache-Disorders-3rd-Edition-2018.pdf
5. Wilkinson D, Ramjee G, Tholandi M, Rutherford G. Nonoxynol-9 for preventing vaginal acquisition of HIV infection by women from men. Cochrane Database Syst Rev 2002;2002:CD003936. PMID:12519622 https://doi.org/10.1002/14651858.CD003936

Appendix F: Classifications for Fertility Awareness–Based Methods

Fertility awareness–based (FAB) methods involve identifying the fertile days of the menstrual cycle, whether by observing fertility signs, such as cervical secretions and basal body temperature or by monitoring cycle days, and might include use of Food and Drug Administration–cleared contraceptive software applications (Box F1) (Table F1). FAB methods can be used in combination with abstinence or barrier methods during the fertile time. If barrier methods are used, see Classifications for Barrier Methods (Appendix E).

No medical conditions worsen because of FAB methods. In general, FAB methods can be used without concern for health effects in persons who choose them. However, multiple conditions make their use more complex. The existence of these conditions suggests that use of these methods should be delayed until the condition is corrected or resolved, or persons using FAB methods need special counseling; and a provider with particular training in use of these methods is generally necessary to ensure correct use.

FAB methods do not protect against sexually transmitted infections (STIs), including HIV infection, and patients using FAB methods should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*1*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*1*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*2*).

BOX F1. Definitions for terms associated with fertility awareness–based methods

- **Symptoms-based methods:** FAB methods based on observation of fertility signs (e.g., cervical secretions or basal body temperature) such as the cervical mucus method, the symptothermal method, and the TwoDay method.
- **Calendar-based methods:** FAB methods based on calendar calculations such as the calendar rhythm method and the standard days method.
- **Accept:** No medical reason exists to deny the particular FAB method to a patient in this circumstance.
- **Caution:** The method normally is provided in a routine setting but with extra preparation and precautions. For FAB methods, this usually means that special counseling might be needed to ensure correct use of the method by a patient in this circumstance.
- **Delay:** Use of this method should be delayed until the condition is evaluated or corrected. Alternative temporary methods of contraception should be offered.

Abbreviation: FAB = fertility awareness–based.

TABLE F1. Fertility awareness–based methods, including symptoms-based and calendar-based methods

Condition	Category: Symptoms-based method	Category: Calendar-based method	Clarification/Evidence/Comment
Personal Characteristics and Reproductive History			
Pregnancy	NA	NA	**Clarification:** FAB methods are not relevant during pregnancy.
Life stage			**Comment:** Menstrual irregularities are common in postmenarche and perimenopause and might complicate the use of FAB methods.
a. Postmenarche	Caution	Caution	
b. Perimenopause	Caution	Caution	
Breastfeeding			**Comment:** Use of FAB methods when breastfeeding might be less effective than when not breastfeeding.
a. <6 weeks postpartum	Delay	Delay	**Comment:** Persons who are primarily breastfeeding and are amenorrheic are unlikely to have sufficient ovarian function to produce detectable fertility signs and hormonal changes during the first 6 months postpartum. However, the likelihood of resumption of fertility increases with time postpartum and with substitution of breast milk by other foods.
b. ≥6 weeks postpartum	Caution	Delay	
c. After menses begin	Caution	Caution	**Clarification:** Once fertility signs are noted, particularly cervical secretions, then symptoms-based methods can be used. First postpartum menstrual cycles while breastfeeding vary significantly in length. Return to regularity takes several cycles. When there have been at least three postpartum menses and cycles are regular again, a calendar-based method can be used. When there have been at least four postpartum menses and the most recent cycle lasted 26–32 days, the standard days method can be used. Before that time, a barrier method should be offered if the patient plans to use a FAB method later.
Postpartum (nonbreastfeeding)			
a. <4 weeks	Delay	Delay	**Clarification:** Nonbreastfeeding persons are not likely to have detectable fertility signs or hormonal changes before 4 weeks postpartum. Although the risk for pregnancy is low, ovulation before first menses is common; therefore, a method appropriate for the postpartum period should be offered.
b. ≥4 weeks	Accept	Delay	**Clarification:** Nonbreastfeeding persons are likely to have sufficient ovarian function to produce detectable fertility signs, hormonal changes, or both at this time; likelihood increases rapidly with time postpartum. Calendar-based methods can be used as soon as three postpartum menses have been completed. Methods appropriate for the postpartum period should be offered before that time.
Postabortion (spontaneous or induced)	Caution	Delay	**Clarification:** After abortion, it is possible for ovarian function to produce detectable fertility signs, hormonal changes, or both; likelihood increases with time postabortion. Calendar-based methods can be used following at least one postabortion menses (e.g., persons who before this pregnancy primarily had cycles of 26–32 days can then use the standard days method). Methods appropriate for the postabortion period should be offered before that time.
Reproductive Tract Infections and Disorders			
Irregular vaginal bleeding	Delay	Delay	**Clarification:** Presence of this condition makes FAB methods unreliable. Therefore, barrier methods should be recommended until the bleeding pattern is compatible with proper method use. The condition should be evaluated and treated as necessary.
Vaginal discharge	Delay	Accept	**Clarification:** Because vaginal discharge makes recognition of cervical secretions difficult, the condition should be evaluated and treated if needed before providing methods based on cervical secretions.
Other			
Use of drugs that affect cycle regularity, hormones, or fertility signs	Caution/ Delay	Caution/ Delay	**Clarification:** Use of certain mood-altering drugs (e.g., lithium, tricyclic antidepressants, and antianxiety therapies), as well as certain antibiotics and anti-inflammatory drugs, might alter cycle regularity or affect fertility signs. The condition should be carefully evaluated and a barrier method offered until the degree of effect has been determined or the drug is no longer being used.
Diseases that elevate body temperature			
a. Chronic diseases	Caution	Accept	**Clarification:** Elevated temperatures might make basal body temperature difficult to interpret but have no effect on cervical secretions. Thus, use of a method that relies on temperature should be delayed until the acute febrile disease abates. Temperature-based methods are not appropriate for persons with chronically elevated temperatures. In addition, certain chronic diseases interfere with cycle regularity, making calendar-based methods difficult to interpret.
b. Acute diseases	Delay	Accept	

Abbreviations: FAB = fertility awareness–based; NA = not applicable.

References

1. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1

2. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf

Appendix G: Lactational Amenorrhea Method

The Bellagio Consensus provided the scientific basis for defining the conditions under which breastfeeding can be used safely and effectively for birth-spacing purposes; programmatic guidelines were developed at a meeting of family planning experts for its use as a method of contraception, and the method was then named the lactational amenorrhea method (LAM) (*1*–*3*). These guidelines include the following three criteria, all of which must be met to ensure adequate protection from pregnancy: 1) amenorrhea, 2) fully or nearly fully breastfeeding (intervals between feedings not exceeding 4 hours during the day or 6 hours at night), and 3) <6 months postpartum (*4*–*6*).

The U.S. Dietary Guidelines for Americans recommend that infants be exclusively breastfed for about the first 6 months, with continued breastfeeding while introducing appropriate complementary foods for 1 year or longer (*7*). The American Academy of Pediatrics (AAP) recommends exclusive breastfeeding for about the first 6 months, with continued breastfeeding along with introducing appropriate complementary foods for up to age 2 years or longer (*8*).

No medical conditions exist for which use of LAM for contraception is restricted. However, breastfeeding might not be recommended for persons or infants with certain conditions

LAM does not protect against sexually transmitted infections (STIs), including HIV infection, and patients using LAM should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*9*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*9*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*10*).

HIV Infection. HIV transmission can occur during breastfeeding. For breastfeeding persons on antiretroviral therapy with a sustained undetectable HIV viral load during pregnancy, the risk for transmission through breastfeeding is <1%, but not zero. Patients with HIV infection should receive evidence-based, person-centered counseling to support shared decision-making about infant feeding. For comprehensive information, refer to Infant Feeding for Individuals with HIV in the United States (https://clinicalinfo.hiv.gov/en/guidelines/perinatal/counseling-and-managing-individuals-with-hiv-united-states-who-desire-breastfeed). These recommendations are included within the U.S. Department of Health and Human Services's Recommendations for the Use of Antiretroviral Drugs During Pregnancy and Interventions to Reduce Perinatal HIV Transmission in the United States (https://clinicalinfo.hiv.gov/en/guidelines/perinatal/whats-new-guidelines) (*11*).

Other Medical Conditions. CDC and AAP also recommend against both breastfeeding and feeding expressed milk for persons with untreated brucellosis, positivity for human T-cell lymphotropic virus types I or II, herpes simplex lesions on a breast, Ebola virus disease, or mpox. In addition, infants with classic galactosemia should not breastfeed (*8*,*12*,*13*) (https://www.cdc.gov/breastfeeding-special-circumstances/hcp/contraindications/index.html).

Medication Used During Breastfeeding. Although many medications do pass into breast milk, most have little or no effect on milk supply or on infant well-being. Few medications are contraindicated while breastfeeding. More information about specific medications and radioactive compounds is provided by AAP (*14*), LactMed (https://www.ncbi.nlm.nih.gov/books/NBK501922), Mother to Baby (http://www.mothertobaby.org), and InfantRisk Center (https://www.infantrisk.com/category/breastfeeding).

References

1. Kennedy KI, Rivera R, McNeilly AS. Consensus statement on the use of breastfeeding as a family planning method. Contraception 1989;39:477–96. PMID:2656086 https://doi.org/10.1016/0010-7824(89)90103-0
2. Labbok M, Cooney K, Coly S. Guidelines: breastfeeding, family planning, and the lactational amenorrhea method—LAM. Washington, DC: Institute for Reproductive Health, 1994.
3. Labbok MH, Perez A, Valdes V, et al. The lactational amenorrhea method (LAM): a postpartum introductory family planning method with policy and program implications. Adv Contracept 1994;10:93–109. PMID:7942265 https://doi.org/10.1007/BF01978103
4. Hight-Laukaran V, Labbok MH, Peterson AE, Fletcher V, von Hertzen H, Van Look PF. Multicenter study of the lactational amenorrhea method (LAM): II. Acceptability, utility, and policy implications. Contraception 1997;55:337–46. PMID:9262928 https://doi.org/10.1016/S0010-7824(97)00041-3
5. Labbok MH, Hight-Laukaran V, Peterson AE, Fletcher V, von Hertzen H, Van Look PF. Multicenter study of the lactational amenorrhea method (LAM): I. Efficacy, duration, and implications for clinical application. Contraception 1997;55:327–36. PMID:9262927 https://doi.org/10.1016/S0010-7824(97)00040-1
6. Peterson AE, Peŕez-Escamilla R, Labbok MH, Hight V, von Hertzen H, Van Look P. Multicenter study of the lactational amenorrhea method (LAM) III: effectiveness, duration, and satisfaction with reduced client-provider contact. Contraception 2000;62:221–30. PMID:11172792 https://doi.org/10.1016/S0010-7824(00)00171-2

7. US Department of Agriculture; US Department of Health and Human Services. Dietary guidelines for Americans, 2020–2025. 9th ed. Washington, DC: US Department of Agriculture and US Department of Health and Human Services; 2020. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf
8. Meek JY, Noble L; Section on Breastfeeding. Policy statement: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057988. PMID:35921640 https://doi.org/10.1542/peds.2022-057988
9. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
10. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
11. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Recommendations for the use of antiretroviral drugs during pregnancy and interventions to reduce perinatal HIV transmission in the United States. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/en/guidelines/perinatal/recommendations-arv-drugs-pregnancy-overview
12. Meek JY, Noble L. Technical report: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057989. PMID:35921641 https://doi.org/10.1542/peds.2022-057989
13. CDC. Contraindications to breastfeeding or feeding expressed breast milk to infants; 2023. https://www.cdc.gov/breastfeeding-special-circumstances/hcp/contraindications/index.html
14. Sachs HC, Frattarelli DAC, Galinkin JL, et al.; Committee on Drugs. The transfer of drugs and therapeutics into human breast milk: an update on selected topics. Pediatrics 2013;132:e796–809. PMID:23979084 https://doi.org/10.1542/peds.2013-1985

Appendix H: Coitus Interruptus (Withdrawal)

Coitus interruptus, also known as withdrawal, is a contraceptive method in which the penis is completely removed from the vagina and away from the external genitalia before ejaculation. Coitus interruptus prevents sperm from entering the vagina, thereby preventing contact between spermatozoa and the ovum.

Coitus interruptus has no directly associated health risks. Coitus interruptus does not protect against sexually transmitted infections (STIs), including HIV infection, and patients using coitus interruptus should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*1*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*1*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*2*).

References

1. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
2. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf

Appendix I: Permanent Contraception

Tubal surgery (including laparoscopic and abdominal approaches) and vasectomy are methods of permanent contraception available in the United States. In general, no medical conditions absolutely restrict a person's eligibility for permanent contraception (with the exception of known allergy or hypersensitivity to any materials used to complete the permanent contraception procedure). However, certain conditions might increase a person's surgical risk during tubal surgery; in these cases, careful consideration can be given to the risks and benefits of other acceptable long-acting or permanent alternatives, including intrauterine device, implant, and vasectomy.

Patients should be appropriately counseled that permanent contraception is intended to be irreversible and about the availability of highly effective, long-acting reversible methods of contraception. Most persons who choose permanent contraception remain satisfied with their decision. However, a small proportion of women regret this decision (1%–26% from different studies, with higher rates of regret reported by women who were younger at time of permanent contraception procedure) (*1*,*2*). Regret among men about vasectomy has been reported to be approximately 5% (*3*), similar to the proportion of women who report regretting their husbands' vasectomy (6%) (*4*).

Permanent contraception does not protect against sexually transmitted infections (STIs), including HIV infection, and patients using permanent contraception should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*5*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*5*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*6*).

References

1. Hillis SD, Marchbanks PA, Tylor LR, Peterson HB. Poststerilization regret: findings from the United States Collaborative Review of Sterilization. Obstet Gynecol 1999;93:889–95. PMID:10362150 https://doi.org/10.1097/00006250-199906000-00001
2. Peterson HB. Sterilization. Obstet Gynecol 2008;111:189–203. PMID:18165410 https://doi.org/10.1097/01.AOG.0000298621.98372.62
3. Ehn BE, Liljestrand J. A long-term follow-up of 108 vasectomized men. Good counselling routines are important. Scand J Urol Nephrol 1995;29:477–81. PMID:8719366 https://doi.org/10.3109/00365599509180030
4. Jamieson DJ, Kaufman SC, Costello C, Hillis SD, Marchbanks PA, Peterson HB; US Collaborative Review of Sterilization Working Group. A comparison of women's regret after vasectomy versus tubal sterilization. Obstet Gynecol 2002;99:1073–9. PMID:12052602 https://doi.org/10.1016/S0029-7844(02)01981-6
5. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
6. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf

Appendix J: Classifications for Emergency Contraception

Classifications are given for the copper intrauterine device (Cu-IUD) as emergency contraception. The Cu-IUD can be placed within 5 days of the first act of unprotected intercourse as emergency contraception. In addition, when the day of ovulation can be estimated, the Cu-IUD can be placed beyond 5 days after sexual intercourse, as long as the placement does not occur >5 days after ovulation. The eligibility criteria for interval Cu-IUD placement also apply for the placement of Cu-IUDs as emergency contraception (Box J1) (Table J1) (*1*).

Classifications for emergency contraceptive pills (ECPs) are given for ulipristal acetate (UPA), levonorgestrel (LNG), and combined oral contraceptives (COCs). ECPs should be taken as soon as possible within 5 days of unprotected sexual intercourse (*1*).

Cu-IUDs, UPA, LNG, and COCs do not protect against sexually transmitted infections (STIs), including HIV infection, and patients using these methods should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*2*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*2*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*3*).

BOX J1. Categories for classifying emergency contraception

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE J1. Classifications for emergency contraception, including the copper intrauterine device, ulipristal acetate, levonorgestrel, and combined oral contraceptives

Condition	Category				Clarification/Evidence/Comment
	Cu-IUD	UPA	LNG	COC	
Personal Characteristics and Reproductive History					
Pregnancy	4	NA	NA	NA	**Clarification (IUD):** The IUD is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion. **Clarification (ECPs):** Although this method is not indicated for a patient with a known or suspected pregnancy, no harm to the patient, the course of pregnancy, or the fetus if ECPs are inadvertently used is known to exist. **Evidence:** Evidence suggests that poor pregnancy outcomes are rare among pregnant women who used ECPs during conception cycle or early in pregnancy (*4*).
Breastfeeding	1	1	1	1	**Evidence:** Breastfeeding outcomes do not seem to differ between women exposed to LNG and those who are not exposed (*4*). One pharmacokinetic study demonstrated that LNG passes to breast milk but in minimal quantities (*4*). UPA and its active metabolite, monodemethyl-ulipristal acetate, are present in human milk in small amounts; no evidence is available on effects of UPA emergency contraception exposure on infants or children who are breastfed (*5*).
Past ectopic pregnancy	1	1	1	1	—
Obesity (BMI ≥30 kg/m^2)	1	2	2	2	**Clarification (ECPs):** ECPs might be less effective among persons with BMI ≥30 kg/m^2 than among persons with BMI <25 kg/m^2. Despite this, no safety concerns exist. **Evidence:** Limited evidence from secondary data analyses suggests that women with BMI ≥30 kg/m^2 experience an increased risk for pregnancy after use of LNG compared with women with BMI <25 kg/m^2. Two analyses suggest that women with obesity might also experience an increased risk for pregnancy after use of UPA compared with those without obesity, although this increase was not significant in one study (*6*).
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	1	**Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications such as long-term diarrhea, vomiting, or both. Because of these malabsorptive concerns, an emergency IUD might be more appropriate than ECPs.
Cardiovascular Disease					
History of severe cardiovascular disease (ischemic heart disease, cerebrovascular attack, or other thromboembolic conditions) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	2	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Rheumatic Diseases					
Rheumatoid arthritis					
a. Not receiving immunosuppressive therapy	1	1	1	1	—
b. Receiving immunosuppressive therapy	2	1	1	1	—
Neurologic Conditions					
Migraine	1	1	1	2	**Comment:** The duration of ECP use is less than that of regular use of COCs and thus would be expected to have less clinical impact.

See table footnotes on the next page.

TABLE J1. (*Continued*) Classifications for emergency contraception, including the copper intrauterine device, ulipristal acetate, levonorgestrel, and combined oral contraceptives

Condition	Category				Clarification/Evidence/Comment
	Cu-IUD	UPA	LNG	COC	
Gastrointestinal Conditions					
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	1	1	1	—
Severe liver disease (including jaundice) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	2	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Solid Organ Transplantation					
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).					
a. No graft failure	1	1	1	1	—
b. Graft failure	2	1	1	1	—
Other					
Repeated ECP use	—	1	1	1	**Clarification (ECPs):** Frequently repeated ECP use might be harmful for persons with conditions classified as category 2, 3, or 4 for CHC or POC use. **Evidence:** In one case-control study, risk for ectopic pregnancy compared with intrauterine pregnancy did not increase after repeated use of LNG ECPs compared with nonuse (*4*).
Sexual assault	2	1	1	1	**Clarification (IUD):** Persons who have experienced sexual assault are at increased risk for STIs, including HIV infection. According to CDC STI treatment guidelines, routine presumptive treatment of chlamydia, gonorrhea, and trichomonas is recommended after sexual assault (*2*). Persons with current purulent cervicitis, chlamydial infection, or gonococcal infection should not undergo IUD placement (category 4).
CYP3A4 inducers (e.g., bosentan, carbamazepine, felbamate, griseofulvin, oxcarbazepine, phenytoin, rifampin, St. John's wort, topiramate, efavirenz, and lumacaftor)	1	2	2	2	**Clarification (ECPs):** Strong CYP3A4 inducers might reduce the effectiveness of ECPs. **Evidence:** According to labelling information, rifampin markedly decreases UPA levels by ≥90%, which might decrease its efficacy (*5*). Therefore, theoretical concerns extend to use of other CYP3A4 inducers as well as to COC and LNG ECPs, which have metabolic pathways similar to those of UPA. A small pharmacokinetic study found that concomitant efavirenz use decreased LNG levels in women taking LNG ECPs (1.5 mg) by 56% compared with LNG ECPs alone (*7*).

Abbreviations: BMI = body mass index; CHC = combined hormonal contraceptive; COC = combined hormonal contraceptive; Cu-IUD = copper intrauterine device; CYP = cytochrome P450; ECP = emergency contraceptive pill; IUD = intrauterine device; LNG = levonorgestrel; NA = not applicable; POC = progestin-only contraceptive; POP = progestin-only pill; STI = sexually transmitted infection; UPA = ulipristal acetate.

References

1. Curtis KM, Nguyen AT, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2024. MMWR Recomm Rep 2024;73(No. RR-3):1–77.
2. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
3. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
4. Jatlaoui TC, Riley H, Curtis KM. Safety data for levonorgestrel, ulipristal acetate and Yuzpe regimens for emergency contraception. Contraception 2016;93:93–112. PMID:26546020 https://doi.org/10.1016/j.contraception.2015.11.001
5. HRA Pharma America. Ella [Package insert]. Morristown, NJ: HRA Pharma America; 2021.
6. Jatlaoui TC, Curtis KM. Safety and effectiveness data for emergency contraceptive pills among women with obesity: a systematic review. Contraception 2016;94:605–11. PMID:27234874 https://doi.org/10.1016/j.contraception.2016.05.002
7. Carten ML, Kiser JJ, Kwara A, Mawhinney S, Cu-Uvin S. Pharmacokinetic interactions between the hormonal emergency contraception, levonorgestrel (Plan B), and efavirenz. Infect Dis Obstet Gynecol 2012;2012:137192. PMID:22536010 https://doi.org/10.1155/2012/137192

Appendix K: Summary of Classifications for Hormonal Contraceptive Methods and Intrauterine Devices

Health care providers can use the summary table as a quick reference guide to the classifications for hormonal contraceptive methods and intrauterine contraception to compare classifications across these methods (Box K1) (Table K1). See the respective appendix for each method for clarifications to the numeric categories, as well as for summaries of the evidence and additional comments. Hormonal contraceptives and intrauterine devices do not protect against sexually transmitted infections (STIs), including HIV infection, and patients using these methods should be counseled that consistent and correct use of external (male) latex condoms reduces the risk for STIs, including HIV infection (*1*). Use of internal (female) condoms can provide protection from transmission of STIs, although data are limited (*1*). Patients also should be counseled that pre-exposure prophylaxis, when taken as prescribed, is highly effective for preventing HIV infection (*2*).

BOX K1. Categories for classifying hormonal contraceptives and intrauterine devices

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used

Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

TABLE K1. Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC
Personal Characteristics and Reproductive History						
Pregnancy	4*	4*	NA*	NA*	NA*	NA*
Age	Menarche to <20 years: 2 ≥20 years: 1	Menarche to <20 years: 2 ≥20 years: 1	Menarche to <18 years: 1 18–45 years: 1 >45 years: 1	Menarche to <18 years: 2 18–45 years: 1 >45 years: 2	Menarche to <18 years: 1 18–45 years: 1 >45 years: 1	Menarche to <40 years: 1 ≥40 years: 2
Parity						
a. Nulliparous	2	2	1	1	1	1
b. Parous	1	1	1	1	1	1
Breastfeeding						
a. <21 days postpartum	—	—	2*	2*	2*	4*
b. 21 to <30 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	2*	2*	2*	3*
ii. Without other risk factors for VTE	—	—	2*	2*	2*	3*
c. 30–42 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1*	2*	1*	3*
ii. Without other risk factors for VTE	—	—	1*	1*	1*	2*
d. >42 days postpartum	—	—	1*	1*	1*	2*
Postpartum (nonbreastfeeding)						
a. <21 days postpartum	—	—	1	2	1	4
b. 21–42 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1	2	1	3*
ii. Without other risk factors for VTE	—	—	1	1	1	2
c. >42 days postpartum	—	—	1	1	1	1
Postpartum (including cesarean delivery, breastfeeding, or nonbreastfeeding)						
a. <10 minutes after delivery of the placenta	2*	2*	—	—	—	—
b. 10 minutes after delivery of the placenta to <4 weeks	2*	2*	—	—	—	—
c. ≥4 weeks	1*	1*	—	—	—	—
d. Postpartum sepsis	4	4	—	—	—	—
Postabortion (spontaneous or induced)						
a. First trimester abortion						
i. Procedural (surgical)	1*	1*	1*	1*	1*	1*
ii. Medication	1*	1*	1*	1/2*	1*	1*
iii. Spontaneous abortion with no intervention	1*	1*	1*	1*	1*	1*

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC
b. Second trimester abortion						
i. Procedural (surgical)	2*	2*	1*	1*	1*	1*
ii. Medication	2*	2*	1*	1*	1*	1*
iii. Spontaneous abortion with no intervention	2*	2*	1*	1*	1*	1*
c. Immediate postseptic abortion	4	4	1*	1*	1*	1*
Past ectopic pregnancy	1	1	1	1	2	1
History of pelvic surgery (see recommendations for Postpartum [including cesarean delivery])	1	1	1	1	1	1
Smoking						
a. Age <35 years	1	1	1	1	1	2
b. Age ≥35 years						
i. <15 cigarettes per day	1	1	1	1	1	3
ii. ≥15 cigarettes per day	1	1	1	1	1	4
Obesity						
a. BMI ≥30 kg/m^2	1	1	1	1	1	2*
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	1	2	1	2*
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).						
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	1	1	1
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	1	3	COCs: 3 Patch and ring: 1
Surgery						
a. Minor surgery without immobilization	1	1	1	1	1	1
b. Major surgery						
i. Without prolonged immobilization	1	1	1	1	1	2
ii. With prolonged immobilization	1	1	1	2	1	4
Cardiovascular Disease						
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	2	2*	3*	2*	3/4*
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 3).						
a. Adequately controlled hypertension	1*	1*	1*	2*	1*	3*
b. Elevated blood pressure levels (properly taken measurements)						
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1*	1*	1*	2*	1*	3*
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1*	2*	2*	3*	2*	4*
c. Vascular disease	1*	2*	2*	3*	2*	4*

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implant	DMPA	POP	CHC
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	1	1	2
Deep venous thrombosis/ Pulmonary embolism This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).						
a. Current or history of DVT/PE, receiving anticoagulant therapy (therapeutic dose) (e.g., acute DVT/PE or long-term therapeutic dose)	2*	2*	2*	2*	2*	3*
b. History of DVT/PE, receiving anticoagulant therapy (prophylactic dose)						
i. Higher risk for recurrent DVT/PE (one or more risk factors)	2*	2*	2*	3*	2*	4*
• Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome)						
• Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer						
• History of recurrent DVT/PE						
ii. Lower risk for recurrent DVT/PE (no risk factors)	2*	2*	2*	2*	2*	3*
c. History of DVT/PE, not receiving anticoagulant therapy						
i. Higher risk for recurrent DVT/PE (one or more risk factors	1	2	2	3	2	4
• History of estrogen-associated DVT/PE						
• Pregnancy-associated DVT/PE						
• Idiopathic DVT/PE						
• Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome)						
• Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer						
• History of recurrent DVT/PE						
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	2	2	2	2	3
d. Family history (first-degree relatives)	1	1	1	1	1	2
Thrombophilia (e.g., factor V Leiden mutation; prothrombin gene mutation; protein S, protein C, and antithrombin deficiencies; or antiphospholipid syndrome) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1*	2*	2*	3*	2*	4*
Superficial venous disorders						
a. Varicose veins	1	1	1	1	1	1
b. Superficial venous thrombosis (acute or history)	1	1	1	2	1	3*

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD		Implant		DMPA	POP		CHC
		Initiation	Continuation	Initiation	Continuation		Initiation	Continuation	
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2	3	2	3	3	2	3	4
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1	2		Initiation 2	Continuation 3	3	Initiation 2	Continuation 3	4
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Uncomplicated	1	1		1		1	1		2
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1		1		2	1		4
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: no limitation of activities or slight, mild limitation of activity) (*3*)									
i. <6 months	2	2		1		2	1		4
ii. ≥6 months	2	2		1		2	1		3
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: marked limitation of activity or should be at complete rest) (*3*)	2	2		2		3	2		4
Renal Disease									
Chronic kidney disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation	Initiation	Continuation			—		
a. Current nephrotic syndrome	1	1	2	2	2	3	2* DRSP POP with known hyperkalemia: 4*		4
b. Hemodialysis	1	1	2	2	2	3	2* DRSP POP with known hyperkalemia: 4*		4
c. Peritoneal dialysis	2	1	2	2	2	3	2* DRSP POP with known hyperkalemia: 4*		4
Rheumatic Diseases									
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	Initiation	Continuation		—		Initiation	Continuation	—	
a. Positive (or unknown) antiphospholipid antibodies	1*	1*	2*	2*		3*	3*	2*	4*
b. Severe thrombocytopenia	3*	2*	2*	2*		3*	2*	2*	2*
c. Immunosuppressive therapy	2*	1*	2*	2*		2*	2*	2*	2*
d. None of the above	1*	1*	2*	2*		2*	2*	2*	2*

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC
Rheumatoid arthritis	Initiation	Continuation	Initiation	Continuation			—	
a. Not receiving immunosuppressive therapy	1	1	1	1	1	2	1	2
b. Receiving immunosuppressive therapy	2	1	2	1	1	2/3*	1	2
Neurologic Conditions								
Headaches								
a. Nonmigraine (mild or severe)	1		1		1	1	1	1*
b. Migraine								
i. Without aura (includes menstrual migraine)	1		1		1	1	1	2*
ii. With aura	1		1		1	1	1	4*
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	1		1		1*	1*	1*	1*
Multiple sclerosis								
a. Without prolonged immobility	1		1		1	2	1	1
b. With prolonged immobility	1		1		1	2	1	3
Depressive Disorders								
Depressive disorders	1*		1*		1*	1*	1*	1*
Reproductive Tract Infections and Disorders								
Vaginal bleeding patterns			Initiation	Continuation			—	
a. Irregular pattern without heavy bleeding	1		1	1	2	2	2	1
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2*		1*	2*	2*	2*	2*	1*
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	Initiation	Continuation	Initiation	Continuation			—	
	4*	2*	4*	2*	3*	3*	2*	2*
Endometriosis	2		1		1	1	1	1
Benign ovarian tumors (including cysts)	1		1		1	1	1	1
Severe dysmenorrhea	2		1		1	1	1	1
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
a. Suspected gestational trophoblastic disease (immediate postevacuation)								
i. Uterine size first trimester	1*		1*		1*	1*	1*	1*
ii. Uterine size second trimester	2*		2*		1*	1*	1*	1*
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)	Initiation	Continuation	Initiation	Continuation			—	
i. Undetectable or nonpregnant β-hCG levels	1*	1*	1*	1*	1*	1*	1*	1*
ii. Decreasing β-hCG levels	2*	1*	2*	1*	1*	1*	1*	1*
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	2*	1*	2*	1*	1*	1*	1*	1*
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	4*	2*	4*	2*	1*	1*	1*	1*
Cervical ectropion	1		1		1	1	1	1
Cervical intraepithelial neoplasia	1		2		2	2	1	2
Cervical cancer (awaiting treatment)	Initiation	Continuation	Initiation	Continuation			—	
	4	2	4	2	2	2	1	2

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC
Breast disease								
Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
a. Undiagnosed mass	1		2*		2*	2*	2*	2*
b. Benign breast disease	1		1		1	1	1	1
c. Family history of cancer	1		1		1	1	1	1
d. Breast cancer								
i. Current	1		4		4	4	4	4
ii. Past and no evidence of current disease for 5 years	1		3		3	3	3	3
Endometrial hyperplasia	1		1		1	1	1	1
Endometrial cancer	Initiation	Continuation	Initiation	Continuation			—	
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).	4	2	4	2	1	1	1	1
Ovarian cancer	1		1		1	1	1	1
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
Uterine fibroids	2		2		1	1	1	1
Anatomical abnormalities								
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD placement)	4		4				—	
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD placement	2		2					
Pelvic inflammatory disease	Initiation	Continuation	Initiation	Continuation			—	
a. Current PID	4	2*	4	2*	1	1	1	1
b. Past PID								
i. With subsequent pregnancy	1	1	1	1	1	1	1	1
ii. Without subsequent pregnancy	2	2	2	2	1	1	1	1
Sexually transmitted infections	Initiation	Continuation	Initiation	Continuation			—	
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2*	4	2*	1	1	1	1
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	1	1	1	1
c. Other factors related to STIs	2*	2	2*	2	1	1	1	1
HIV								
	Initiation	Continuation	Initiation	Continuation			—	
High risk for HIV infection	1*	1*	1*	1*	1	1	1	1
HIV infection	—	—	—	—	1*	1*	1*	1*
For persons with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
a. Clinically well receiving ARV therapy	1	1	1	1	—	—	—	—
b. Not clinically well or not receiving ARV therapy	2	1	2	1	—	—	—	—

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC	
Other Infections									
Schistosomiasis									
Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Uncomplicated	1		1		1	1	1	1	
b. Fibrosis of the liver (if severe, see recommendations for Cirrhosis)	1		1		1	1	1	1	
Tuberculosis	Initiation	Continuation	Initiation	Continuation			—		
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Nonpelvic	1	1	1	1	1*	1*	1*	1*	
b. Pelvic	4	3	4	3	1*	1*	1*	1*	
Malaria	1		1		1	1	1	1	
Endocrine Conditions									
Diabetes									
Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. History of gestational disease	1		1		1	1	1	1	
b. Nonvascular disease									
i. Non-insulin dependent	1		2		2	2	2	2	
ii. Insulin dependent	1		2		2	2	2	2	
c. Nephropathy, retinopathy, or neuropathy	1		2		2	3	2	3/4*	
d. Other vascular disease or diabetes of >20 years' duration	1		2		2	3	2	3/4*	
Thyroid disorders									
a. Simple goiter	1		1		1	1	1	1	
b. Hyperthyroid	1		1		1	1	1	1	
c. Hypothyroid	1		1		1	1	1	1	
Gastrointestinal Conditions									
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1		1		1	2	2	2/3*	
Gallbladder disease									
a. Asymptomatic	1		2		2	2	2	2	
b. Symptomatic									
i. Current	1		2		2	2	2	3	
ii. Treated by cholecystectomy	1		2		2	2	2	2	
iii. Medically treated	1		2		2	2	2	3	
History of cholestasis									
a. Pregnancy related	1		1		1	1	1	2	
b. Past COC related	1		2		2	2	2	3	
Viral hepatitis								Initiation	Continuation
a. Acute or flare	1		1		1	1	1	3/4*	2
b. Chronic	1		1		1	1	1	1	1
Cirrhosis									
Decompensated cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 3).									
a. Compensated (normal liver function)	1		1		1	1	1	1	
b. Decompensated (impaired liver function)	1		2		2	3	2	4	

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC
Liver tumors								
Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
a. Benign								
i. Focal nodular hyperplasia	1		2		2	2	2	2
ii. Hepatocellular adenoma	1		2		2	3	2	4
b. Malignant (hepatocellular carcinoma)	1		3		3	3	3	4
Respiratory Conditions								
Cystic fibrosis	1*		1*		1*	2*	1*	1*
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
Hematologic Conditions								
Thalassemia	2		1		1	1	1	1
Sickle cell disease	2		1		1	2/3*	1	4
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
Iron-deficiency anemia	2		1		1	1	1	1
Solid Organ Transplantation								
Solid organ transplantation	Initiation	Continuation	Initiation	Continuation		—		
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 3).								
a. No graft failure	1	1	1	1	2	2/3*	2	2*
b. Graft failure	2	1	2	1	2	2/3*	2	4
Drug Interactions								
Antiretrovirals used for prevention (PrEP) or treatment of HIV infection								
See the following guidelines for the most up-to-date recommendations on drug-drug interactions between hormonal contraception and antiretrovirals: 1) Recommendations for the Use of Antiretroviral Drugs During Pregnancy and Interventions to Reduce Perinatal HIV Transmission in the United (https://clinicalinfo.hiv.gov/en/guidelines/perinatal/prepregnancy-counseling-childbearing-age-overview?view=full#table-3) (*4*) and 2) Guidelines for the Use of Antiretroviral Agents in Adults and Adolescents with HIV (https://clinicalinfo.hiv.gov/en/guidelines/hiv-clinical-guidelines-adult-and-adolescent-arv/drug-interactions-overview?view=full) (*5*).								
a. Nucleoside reverse transcriptase inhibitors (NRTIs)	Initiation	Continuation	Initiation	Continuation		—		
i. Abacavir (ABC)	1/2*	1*	1/2*	1*	1	1	1	1
ii. Tenofovir (TDF)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Zidovudine (AZT)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Lamivudine (3TC)	1/2*	1*	1/2*	1*	1	1	1	1
v. Didanosine (DDI)	1/2*	1*	1/2*	1*	1	1	1	1
vi. Emtricitabine (FTC)	1/2*	1*	1/2*	1*	1	1	1	1
vii. Stavudine (D4T)	1/2*	1*	1/2*	1*	1	1	1	1
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)								
i. Efavirenz (EFV)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
ii. Etravirine (ETR)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Nevirapine (NVP)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Rilpivirine (RPV)	1/2*	1*	1/2*	1*	1	1	1	1
c. Ritonavir-boosted protease inhibitors								
i. Ritonavir-boosted atazanavir (ATV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
ii. Ritonavir-boosted darunavir (DRV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
iii. Ritonavir-boosted fosamprenavir (FPV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
iv. Ritonavir-boosted lopinavir (LPV/r)	1/2*	1*	1/2*	1*	1	1	1	1
v. Ritonavir-boosted saquinavir (SQV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
vi. Ritonavir-boosted tipranavir (TPV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*

See table footnotes on page 126.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC
d. Protease inhibitors without ritonavir								
i. Atazanavir (ATV)	1/2*	1*	1/2*	1*	1	1	1	2*
ii. Fosamprenavir (FPV)	1/2*	1*	1/2*	1*	2*	2*	2*	3*
iii. Indinavir (IDV)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Nelfinavir (NFV)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
e. CCR5 co-receptor antagonists								
i. Maraviroc (MVC)	1/2*	1*	1/2*	1*	1	1	1	1
f. HIV integrase strand transfer inhibitors								
i. Raltegravir (RAL)	1/2*	1*	1/2*	1*	1	1	1	1
ii. Dolutegravir (DTG)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Elvitegravir (EVG)	1/2*	1*	1/2*	1*	1	1	1	1
g. Fusion inhibitors								
i. Enfuvirtide	1/2*	1*	1/2*	1*	1	1	1	1
Anticonvulsant therapy								
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	1		1		2*	1*	3*	3*
b. Lamotrigine	1		1		1	1	1	3*
Antimicrobial therapy								
a. Broad-spectrum antibiotics	1		1		1	1	1	1
b. Antifungals	1		1		1	1	1	1
c. Antiparasitics	1		1		1	1	1	1
d. Rifampin or rifabutin therapy	1		1		2*	1*	3*	3*
Psychotropic medications								
a. Selective serotonin reuptake inhibitors (SSRIs)	1		1		1	1	1	1
St. John's wort	1		1		2	1	2	2

Abbreviations: ARV = antiretroviral; BMI = body mass index; CHC = combined hormonal contraceptive; COC = combined oral contraceptive; Cu-IUD = copper intrauterine device; DMPA = depot medroxyprogesterone acetate; DRSP = drospirenone; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; IUD = intrauterine device; LDL = low-density lipoprotein; LNG-IUD = levonorgestrel intrauterine device; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; POP = progestin-only pill; PrEP = pre-exposure prophylaxis; STI = sexually transmitted infection; VTE = venous thromboembolism.
* Consult the appendix for this contraceptive method for a clarification to this classification.

References

1. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. MMWR Recomm Rep 2021;70(No. RR-4):1–187. PMID:34292926 https://doi.org/10.15585/mmwr.rr7004a1
2. CDC. US Public Health Service preexposure prophylaxis for the prevention of HIV infection in the United States—2021 update: a clinical practice guideline. Atlanta, GA: US Department of Health and Human Services, CDC; 2021. https://www.cdc.gov/hiv/pdf/risk/prep/cdc-hiv-prep-guidelines-2021.pdf
3. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
4. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Recommendations for the use of antiretroviral drugs during pregnancy and interventions to reduce perinatal HIV transmission in the United States. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/en/guidelines/perinatal/recommendations-arv-drugs-pregnancy-overview
5. Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in adults and adolescents with HIV. Washington, DC: US Department of Health and Human Services; 2023. https://clinicalinfo.hiv.gov/sites/default/files/guidelines/documents/adult-adolescent-arv/guidelines-adult-adolescent-arv.pdf

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ISSN: 1057-5987 (Print)