

method is often recommended, especially when treatment includes prolonged estrogen deprivation. Of the nonhormonal methods, the copper IUD is the most effective option for preventing pregnancy. For hormone receptor-negative breast cancers, methods that minimize hormone exposure are often recommended by oncologists, although there is no evidence to suggest increased risks of adverse outcomes with hormonal contraception. It is unclear if the use of the LNG 52 mg IUD impacts long-term breast cancer recurrence. The LNG 52 mg IUD significantly reduces the risk of endometrial polyps. For individuals taking tamoxifen, which increases the risk of endometrial polyps, this can be an important benefit to consider in selecting a contraceptive method [12]. However, there is no evidence that the LNG 52 mg IUD would decrease endometrial cancer risks in premenopausal tamoxifen users.

2.1.2. Does the use of hormonal contraception increase the risk of new-onset breast cancer for those at increased risk for familial or hereditary breast and ovarian cancer?

For individuals at significantly increased risk for familial or hereditary breast and ovarian cancer (HBOC), we recommend clinicians provide access to all available contraceptive methods utilizing a person-centered approach (GRADE 1B).

Validated models and genetic testing now allow widespread identification of those at significantly increased risk for familial or hereditary breast and ovarian cancer (HBOC). Individuals at high risk for breast cancer without a personal history can be safely offered hormonal contraception regardless of genetic risk [5,13,14]. Combined hormonal contraceptives (CHC) have been associated with significant reductions in both ovarian and endometrial cancer in those who carry a pathogenic variant in *BRCA1* or *BRCA2*, and longer duration of use is associated with greater protection [5]. Less data for ovarian cancer prevention is available for newer, lower dose or progestin-only formulations. Meta-analyses and systematic reviews have shown either minimal or no increase in breast cancer risk in individuals with genetic risk for breast or ovarian cancer using formulations of 35 µg ethinyl estradiol or less [5,15]. Shared decision-making is key when working with individuals at high risk for breast cancer. The CDC MEC places no restrictions on hormonal contraceptive use by those who are high-risk, without current or recent personal history of breast cancer [13]. In those who carry genetic variants increasing both breast and ovarian cancer risk, the balance of the small potential for increased breast cancer risk and considerably decreased ovarian cancer risk should be discussed.

2.2. Ovarian cancer

2.2.1. Does the use of hormonal or permanent contraception impact outcomes in those who have completed ovarian cancer treatment or are at very high risk of ovarian cancer?

For individuals with a history of or active ovarian cancer, we recommend clinicians provide access to all available contraceptive methods utilizing a person-centered approach (GRADE 1B). For individuals at high risk for ovarian cancer, we recommend clinicians offer hormonal contraception with the goal of ovarian suppression for ovarian cancer prevention (GRADE 1B). For individuals diagnosed with hormonally-sensitive ovarian malignancies, such as adult granulosa cell tumors, low-grade serous, and endometrioid adenocarcinomas, who are considering hormonal contraception, we suggest shared decision-making with the individual and their oncologist (GRADE 2C). Estrogen-containing contraceptives should be avoided by individuals treated with estrogen-blocking therapy (Best Practice).

Data from both the general population and individuals who carry germline pathogenic *BRCA1* and *BRCA2* genes can help inform contraceptive decisions among individuals with a history of ovarian

cancer, as data specific to individuals with a personal history of ovarian cancer are not available.

2.2.1.1. Combined hormonal contraceptives (CHCs). Estrogen and progestin-containing CHCs have consistently been shown to halve the risk of ovarian cancer diagnosis, both in the general population as well as in those who carry germline pathogenic variants in *BRCA1* and *BRCA2* genes [5,14,16]. Increased duration of hormonal contraception use leads to more protective benefits, regardless of the formulation [17].

2.2.1.2. Depot medroxyprogesterone acetate (DMPA). Studies of non-oral hormonal contraception formulations' effect on ovarian cancer are more limited. A systematic review of DMPA injection users showed a reduction in ovarian cancer diagnosis (OR, 0.65; 95% CI, 0.50–0.85) [18].

2.2.1.3. Progestin-only pills. Low-dose progestin-only pills, which less reliably suppress ovulation, have not consistently been shown to lower the risk of ovarian cancer [17]. Given that different progestin-only methods have variable effects on ovulation suppression, more studies are needed to understand the relationship between ovulation and ovarian cancer prevention.

2.2.1.4. Intrauterine devices (IUDs). Hormonal IUDs may have a role in ovarian cancer prevention; a large prospective cohort study reported a 50% risk reduction in ovarian cancer with use of a hormonal IUD, a level of risk reduction similar to the use of oral contraceptives, though meta-analyses have mixed findings [19–21].

2.2.1.5. Permanent contraception. Laparoscopic sterilization using tubal occlusion techniques such as electrosurgical desiccation, a silicone band, or a titanium clip and partial or complete salpingectomy (removal of bilateral fallopian tubes) have been associated with lower rates of ovarian cancer [22,23]. Complete salpingectomy has the potential for greater ovarian cancer risk reduction and should be considered when laparoscopic sterilization is planned, and ovarian cancer risk reduction is desired [24].

Given the neutral or protective benefits of hormonal contraception, most individuals with a history of epithelial or borderline ovarian cancer, with retained ovaries, can safely use any hormonal contraceptive [13,25]. Less common ovarian cancer subtypes may be estrogen sensitive, such as adult granulosa cell tumors, low-grade serous, or endometrioid adenocarcinomas [26]. Prior use of oral contraception in those diagnosed with granulosa cell tumors has been associated with improved survival rates [27]. Clinicians should engage in shared decision-making with the individual and their oncology team when those with hormonally-sensitive ovarian cancer subtypes are considering hormonal contraception. Active ovarian cancer increases the risk of thrombosis, which should also be considered.

2.3. Uterine cancer

2.3.1. Does the use of hormonal contraception or intrauterine devices impact the effectiveness of uterine cancer treatment or increase the risk of uterine cancer recurrence or morbidity?

2.3.1.1. Endometrial cancer. For individuals with a history of endometrial cancer, we recommend clinicians provide access to all available contraceptive methods utilizing a person-centered approach (GRADE 1B). For individuals with active endometrial cancer requesting an IUD, we suggest shared decision-making with the individual and their oncologist (GRADE 1B).

When uterine preservation is planned in the setting of endometrial carcinoma, hormonal therapies may offer effective contraception. The