

Fertility Recovery After Long-Term Hormonal Contraception: A Narrative Review of Method-Specific Timelines, Biological Mechanisms, and Clinical Counseling

Mariam A Alkhatib¹, Ahed J Alkhatib^{2*} and Nawal A Alkhatib³

¹*Faculty of Nursing, Jordan University of Science and Technology, Jordan*

²*Department of Legal Medicine, Toxicology and Forensic Medicine, Jordan University of Science and Technology, Jordan*

³*Faculty of Pharmacy, Jordan University of Science and Technology, Jordan*

***Corresponding Author:** Ahed J Alkhatib Department of Legal Medicine, Toxicology and Forensic Medicine, Jordan University of Science and Technology, Jordan.

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Abstract

Background: Hormonal contraceptives are considered reversible. Yet, some women report delayed conception after discontinuation. Temporary endocrine readjustment, the pharmacokinetics of the method, reproductive aging, and previously masked disorders might be confused with contraceptive-induced infertility, which is a clinical concern.

Objective: Evidence is synthesized to describe the restoration of fertility after long-term hormonal contraception, emphasizing differences among methods, possible biological mechanisms, susceptible subgroups, and counseling implications.

Methods: In order to synthesize systematic reviews, prospective cohort studies, physiological studies, and pharmacogenomic studies related to return of ovulation, time to pregnancy, ovarian reserve markers, endometrial receptivity, and hormonal clearance, we used a narrative review framework.

Results: The majority of women become pregnant within one year of stopping reversible contraception, and there is currently no evidence to suggest that oral contraceptives, implants, or intrauterine contraception cause permanent infertility. Of all the contraceptives given, depot medroxyprogesterone acetate has the most consistent delay because the suppressive effect persists after the last injection. Using combined oral contraceptives can temporarily change the cycle biomarkers and suppress the anti-Müllerian hormone and antral follicle count, however these changes recover on stopping them. There is little evidence remaining endometrial, cervical, neuroendocrine or genetic effects. Conception is mostly delayed due to multiple factors. It may be a reflection of age, polycystic ovary syndrome, endometriosis, thyroid or metabolic disease, tubal disease, male-factor infertility, and lifestyle factors.

Conclusion: Most users can easily reverse long-term hormone contraception. The clinically relevant question is not whether contraception generally causes infertility, but why in some cases recovery takes longer, and then how the type of method, baseline reproductive health, and individual susceptibility interact. Studies that will prospectively link contraceptive exposure with hormonal recovery, ovulation, endometrial function and time to pregnancy are warranted.

Keywords: Hormonal Contraception; Return to Fertility; Time to Pregnancy; Ovarian Reserve; Depot Medroxyprogesterone Acetate; Endometrial Receptivity; Pharmacogenomics

Introduction

Hormonal contraception has revolutionized reproductive health by not only ensuring reliable prevention of pregnancy but also better control over the timing of childbearing. Due to the prolonged use of hormonal methods by many women, it is not uncommon to be questioned about the speed and completeness of fertility recovery after stopping the use of these methods. Most users of reversible contraception will not develop permanent infertility according to available evidence. As a systematic review showed, about 83% of women conceived within one year of stopping contraception [1]. Yet, this conclusion at the population level does not mean that recovery is instant and the same for all women.

The process of restoration of fertility is multifaceted. The process involves the elimination of external hormones, reactivation of the hypothalamic-pituitary-ovarian axis, recruitment of follicles, ovulation, appropriate luteal function, re-establishment of sperm-supportive cervical mucus, endometrial maturation, transport through the tubes, and successful implantation of the embryo. If any component is delayed, there may be delay in time to pregnancy despite menstruation resuming. Additionally, used contraceptive method may mask a pre-existing condition of menstrual irregularity, polycystic ovary syndrome, endometriosis, or any other condition which becomes apparent after cessation of the treatment [2,3].

Thus, the principal issue is causal interpretation. Women might discontinue contraception at an old age after which naturally their ovarian reserve and oocyte quality is reduced. On the other hand, a different method like depot medroxyprogesterone acetate may inhibit ovulation for months even after the faded last taking. In other instances, delayed conception may not be due to prior contraception and may represent male-factor infertility, tubal disease, thyroid disease, obesity, insulin resistance, stress, or lifestyle exposure.

This paper creatively synthesizes existing literature in journal formats and focuses on answering four key questions: How quickly does fertility return after different contraceptive methods? What biological processes may account for delay? Who should be assessed sooner? What should reproductive counseling include?

Review Approach

This paper represents a narrative review emanating from a systematic compilation of literature on long-term hormonal contraception and recovery of fertility. Based on evidence 8 sections were defined for the assessment of individualised controlled ovarian stimulation (ICOS). Through method specific recovery, Neuroendocrine and ovulatory recovery, Ovarian and follicular markers and Endometrial and cervical mechanisms. Through Metabolic and pharmacogenomic variability. Unmasked reproductive pathology and Confounding factors. Clinical counselling 8 sections were critically assessed. The studies which were given priority were systematic reviews, prospective studies, physiological studies and other clinically related reviews. In view of the differences in definitions of exposure, duration of use, intention with respect to pregnancy, and measurement of the outcome in the literature, the findings are interpreted qualitatively instead of pooled anew (Figure 1).

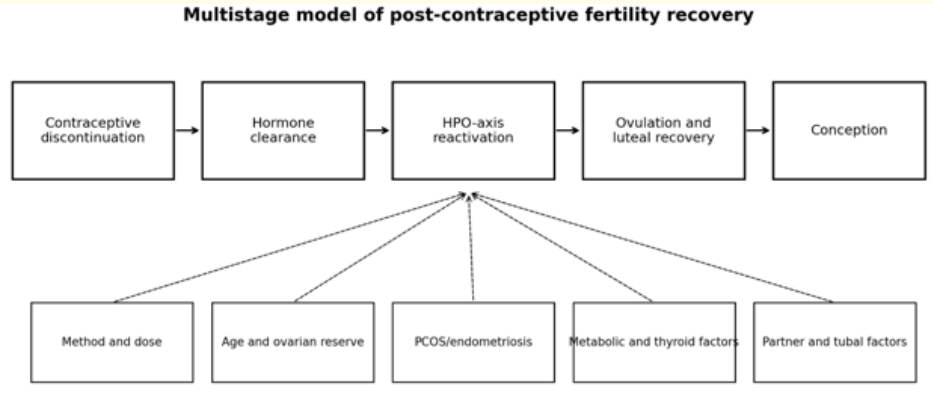


Figure 1: Multistage conceptual model of post-contraceptive fertility recovery and major modifying factors.

Note: HPO: Hypothalamic-Pituitary-Ovarian. The diagram is a conceptual synthesis of the reviewed evidence rather than a validated prediction model.

General evidence for reversibility

Available evidence suggests that previous use of reversible contraception does not significantly impair overall long-term fecundability. Research, from Girum and Wasie [1], synthesizing 22 studies involving 14884 women which estimated of 83.1% becoming pregnant within 12 months of cessation. Rates of pregnancy were relatively similar among former users of hormonal methods and intra-uterine devices, and the duration of use of oral contraceptives was not associated with clinically important long-term infertility [1].

This evidence shows that temporary delay should not lead us to suspect permanent infertility. Pooled pregnancy rates, though, may camouflage short early delays, method-specific differences, and susceptible subgroups. The duration of pregnancy also relates to frequency of intercourse, intention of pregnancy, fertility of partner, age of partner, which are not measured evenly across studies.

Therefore, it should combine reassurance and precision. Fertility will return in most users, although the time interval may be instant or take several months. For injectable progestins, we can clearly say that the recovery is rapid while for other contraceptives like, oral contraceptives, implants and intrauterine devices, we usually see a relatively shorter delay.

Method-specific patterns of fertility recovery

Combined oral contraceptives, patch, and vaginal ring

After stopping the combined oral contraceptive pill, ovulation usually resumes after a few weeks but the first few cycles may not be a woman's untreated baseline. According to Nassaralla, *et al.* [4], menstrual-cycle biomarkers were altered for at least 2 cycles after OPD. Research has shown that menstrual cycle length and quality disturbance reversibility takes place after discontinuation [5]. Research indicates that endocrine recalibration, not ovarian injury, occurs.

The length of use of a prior pill has not consistently predicted sustained infertility. Any perceived delays may be due to the outcome of a pre-existing irregular cycle pattern, masked by scheduled withdrawal bleeding. It is expected that patch and vaginal-ring users will have broadly similar endocrine recovery patterns, although comparative data are more limited.

Implants and intrauterine contraception

After being removed, contraceptive implants and both hormonal and copper intrauterine devices usually return fertility quickly. One should not confuse their long duration of action with effect of post-removal prolonged suppression. If the device is removed, the circulating exposure falls and pregnancy might occur in the first cycle. Long-acting reversible contraception should not be portrayed as more detrimental to future fertility than shorter-acting methods.

Injectable progestins

There is a consistent association between depot medroxyprogesterone acetate (DMPA) and delayed return of ovulation and conception. The depot formulation continues to release the active drug after the scheduled time of contraception. According to reviews [6], return to fertility is on average about 10 months after the last injection, although there is considerable variability. This delay is due to persistent pharmacologic suppression, not permanent infertility. We do not currently have access to this document.

Using DMPA for a longer period of time does not mean that delay will have a simple dose-response effect, but rather recovery is determined by the absorption at the injection site, composition of the body, metabolism and time elapsed since last dose. Women that will become pregnant in the near future should be given method-specific counselling before choosing or continuing injectable contraception [7-9] (Table 1 and figure 2).

Proposed mechanism	Supporting observations	Current interpretation	Evidence strength
Persistent depot progestin exposure	Delayed ovulation after DMPA; median ovulation recovery measured in months	Established pharmacologic delay, generally reversible	Moderate to strong
Early HPO-axis recalibration	Altered cycle biomarkers during initial cycles after oral contraceptive cessation	Plausible explanation for short-term delay	Moderate
Temporary suppression of AMH/AFC	Markers rise after combined oral contraceptive discontinuation	Functional suppression rather than follicle depletion	Moderate
Delayed endometrial normalization	Altered morphology and receptivity markers during use	Biologically plausible; persistent clinical effect unproven	Limited
Genetic variation in hormone metabolism	Interindividual etonogestrel concentration variability	Hypothesis-generating; no validated prediction of fertility recovery	Limited
Unmasked reproductive disease	PCOS, endometriosis, thyroid or tubal disease emerges after cessation	Common alternative explanation and confounder	Strong clinical plausibility

Table 2: Proposed mechanisms of delayed fertility recovery and strength of current evidence.

Sources: Gnoth., et al. [5], Nassaralla., et al. [4], Landersoe., et al. [12], Lazowitz., et al. [16], and Osoti., et al. [9].

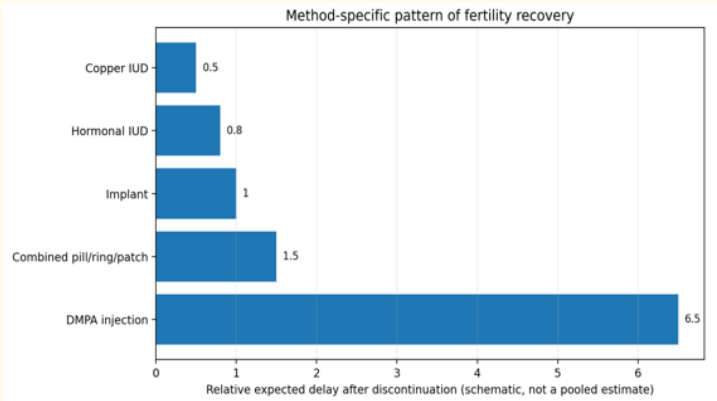


Figure 2: Schematic comparison of method-specific recovery patterns after contraceptive discontinuation.

Note: Values are illustrative relative-delay scores used to visualize the evidence hierarchy; they are not pooled clinical estimates. DMPA shows the clearest prolonged delay [7,9].

Neuroendocrine and ovulatory recovery

Hormonal contraceptives alter the signaling of hormones involved in ovulation to prevent synthesis of oocytes. After stopping, there must be resumption of coordinated follicular growth, production of estradiol, release of luteinizing hormone, ovulation and corpus luteum function. Most women’s bodies adapt to this change quickly, but some have ovulatory or hormone problems early on.

Post-pill amenorrhea has to be treated as a clinical sign requiring assessment rather than assume it to denote contraceptive injury. We need to check for pregnancy, pre-existing oligomenorrhea, polycystic ovary syndrome, thyroid disease, hyperprolactinemia, hypothalamic dysfunction, marked weight change and stress. The clearest genuine pharmacological delay is observed with DMPA, after which the reproductive axis remains suppressed until the decline in medroxyprogesterone exposure. Citing the following resources [7-9].

Menstruation's return is not equal to normal ability to conceive. Prior to the establishment of consistent ovulation or full luteal-phase function normalization, bleeding may occur. This distinction explains why conception can take few cycles after resumption of menses.

Ovarian reserve and follicular recruitment

Using combined hormonal contraception can have a temporary negative impact on anti-Müllerian hormone, antral follicle count, and ovarian volume. According to Bentzen, *et al.* [10], hormonal contraceptive users have lower AMH and AFC than non-users in large cross-sectional studies. These results may be understood as a permanent loss of ovarian reserve if testing is done during active use [10-12].

Support for reversibility in prospective evidence. Landersoe, *et al.* [12] observed AMH increased by 53% and AFC by 41% when long-term combined oral contraception was stopped. It was stabilized in around 2 months. Thus, low AMH or AFC observed during use or just after stopping should be interpreted carefully, especially when the result is crucial for fertility preservation or infertility treatment choices.

AMH and AFC refer to the determination of the number of follicles in a woman's ovary. Neither of them, on their own, indicates if a woman can conceive naturally. It is necessary to keep in mind, while interpreting the findings, the factors like age, menstrual history, ultrasound findings, previous surgery on ovaries, exposure to chemotherapy and condition of polycystic ovary. It's possible to have high AMH and AFC in PCOS without ovulation. The markers of ovarian reserve are not mechanisms to evaluate ovulatory function. This research is based on a few investigations of [10-12].

Endometrial and cervical recovery

Medroxyprogesterone, a contraceptive, keeps the endometrium thin and inactive and alters cervical mucus to inhibit sperm penetration. According to Somkuti, *et al.* [13], the biological implantation caused by endometrial integrins may be influenced by the sperm's oral-conception activities. These observations make biological sense for post-discontinuation time frame with recovering endometrial proliferation as well as secretory transformation and ovulatory-endometrial synchrony.

The proof that routine hormonal contraception inflicts persistent endometrial damage is weak. The pregnancy rates in the population post-cessation indicates against a clinically important long-term implantation defect for most women. The relevant unanswered question is whether a small subgroup such as women with persistently thin endometrium, endometriosis, advanced reproductive age, or prolonged progestin exposure have slower restoration of receptivity [14,15].

Cervical mucus typically resumes a sperm-friendly pattern after progestin withdrawal. It has not been proven that mucus-related infertility is persistent and cervical observations should be considered along with ovulation, semen quality, and tubal patency (Table 2).

Pharmacokinetic, metabolic, and genetic variability

The circulating levels of hormones may be substantially different among women using the same formulation of contraceptive. Differences can occur due to body mass index, adherence, route of administration, drug interactions, hepatic enzyme activity and genetic differences. Numerous synthetic estrogens and progestins are subject to CYP3A-mediated metabolism followed by conjugation, predominantly glucuronidation and sulfation.

Etonogestrel implants differ across individuals according to pharmacogenomic studies. Lazorwitz and colleagues [16] investigated genetic variants influencing etonogestrel levels in a steady state, and subsequent genome-wide work further investigated this variability. The studies demonstrate that inherited factors can affect the pharmacokinetics of contraceptives, but as yet they do not show specific variants that delay ovulation or conception after stopping. Consequently, the likelihood of having a genetic predisposition towards slow recovery after stopping contraceptives. At present, predictive return to fertility is not possible with routine genetic testing. Future research should link genotype with measured hormone clearance, ovulatory recovery, endometrial markers and prospective time-to-pregnancy outcomes.

Unmasked pathology and alternative explanations

Polycystic ovary syndrome and chronic anovulation

The use of combined hormonal contraceptives may create a regular withdrawal bleed and decrease androgen-related symptoms without correcting the underlying tendency toward irregular ovulation. The use of oligomenorrhoea or anovulation may recur after discontinuation of the contraceptive. Therefore, a detailed menstrual history before contraception is essential.

Endometriosis and pelvic disease

Hormonal suppression can reduce endometriosis-associated pain while disease-related adhesions, inflammation, or altered pelvic anatomy continue to influence fertility. Delayed conception after stopping treatment may therefore reflect endometriosis rather than delayed endocrine recovery. Tubal disease, prior pelvic infection, and uterine abnormalities must also be assessed [14,15].

Reproductive aging

The use of contraceptives for long-term often overlaps with postponement of pregnancy. If a less-recessive woman stops using contraception during the middle-to-late phase of her reproductive life, she may not have difficulty getting pregnant. However, she may have a lower fecundity as age-induced deterioration in ovarian reserve and oocyte quality takes place. When contraceptives are stopped, it can result in the wrong attribution of causation.

Partner, metabolic, and lifestyle factors

The time to pregnancy may take longer because of male-factor infertility, timing of intercourse, smoking, exposure to alcohol, obesity, insulin resistance, thyroid disease, significant weight change, and psychological stress. Research models and clinical assessment should include these factors rather than assuming that the previous contraception is the only exposure.

Identifying women who may need earlier evaluation

Most women can be reassured, although earlier or more structured assessment may be appropriate when risk factors are present. If there is an identifiable cause of infertility, the couple should still be investigated. This will help prevent the initiation of other diseases or conditions which may have an unrecognised psychiatric origin. When independent fertility risk is present, evaluation should not be deferred simply because contraception was recently discontinued [7-9] (Figure 3).

Clinical and nursing counseling implications

Counseling should distinguish four concepts: contraceptive efficacy during use, return of ovulation, return of fecundability and ultimate time to pregnancy. These results are similar but not synonymous. Women should know that most reversible methods allow for quick return of fertility while DMPA often causes a longer delay [7-9].

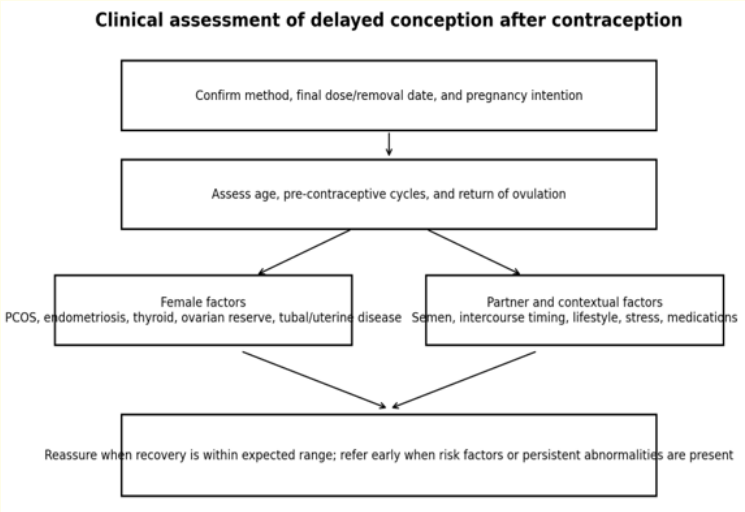


Figure 3: Practical assessment framework for delayed conception following contraceptive discontinuation.

Health professionals involved in reproductive health, above all nurses, can prevent the commission of two errors that repeat themselves: the first is that one hormonal contraception naturally causes permanent infertility, the opposite error is that every delay following the discontinuation of contraception is naturally normal and do not require further evaluation. The pre-contraceptive cycle pattern, method and date of last dose/removal, age, pregnancy desires and known reproductive conditions should be noted in counselling.

Use of hormonal contraceptives when obtaining ovarian reserve tests may underestimate AMH and AFC and this should be explained in the advice. Repeat testing after a washout period, when clinically appropriate, can distinguish reversible suppression from diminished reserve. The counselor should encourage use of folic acid before conception, healthy weight maintenance, avoidance of smoking, review of medications, and conduct timely assessment of partner [10-12].

Counseling domain	Key message	When to escalate
Expected recovery	Most methods are reversible; recovery speed varies by method	Persistent amenorrhea, severe symptoms, or independent fertility risk
Injectable contraception	DMPA may delay ovulation and conception for months after the final injection	Delay substantially beyond the expected interval or additional risk factors
Cycle history	Withdrawal bleeding during use may mask prior irregular ovulation	Cycles remain irregular or absent after discontinuation
Ovarian reserve tests	AMH and AFC may be temporarily suppressed during hormonal use	Repeat or specialist interpretation when results affect treatment decisions
Comprehensive fertility	Assess both partners and consider tubal, uterine, endocrine, and lifestyle factors	No pregnancy after the age-appropriate evaluation interval

Table 3: Suggested counseling framework after hormonal contraceptive discontinuation.

Research gaps and proposed study design

Current studies frequently rely on retrospective contraceptive histories, self-reported discontinuation dates, and pregnancy as the only endpoint. This design makes it difficult to separate hormone clearance, ovulatory recovery, endometrial recovery, intercourse patterns, and underlying infertility.

A stronger prospective design would enroll women before discontinuation and record formulation, dose, route, duration, adherence, age, baseline cycle history, body composition, metabolic status, and reproductive diagnoses. Follow-up could include serial serum or salivary hormone concentrations, ovulation confirmation, AMH and AFC, luteal progesterone, endometrial thickness, cervical mucus assessment, and monthly time-to-pregnancy data. Partner semen analysis and tubal evaluation should be incorporated when conception is delayed [10-12].

Method-specific cohorts should be large enough to evaluate DMPA separately from pills, rings, patches, implants, and intrauterine methods. Genetic and pharmacokinetic analyses are justified only when linked to clinically meaningful outcomes. The primary objective should be to identify reproducible recovery phenotypes rather than to assume a uniform contraceptive effect [7-9] (Table 4).

Domain	Recommended variables	Suggested timing	Primary analytical role
Contraceptive exposure	Method, formulation, dose, route, duration, adherence, final use date	Baseline	Define exposure precisely
Endocrine recovery	FSH, LH, estradiol, progesterone, ovulation testing	Baseline and serial cycles	Characterize HPO-axis recovery
Ovarian activity	AMH, AFC, ovarian volume, follicular development	Baseline, 2 months, 6 months	Differentiate temporary suppression from low reserve
Endometrial function	Thickness, pattern, luteal timing; selected receptivity markers	Periovulatory and luteal phases	Assess implantation environment
Clinical confounders	Age, pre-use cycles, PCOS, endometriosis, thyroid, BMI, smoking, stress	Baseline and follow-up	Control confounding and define subgroups
Couple-level fertility	Intercourse frequency, semen analysis, tubal status	At enrollment or when delay emerges	Avoid female-only causal attribution
Outcome	Time to ovulation, time to pregnancy, live birth	Monthly follow-up	Measure clinically meaningful recovery

Table 4: Priority variables for future prospective studies of post-contraceptive fertility recovery.

Conclusion

Most women’s infertility is absent evidence. Similarly, since hormonal contraception is reversible for most women, its use does not cause an irreversible cause. Real temporary delays depend on the method, with the maximum delay occurring after DMPA. While oral contraceptives may temporarily change the characteristics of the menstrual cycle and suppress markers of ovarian reserve, these effects generally recover. The underlying endometrial, cervical, neuroendocrine, or pharmacogenomic causes of endometrial cancer remain research questions rather than causes we can suspect clinically. So, when conception is delayed after taking a contraceptive pill, there should be a balanced assessment of pharmacology specific to the method, reproductive aging, baseline cycle function, PCOS, Endometriosis, Tubal and male factors, metabolic health and lifestyle. Personalized counselling can assure most women while facilitating timely assessment for those with clinically significant risk [7-9].

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