The Impact of Age on Fertility in Men and Women: A Comprehensive Review of Age-Specific Reproductive Decline and Lifestyle Countermeasures

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1. Introduction

Age is the single most powerful determinant of reproductive potential in both sexes. For women, the biological clock is governed by the finite and progressively depleting pool of oocytes, with quality and quantity declining non-linearly from the early thirties onward. For men, although spermatogenesis continues throughout life, advancing paternal age is associated with declining testosterone, deteriorating sperm parameters, and increasing sperm DNA fragmentation that carries implications for both fertility treatment outcomes and offspring health.[1][2]

Globally, the trend toward delayed parenthood has intensified the intersection between age-related fertility decline and assisted reproductive technology (ART). In many developed nations, the average age of women at first birth has risen past 30, while couples increasingly seek IVF and ICSI in their late thirties and forties. This paper presents an evidence-based analysis of how age impacts fertility across distinct age brackets for both women and men, integrating data from large population-based studies, IVF registries, and systematic reviews. Critically, it examines the lifestyle modifications — dietary, physical, supplementary, and environmental — that can partially counteract or delay age-related reproductive decline.[3]

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2. Female Fertility and Age

2.1 The Ovarian Reserve: A Biological Timeline

Women are born with approximately 6–7 million oocytes at 20 weeks of gestation, a number that declines to 1–2 million at birth and approximately 300,000–500,000 at puberty. By age 37, roughly 25,000 follicles remain, at which point the rate of follicular loss accelerates. Menopause occurs when approximately 1,000 follicles are left, typically around age 49–51.[3]

Anti-Müllerian Hormone (AMH) and Antral Follicle Count (AFC) are the two most reliable markers of this reserve. In a landmark cross-sectional study of 252 Caucasian women aged 25–45, Rosen et al. demonstrated that AMH and AFC are the only serological and ultrasound markers that follow the histologically documented pattern of gradual, accelerated oocyte loss. The average rate of AMH decline per year was −1.09 pmol/L at age 30, accelerating to −3.06 pmol/L at age 40. AFC declined at a rate of −0.57 follicles/year at age 30, increasing to −1.33 follicles/year at age 40.[4]

2.2 Age-Specific AMH and AFC Nomograms

A large-scale Indian study (n = 3,240; 1,338 fertile, 1,902 infertile) generated age-specific centile charts for both fertile and infertile women using the Beckman Coulter Gen II ELISA for AMH and transvaginal ultrasound for AFC. Key findings include:[5]

Age	Fertile Median AMH (ng/mL)	Infertile Median AMH (ng/mL)	Fertile Median AFC	Infertile Median AFC
20	5.70	5.01	17.7	16.2
25	4.94	3.99	16.3	14.1
30	3.76	3.17	14.3	11.9
35	2.64	2.43	10.5	9.8
40	1.87	1.57	6.1	7.7

The decline was non-linear in fertile women and linear in infertile women. Using ROC analysis with the POSEIDON criteria for diminished ovarian reserve (AMH < 1.2 ng/mL, AFC < 5), the age cut-off for clinically significant decline was approximately 31 years in fertile women and 34 years in infertile women. The rate of AMH decline was 0.192 ng/mL per year in the fertile group and 0.172 ng/mL per year in the infertile group.[5]

2.3 IVF Outcomes by Female Age Group

A retrospective analysis of 3,412 stimulated IVF/ICSI cycles at KK Women's and Children's Hospital, Singapore (2008–2010), stratified outcomes across seven age groups:[3]

Age Group	Mean Oocytes Retrieved	Clinical Pregnancy Rate (%)	Live Birth Rate (%)	Miscarriage Rate (%)
< 30	18.5 ± 10.3	50.0	40.0	15.1
30–35	14.9 ± 8.4	39.2	32.2	18.5
36–37	11.8 ± 7.3	28.0	21.4	22.7
38	10.2 ± 6.2	29.2	19.6	30.0
39	9.1 ± 6.3	17.6	8.8	47.7
40–44	7.2 ± 5.3	13.2	5.9	55.3
≥ 45	4.5 ± 2.3	0.0	0.0	—

Age had a statistically significant effect on the number of cycles reaching embryo transfer ($p < 0.001$), clinical pregnancy rates ($p < 0.001$), live birth rates ($p < 0.001$), and miscarriage rates ($p < 0.001$). The data demonstrate that live birth rates decline approximately sevenfold from under 30 to 40–44 years, and miscarriage rates nearly quadruple over the same interval.[3]

2.4 SART CORS Registry Data (2014–2020)

The largest dataset comes from the SART CORS database, analysing 228,171 first autologous ART cycles without PGT across the United States (2014–2020). Key outcomes by age for frozen embryo transfer (FET) versus fresh transfer:[6]

Age Group	N (Total)	FET Live Birth Rate (%)	Fresh Live Birth Rate (%)	aOR for FET vs Fresh
< 35	133,096	Higher	Lower	1.24 (p < 0.001)
35–37	47,433	Higher	Lower	1.24 (p < 0.001)
38–40	30,211	Higher	Lower	1.43 (p < 0.001)
41–42	11,107	Higher	Lower	1.53 (p < 0.001)
> 42	6,324	Higher	Lower	1.98 (p < 0.001)

Overall LBR was 48.3% for FET vs 39.8% for fresh (p < 0.001); cumulative LBR was 74.0% vs 60.0% (p < 0.0001). Critically, the adjusted odds ratio for live birth with FET versus fresh transfer progressively increased with advancing age, suggesting that supraphysiological stimulation during fresh IVF cycles is increasingly detrimental to endometrial receptivity in older women.[6]

For women with diminished ovarian reserve (DOR), the live birth rate was 33.9% with FET vs 26.0% fresh (p < 0.001), and for women over 42 with DOR, the aOR for live birth with FET reached 2.63 (p < 0.0001).[6]

2.5 Age-Related Mechanisms of Decline

The decline in female fertility with age is attributed to multiple mechanisms:

- **Quantitative oocyte depletion:** Progressive atresia of the primordial follicle pool.[4]
- **Oocyte quality deterioration:** Increased rates of meiotic nondisjunction, mitochondrial dysfunction, and chromosomal aneuploidy.[3]
- **Endometrial changes:** Altered receptivity and decidualisation capacity with age.[6]
- **Hormonal shifts:** Rising FSH (reflecting reduced negative feedback from declining follicles), declining AMH and inhibin B, and altered LH dynamics.[4]
- **Accumulation of gynaecological conditions:** Higher prevalence of endometriosis, fibroids, and tubal disease in older women.[3]

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3. Male Fertility and Age

3.1 Hormonal Changes with Paternal Ageing

Advanced paternal age (APA) induces progressive alterations in the hypothalamic-pituitary-testicular axis:

Hormone	Direction of Change with Age	Mechanism
Testosterone	↓	Reduced Leydig cell number and function[2]
Free testosterone	↓ (1.2%/year after 50)	Increased SHBG binding[2]
FSH	↑	Compensatory response to declining spermatogenesis[2]
LH	↑	Compensatory response to reduced testosterone[2]
SHBG	↑	Age-dependent increase reducing bioavailable testosterone[2]

| DHEA | ↓ | Adrenal androgen decline[2] |
| Estrogen | ↓ | Secondary to testosterone decline[2] |

The number of Leydig cells decreases by approximately 50% between the ages of 20–48 and 50–76 years. Testosterone decline contributes to andropausal symptoms including reduced libido, fatigue, and cognitive changes.[2]

3.2 Sperm Parameters and Age

A comprehensive review by Sharma et al. (2015) compiled multiple studies showing that with advancing age:[2]

- **Semen volume:** Decreases by 3%–22% between ages 30 and 50.[2]
- **Sperm motility:** Decreases by 3%–37% between ages 30 and 50; progressive motility begins to decline at age 40.[2]
- **Sperm morphology:** Decreases by 4%–18% between ages 30 and 50; normal morphology declines after age 40.[2]
- **Sperm concentration:** Generally shows no significant decline or may even increase slightly, though this finding remains controversial.[2]

A recent analysis of 6,805 sperm samples from Chinese males aged 20–63 confirmed that semen volume, progressive motility, and total motility significantly decline with advancing paternal age.[7]

3.3 Sperm DNA Fragmentation and Age

DNA fragmentation index (DFI) is one of the most clinically significant age-related changes in male reproductive biology:

Age Group	DFI (%)	Source
< 30	15.2	Moskovtsev et al. (cited in[2])
30–35	19.4	Moskovtsev et al.
35–40	20.1	Moskovtsev et al.
40–45	26.4	Moskovtsev et al.
≥ 45	32.0	Moskovtsev et al.

DFI approximately doubles from age < 30 to ≥ 45. A separate study of 215 couples demonstrated that sperm DNA damage doubled from paternal age 25 to 55 years. In men over 40, the higher incidence of DFI > 10% is classified as "low fertility potential" and >30% indicates clinical significance.[8][2]

In ART settings, high sperm DFI has been associated with lower blastocyst formation rates in IVF and higher miscarriage rates in couples with male partners aged 40+. A study of 1,205 ART cases found that while sperm quality declined with age, ICSI could partially compensate — male age did not exhibit a pronounced impact on ART pregnancy outcomes when ICSI was used, though this finding requires cautious interpretation.[9][7]

3.4 Paternal Age and Offspring Health

Advanced paternal age carries implications beyond fertility:

- **Chromosomal aneuploidies:** 10% of sperm cells in healthy males carry chromosomal abnormalities, with the incidence of sex chromosome disomy 18 significantly increasing after age 50.[2]
- **De novo mutations:** The paternal contribution to offspring de novo mutations increases by approximately 4% per year (2 base pair mutations per successive year).[2]
- **Neurodevelopmental disorders:** Strong correlations exist between APA and autism spectrum disorders, schizophrenia, and bipolar disorders in offspring.[10][2]
- **Spontaneous abortion:** Increased risk due to elevated DNA fragmentation and oxidative stress.[11]

3.5 Age Thresholds for Male Fertility Decline

Based on the available evidence:[2]

Parameter	Age of Onset of Decline
Total sperm count	> 34 years
Sperm concentration	> 40 years
Normal morphology	> 40 years
Sperm motility	> 43 years
Semen volume	> 45 years
DNA fragmentation (clinically significant)	> 40 years

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4. Lifestyle Modifications to Counteract Age-Related Fertility Decline

4.1 Weight Management and BMI Optimisation

Women: A systematic review and network meta-analysis (35 RCTs, 4,242 women) found that combined exercise plus diet was most effective for BMI reduction (SMD −1.59, 95% CI: −2.56 to −0.63) in overweight/obese reproductive-aged women, and combined exercise plus diet plus ovulation inducers was ranked first (89% probability) for ovulation improvement. In PCOS women undergoing IVF, normal BMI was associated with significantly higher clinical pregnancy rates (OR 1.16, 95% CI: 1.04–1.29) and live birth rates (OR 1.88, 95% CI: 1.56–2.27), while high BMI was linked to increased miscarriage, gestational diabetes (OR 3.96), and gestational hypertension (OR 2.16).[12][13]

Men: A systematic review on obesity interventions and male fertility found that weight loss through lifestyle modification or bariatric surgery significantly improved testosterone levels. In a study of men post-bariatric surgery, total testosterone increased significantly (2.23 to 2.74 nmol/L, p = 0.009), along with improved sexual function. A 5–10% weight loss in obese men is recommended to improve hormonal profiles and semen parameters.[14][15]

4.2 Diet and Nutrition

Mediterranean Diet: In PCOS women, Mediterranean-style and low-glycaemic diets were consistently associated with reduced total and free testosterone, improved insulin sensitivity, and enhanced menstrual regularity. This dietary pattern — rich in fruits, vegetables, whole grains, fish, olive oil, and nuts — provides the micronutrient foundations for reproductive health in both sexes.[16]

Antioxidant-Rich Nutrition: A systematic review and meta-analysis of antioxidant supplementation (vitamins C and E, selenium, zinc, L-carnitine, CoQ10) in subfertile men found significant improvements in sperm DNA fragmentation, sperm concentration, and motility. A randomised clinical trial confirmed that antioxidants were effective in improving sperm DNA integrity and semen parameters in males aged 40 and above. A 6-month oral antioxidant regimen (vitamin C, vitamin E, selenium, zinc, arginine, L-carnitine, CoQ10) in 420 men with idiopathic oligoasthenoteratozoospermia produced significant improvements in sperm concentration ($p < 0.001$), progressive motility ($p < 0.01$), normal morphology ($p < 0.001$), and DFI ($p < 0.001$).[17][18][19]

Specific nutrients for women:
- Folate (400–800 µg/day): Essential for DNA synthesis and neural tube defect prevention.
- Omega-3 fatty acids: May improve oocyte quality and endometrial receptivity.
- Iron: Deficiency impairs ovulation.

Specific nutrients for men:
- Zinc: Critical for testosterone synthesis and spermatogenesis.
- Selenium: Protects against oxidative sperm damage.
- L-carnitine: Supports sperm metabolism and mitochondrial function.

4.3 Targeted Supplementation

4.3.1 Coenzyme Q10 (CoQ10)

CoQ10 is an essential mitochondrial antioxidant. In a network meta-analysis of 16 RCTs (2,323 women with poor ovarian response), CoQ10 demonstrated the strongest effect on live birth rate (OR 2.36, 95% CI: 1.07–5.38; SUCRA 89.9%) and significantly improved clinical pregnancy rate (OR 2.22, 95% CI: 1.05–4.71) and number of oocytes retrieved (WMD 1.34). Given that mitochondrial dysfunction is a primary driver of oocyte ageing, CoQ10 is particularly relevant for women over 35.[20]

4.3.2 Dehydroepiandrosterone (DHEA)

DHEA (typically 75 mg/day for 8–12 weeks) improved IVF outcomes in women with diminished ovarian reserve. A meta-analysis of RCTs showed improved clinical pregnancy rates and reduced miscarriage rates. In IUI patients, 12 weeks of DHEA significantly increased AMH ($p < 0.01$) and decreased FSH ($p = 0.03$). Notably, DHEA was shown to eliminate the treatment resistance seen in hypo-androgenic PCOS-like phenotype (H-PCOS), which evolves from "lean" PCOS in the mid-thirties through declining adrenal androgen production.[21][22][23]

4.3.3 Vitamin D

Women with sufficient vitamin D (> 30 ng/mL) had live birth rates of 37.7% in ART compared to 23.2% in deficient women (< 20 ng/mL). In a retrospective study of 1,113 IVF patients, cumulative live birth rate was 50.9% in vitamin D-replete women versus 43.9% in deficient women. In PCOS patients, a systematic review confirmed that higher baseline vitamin D levels were associated with improved IVF outcomes. Supplementation of 1,000–4,000 IU/day is recommended for deficient women before and during ART cycles.[24][25][26]

4.3.4 Myo-Inositol

A meta-analysis of 11 RCTs (981 participants) found that myo-inositol significantly improved metaphase II oocyte rates in IVF (OR 1.55), with PCOS women showing even greater benefit (OR 1.97). For men, myo-inositol improved total motility (SMD 0.90), progressive motility (SMD 1.48), testosterone levels (SMD 0.54), and reduced sperm DNA fragmentation.[27][28]

4.4 Physical Activity

Regular moderate exercise (150–300 minutes/week aerobic activity plus 2–3 resistance sessions) improves insulin sensitivity, reduces systemic inflammation, and supports hormonal balance. The network meta-analysis demonstrated that exercise combined with dietary modification achieved the greatest ovulation improvement and BMI reduction in overweight/obese women.[29][12]

However, excessive exercise can be counterproductive:
- **Women:** Intense endurance training can suppress GnRH pulsatility, leading to hypothalamic amenorrhoea.
- **Men:** Overtraining can reduce testosterone and impair spermatogenesis.

A 3-month combined lifestyle intervention (personalised diet, moderate exercise) plus antioxidant therapy significantly reduced sperm DNA fragmentation index in infertile men with elevated SDF and history of failed IVF/ICSI.[30]

4.5 Smoking Cessation

Smoking has well-documented detrimental effects across both sexes:
- **Women:** Accelerates ovarian ageing (smokers reach menopause 1–4 years earlier), reduces AMH levels, impairs oocyte quality, and decreases IVF success rates.
- **Men:** Classified as a minor risk factor for male infertility, associated with reduced sperm concentration, motility, morphology, and increased DNA fragmentation. Advanced paternal age compounds the effects of smoking on sperm DFI.[31]

Smoking cessation is one of the highest-impact modifiable factors for both age groups.

4.6 Alcohol and Caffeine Reduction

- **Alcohol:** Even moderate intake is associated with reduced fecundity. Complete avoidance is recommended during ART cycles and pregnancy.
- **Caffeine:** Moderate intake (< 200 mg/day; approximately 1–2 cups of coffee) is generally safe, though some guidelines recommend further restriction during treatment.

4.7 Environmental and Occupational Modifications

Endocrine-disrupting chemicals (EDCs), heavy metals, and environmental pollutants pose age-compounding risks to fertility:[32][31]

- Avoid BPA (found in plastics, receipt paper) and phthalates (found in personal care products, food packaging).
- Minimise testicular heat exposure (avoid hot baths, saunas, tight clothing, laptop on lap).
- Reduce occupational chemical exposure (pesticides, solvents, heavy metals) where possible.
- Maintain adequate sleep (7–8 hours) to support hormonal regulation.

4.8 Stress Management and Psychological Support

While direct causal links between psychological stress and IVF failure remain debated, chronic stress activates the hypothalamic-pituitary-adrenal (HPA) axis, which can interfere with GnRH pulsatility and reproductive hormone secretion. A randomised controlled trial of smartphone-supported Positive Adjustment Coping Intervention (PACI) for couples undergoing fertility treatment demonstrated psychological benefits. Mindfulness, cognitive-behavioural therapy, and sleep optimisation are recommended adjuncts.[33]

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5. Age-Specific Recommendations: A Clinical Summary

5.1 Women

Age Group	Fertility Status	Key Blood Markers	Priority Interventions
20–29	Peak fertility; highest IVF success (LBR ~40%)	AMH typically 3.5–7.0+ ng/mL; FSH < 8 mIU/mL	Establish healthy habits; smoking cessation; folate supplementation; address PCOS if present
30–34	Gradual decline begins; AMH decline rate ~0.2 ng/mL/year	AMH 2.0–5.0 ng/mL; AFC 12–16	Weight optimisation; Mediterranean diet; consider AMH/AFC baseline; fertility counselling if not yet trying
35–37	Accelerated decline; IVF LBR drops to ~21%	AMH 1.5–3.0 ng/mL; FSH may begin rising	Active fertility assessment; consider egg freezing; CoQ10 (400–600 mg/day); vitamin D optimisation; DHEA if DOR
38–40	Steep decline; miscarriage rate doubles to 30%	AMH 0.5–2.5 ng/mL; FSH often > 10 mIU/mL	Urgent fertility evaluation; aggressive ART protocols; freeze-all strategy may benefit; full supplement regimen

| 40–44 | Very low natural fertility; IVF LBR ~6%; miscarriage ~55% | AMH often < 1.0 ng/mL | Realistic counselling; donor oocyte discussion; if using own oocytes, FET preferred (aOR 1.53–2.63 vs fresh)[6] |
| ≥ 45 | Negligible fertility with own oocytes | Severely diminished AMH/AFC | Donor oocytes primary option[3] |

5.2 Men

Age Group	Fertility Status	Key Changes	Priority Interventions
20–34	Peak sperm parameters	DFI ~15–19%; testosterone optimal	Healthy lifestyle foundation; avoid tobacco, excess alcohol; maintain BMI 18.5–25
35–39	Subtle decline begins	DFI ~20%; morphology starts declining	Antioxidant-rich diet; regular moderate exercise; avoid testicular heat; consider DFI testing if struggling
40–44	Measurable decline	DFI ~26%; motility declining; testosterone falling	Active antioxidant supplementation (vitamin C, E, zinc, selenium, CoQ10); sperm DNA fragmentation test recommended; consider ICSI over IVF
45+	Significant decline	DFI ~32%; volume and motility reduced; 2× de novo mutation rate vs age 20	Full fertility evaluation; lifestyle + antioxidant intervention (3–6 months before ART); ICSI preferred; genetic counselling for offspring risks

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6. Discussion

The evidence presented in this report demonstrates that age-related fertility decline is a multifactorial, progressive process affecting both sexes, though with different timelines and mechanisms. For women, the critical window of accelerated decline occurs between ages 35 and 40, driven by quantitative and qualitative oocyte depletion. The SART CORS data (228,171 cycles) provide compelling evidence that even within ART, success rates decline dramatically with age — but strategic choices such as freeze-all protocols can partially compensate.[6]

For men, the fertility decline is more gradual but no less significant. The doubling of sperm DNA fragmentation from age 25 to 45, combined with epigenetic changes and increasing de novo mutations, underscores the importance of not dismissing paternal age as a fertility factor. The finding that antioxidant therapy can significantly reduce DFI in men over 40 offers a concrete, actionable intervention.[19][17][2]

Lifestyle modifications represent the most accessible and cost-effective strategy for mitigating age-related decline. The evidence is strongest for weight optimisation in women (particularly those with PCOS or BMI > 25), where combined diet and exercise interventions improve ovulation rates and IVF outcomes. For men, the combination of antioxidant supplementation with lifestyle modification reduced DFI in men with prior ART failure.[13][30][12]

Supplementation with CoQ10, DHEA, vitamin D, and myo-inositol has demonstrated promising results in improving ART outcomes, particularly for women with diminished

ovarian reserve. CoQ10's effect on live birth rates (OR 2.36) in poor responders is particularly noteworthy and mechanistically logical, given the role of mitochondrial function in oocyte competence.[20]

A critical limitation of the current evidence base is the predominance of observational and retrospective studies, with relatively few large RCTs specifically designed to evaluate lifestyle interventions in age-stratified fertility populations. Future research should prioritise prospective, randomised studies integrating multiple biomarkers with treatment outcomes across defined age brackets.

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