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Review Article

Impact of advanced maternal age on assisted reproductive technology outcomes: biological mechanisms, clinical implications and management strategies

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ABSTRACT

Delayed childbearing has increased the number of women of advanced maternal age (AMA) seeking assisted reproductive technology (ART). AMA, defined as ≥35 years, is linked to reduced ovarian reserve, poor oocyte quality, and higher pregnancy risks. Despite advances in IVF, ICSI and frozen embryo transfer (FET), maternal age remains a key factor affecting success rates. This review summarizes the biological changes associated with reproductive aging and their impact on ART outcomes such as implantation, pregnancy rates, miscarriage and perinatal outcomes. Literature shows that aging leads to mitochondrial dysfunction, chromosomal abnormalities and reduced embryo quality, resulting in lower pregnancy and live birth rates and higher miscarriage rates in women ≥35 years. While FET may improve uterine conditions, it cannot fully overcome the decline in oocyte quality. AMA is also associated with increased risks such as hypertension, gestational diabetes and preterm birth. In conclusion, advanced maternal age significantly affects ART outcomes, and careful patient counselling and individualized treatment strategies are essential to improve success rates.

Keywords: Advanced maternal age, Assisted reproductive technology, In vitro fertilization, Frozen embryo transfer

INTRODUCTION

Over the past few decades, there has been a profound demographic shift toward delayed childbearing across both developed and developing nations. Increasing educational attainment, career prioritization, financial stability and evolving social norms have contributed to women postponing pregnancy into their mid-to-late thirties and beyond. Consequently, the proportion of women seeking fertility treatment at AMA, traditionally defined as 35 years or older, has risen substantially.[1] This trend has significant implications for reproductive medicine and ART practice. Female reproductive potential is intrinsically linked to age. Unlike many other physiological systems, ovarian aging begins early and progresses steadily over time. Women are born with a finite pool of primordial follicles, estimated at approximately one to two million at birth, which declines to around 300,000-500,000 at puberty.[2] This follicular reserve continues to decrease with age through atresia and ovulation, with a marked acceleration after the age of 35. However, the decline in fertility is not solely quantitative; qualitative deterioration of oocytes plays a pivotal role in reduced fecundity among older women.[3] Advanced maternal age is associated with several biological alterations that compromise reproductive outcomes. These include diminished ovarian reserve, mitochondrial dysfunction, oxidative stress, meiotic spindle instability, telomere shortening and impaired chromosomal segregation during meiosis. Such changes increase the likelihood of oocyte aneuploidy, which is the primary contributor to reduced implantation rates and increased miscarriage risk in older women.[4] Studies evaluating trophectoderm biopsies have demonstrated a progressive rise in embryonic chromosomal abnormalities with advancing maternal age, particularly after 37-38 years. These genetic abnormalities significantly influence ART success rates, even in the presence of optimal laboratory conditions.[5] (Figure 1).

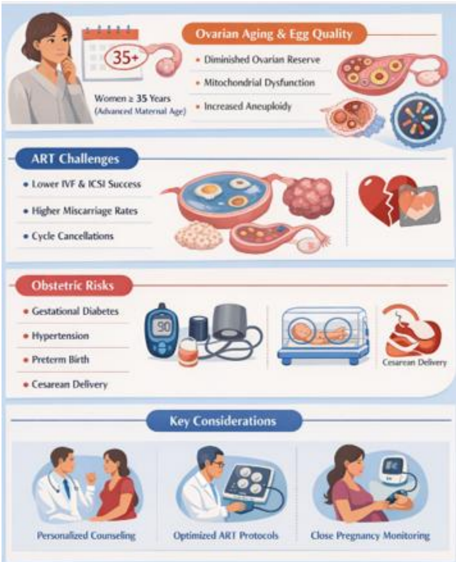

Figure 1: Impact of advanced maternal age on reproductive biology and assisted reproductive technology outcomes.[26]

In the context of assisted reproductive technology, maternal age remains one of the most robust predictors of treatment outcome. Clinical pregnancy and live birth rates decline progressively with increasing age, while miscarriage and cycle cancellation rates rise.[6] The introduction of ICSI, time-lapse embryo monitoring, PGT-A, and vitrification-based cryopreservation has improved ART efficiency; however, these technological advancements cannot fully reverse the intrinsic biological limitations imposed by oocyte aging.[7] The adoption of the "freeze-all" strategy and subsequent FET cycles has further transformed ART practice.[8] FET is associated with improved endometrial receptivity by avoiding the supraphysiological hormonal milieu induced by controlled ovarian stimulation. Moreover, vitrification has significantly enhanced post-thaw embryo survival rates.[9] Despite these advances, it remains uncertain whether optimizing the uterine environment through FET can meaningfully offset age-related declines in oocyte competence.[10] While endometrial receptivity appears relatively preserved with advancing age compared to oocyte quality, implantation potential is ultimately dependent on embryonic chromosomal integrity, which is strongly age-dependent.[11] Beyond implantation and early pregnancy outcomes, advanced maternal age is independently associated with increased obstetric and perinatal risks.[12] These include gestational diabetes mellitus, hypertensive disorders of pregnancy, placenta previa, caesarean delivery, preterm birth, foetal growth restriction, and stillbirth. Even in ART conceived pregnancies, where close monitoring is standard, maternal morbidity increases with advancing age.[13] Thus, the implications of delayed childbearing extend beyond conception to encompass the entire continuum of pregnancy and neonatal health.

Given the increasing reliance on ART among women of advanced reproductive age, a comprehensive understanding of the biological mechanisms underlying reproductive aging and its clinical consequences is essential for optimizing treatment strategies and counselling.[14] Although numerous studies have explored age-related ART outcomes, variability in patient selection, embryo stage at transfer, and inclusion of donor cycles complicates interpretation. Furthermore, rapid technological advancements necessitate continual reassessment of how maternal age influences outcomes in contemporary ART practice.[15] This narrative review aims to provide a detailed overview of the biological basis of reproductive aging and its impact on assisted reproductive technology outcomes, including fertilization, embryo development, implantation, clinical pregnancy, miscarriage, and perinatal complications.[16] In addition, we discuss the role of frozen embryo transfer strategies, clinical counselling considerations, and emerging therapeutic directions aimed at mitigating age-related reproductive decline.

BIOLOGICAL BASIS OF REPRODUCTIVE AGING

Female reproductive aging is a complex, multifactorial biological process characterized by both quantitative depletions of ovarian reserve and qualitative deterioration of oocyte competence.[17] Unlike other organ systems that rely on continuous cellular renewal, the ovary contains a finite number of primordial follicles established during foetal life.[18] This non-renewable follicular pool undergoes continuous atresia throughout childhood and the reproductive years, with an accelerated decline beginning in the mid-30s. While the reduction in follicle number contributes to diminished ovarian reserve, it is increasingly recognized that the decline in oocyte quality plays a more critical role in age-related infertility.[19]

At the cellular level, oocyte aging is strongly associated with mitochondrial dysfunction. Oocytes are highly metabolically active cells that depend on mitochondrial ATP production to support spindle formation, chromosomal segregation, fertilization, and early embryonic development.[20] With advancing age, mtDNA accumulates mutations, oxidative damage increases, and mitochondrial membrane potential declines.[21] These alterations impair ATP synthesis, leading to defective meiotic spindle assembly and reduced developmental competence. Because mitochondria are maternally inherited, age-related mitochondrial defects can also influence early embryogenesis and implantation potential.[22]

Chromosomal segregation errors represent another central mechanism underlying reproductive aging. Human oocytes remain arrested in prophase I of meiosis from foetal life until ovulation, a period that may span decades.[23] Prolonged meiotic arrest predisposes oocytes to cohesion loss between sister chromatids and deterioration of spindle assembly checkpoints. As maternal age increases, these molecular safeguards become less efficient, resulting in nondisjunction events and aneuploidy.[24] The incidence of embryonic chromosomal abnormalities rises sharply after 35 years and increases exponentially after 40 years, explaining the higher rates of implantation failure, biochemical pregnancy, and spontaneous miscarriage observed in advanced maternal age. Studies using comprehensive chromosomal screening have demonstrated that aneuploidy rates may exceed 60-80% in women over 40 years undergoing assisted reproduction.[25] (Figure 2).

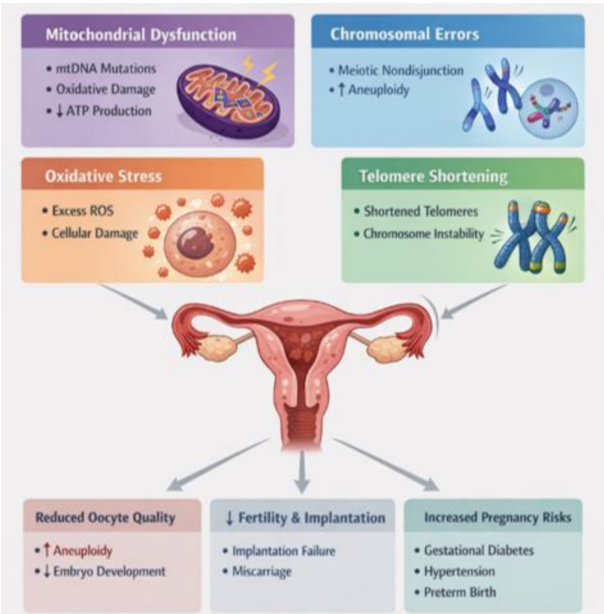

Figure 2: Biological mechanisms underlying age-related decline in female reproductive function.[26]

Oxidative stress further contributes to age-related reproductive decline. ROS accumulate within aging oocytes due to reduced antioxidant defence mechanisms and impaired mitochondrial function.[26] Excess ROS can damage DNA, proteins, and lipid membranes, thereby compromising cytoplasmic maturation and meiotic integrity. Additionally, oxidative stress affects the surrounding granulosa cells and follicular microenvironment, disrupting oocyte–somatic cell communication that is essential for optimal folliculogenesis.[26] Telomere shortening is another hallmark of reproductive aging. Telomeres, the protective nucleotide sequences at chromosomal ends, progressively shorten with each cell division. In oocytes, shortened telomeres may impair chromosomal stability and increase susceptibility to meiotic errors. Experimental studies suggest that telomere dysfunction may contribute to abnormal spindle formation and reduced embryo viability in older women.[20,21] Beyond intrinsic oocyte factors, alterations in ovarian stromal function and hormonal signalling also influence reproductive aging. Aging ovaries exhibit decreased responsiveness to FSH, altered steroidogenesis, and impaired follicular vascularization. AMH levels decline progressively, reflecting diminished follicular reserve.[19,20] Although endometrial receptivity appears relatively preserved compared to oocyte competence, subtle age-related changes in uterine blood flow, immune modulation, and decidualization capacity may also affect implantation success. Importantly, reproductive aging is not a threshold phenomenon but rather a continuum. While chronological age is commonly used as a clinical marker, biological ovarian age varies among individuals.[21]

Genetic predisposition, environmental exposures, lifestyle factors, and metabolic health can influence the rate of ovarian aging. Nevertheless, maternal age remains the strongest independent predictor of oocyte aneuploidy and ART outcomes.[25,26] Collectively, these interconnected mechanisms mitochondrial dysfunction, chromosomal segregation errors, oxidative stress, telomere shortening, and altered hormonal dynamics explain the progressive decline in fertility observed with advancing maternal age.[22,23] Understanding these biological processes is essential for interpreting age-related ART outcomes and for developing emerging therapeutic strategies aimed at improving reproductive potential in older women.[20]

Impact of advanced maternal age on ART outcomes

Maternal age is one of the strongest independent predictors of ART success. Despite advances in ovarian stimulation, embryo culture, and cryopreservation, reproductive outcomes decline significantly with age, primarily due to reduced oocyte quality rather than uterine factors.[21]

Fertilization and ovarian response

Women of advanced maternal age exhibit diminished ovarian reserve, reflected by lower AMH levels, higher basal FSH, and reduced antral follicle count. This results in a poorer response to ovarian stimulation, fewer retrieved oocytes, and reduced embryo availability. Although fertilization rates per oocyte may remain relatively stable with ICSI, the overall number of viable embryos declines.[22] (Figure 3).

Embryo development

Embryos from older women demonstrate higher rates of developmental arrest and reduced progression to the blastocyst stage. Increased chromosomal abnormalities and compromised cytoplasmic competence contribute to reduced embryo quality and viability.[23] (Figure 3).

Implantation

Implantation rates decline with advancing age, primarily due to increased embryonic aneuploidy. While endometrial receptivity is relatively preserved, FET improves uterine conditions but does not overcome intrinsic embryo defects.[24]

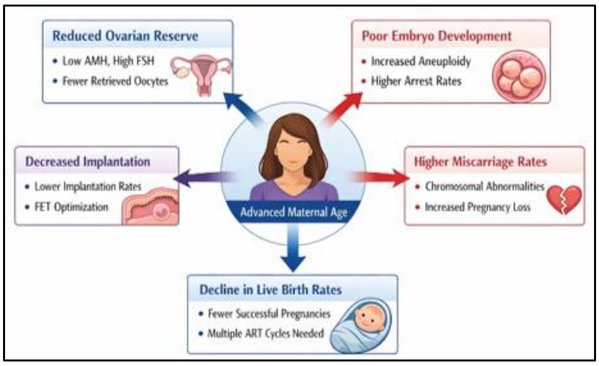

Figure 3: Impact of advanced maternal age on assisted reproductive technology outcomes.[26]

Clinical pregnancy and live birth

Clinical pregnancy and live birth rates decrease progressively after 35 years, with a sharp decline beyond 38-40 years. Technological advancements improve efficiency but cannot fully compensate for age-related biological limitations.[25]

Miscarriage

Miscarriage rates increase significantly with age, largely due to chromosomal abnormalities. While PGT-A reduces miscarriage risk per transfer, it does not improve cumulative live birth rates due to limited euploid embryos.[26]

Cumulative outcomes

Cumulative live birth rates decline with age due to lower oocyte yield and embryo availability. Older women often require multiple ART cycles, increasing physical, emotional, and financial burden. Donor oocytes, however, largely overcome age-related declines.[27] (Figure 3).

ROLE OF FROZEN EMBRYO TRANSFER IN ADVANCED MATERNAL AGE

Frozen embryo transfer has become integral to modern ART practice due to improved vitrification techniques and better endometrial preparation. By avoiding the supraphysiological hormonal environment of fresh cycles, FET may enhance implantation conditions.[29] However, in advanced maternal age, the benefits of FET are limited by poor oocyte quality and high aneuploidy rates. While post-thaw embryo survival is high and comparable across age groups, the main challenge remains the reduced availability of euploid embryos.[30] FET is particularly useful in conjunction with PGT-A, allowing selection of chromosomally normal embryos and reducing miscarriage risk. Nevertheless, it does not increase the total number of viable embryos or fully overcome age-related fertility decline.[31]

MATERNAL AND NEONATAL RISKS IN ADVANCED MATERNAL AGE

Advanced maternal age is associated with increased obstetric and neonatal risks, even in ART pregnancies. These include hypertensive disorders, gestational diabetes, placental abnormalities, caesarean delivery, and preterm birth. Stillbirth risk increases with age due to placental insufficiency and chromosomal abnormalities. Neonates are at higher risk of low birth weight, prematurity, and NICU admission, although outcomes improve with singleton pregnancies and proper care. Despite these risks, many women achieve successful outcomes with appropriate monitoring, risk stratification, and modern obstetric management.[31]

CLINICAL COUNSELING AND MANAGEMENT STRATEGIES

Management of advanced maternal age requires individualized, evidence-based approaches. Counselling should address realistic expectations regarding reduced fertility, increased miscarriage risk, and the potential need for multiple cycles. Assessment of ovarian reserve like AMH, AFC, FSH helps guide treatment planning. Individualized stimulation protocols aim to maximize oocyte yield while minimizing burden. Blastocyst culture and selective use of PGT-A may improve per-transfer outcomes. FET and single embryo transfer are recommended to optimize safety and reduce multiple pregnancy risk. Optimization of general health, including metabolic and lifestyle factors, is essential to reduce obstetric complications. Psychological support is important due to increased emotional stress in this population. In selected cases, donor oocyte programs should be discussed as they significantly improve success rates.[20,21]

METHODS

Literature search strategy

A comprehensive literature search was conducted to identify studies evaluating the impact of advanced maternal age on assisted reproductive technology outcomes and associated obstetric complications. Electronic databases, including PubMed, Scopus, and Google Scholar, were searched for articles published up to January 2026. The search strategy incorporated combinations of the following keywords: “advanced maternal age,” “reproductive aging,” “assisted reproductive technology,” “in vitro fertilization,” “intracytoplasmic sperm injection,” “frozen embryo transfer," "aneuploidy," "implantation rate," "miscarriage," and "perinatal outcomes." Boolean operators like AND/OR were applied to refine the search. Original research articles, clinical trials, observational cohort studies, registry-based analyses, and relevant review articles published in English were included. Studies involving donor oocytes were considered for comparative purposes but analysed separately from autologous cycles. Animal studies were excluded unless they provided significant mechanistic insights into oocyte aging. Priority was given to large cohort studies, registry data, and recent publications reflecting contemporary ART practices, including vitrification-based cryopreservation and preimplantation genetic testing for aneuploidy. Additional studies were identified through manual screening of reference lists of selected articles. As this is a narrative review, formal systematic review methodology, quality assessment, and meta-analytic synthesis were not performed. The aim was to provide a comprehensive and clinically relevant overview of current evidence on the biological mechanisms and ART outcomes associated with advanced maternal age.

Future directions and emerging therapies

Despite advances in assisted reproductive technology, age-related decline in oocyte quality remains the primary limitation to reproductive success in AMA.[30] Current research focuses on improving oocyte competence, reducing aneuploidy, and enhancing cumulative live birth rates. Mitochondrial-targeted therapies, including antioxidants and coenzyme Q10, aim to improve oocyte bioenergetics and reduce oxidative stress.[29] While preclinical studies show promising results, clinical evidence remains limited and requires validation through large randomized controlled trials. Advances in PGT-A and next-generation sequencing have improved embryo selection accuracy.[20] Integration of AI with morpho kinetics and imaging analysis may further refine embryo selection, although these approaches cannot overcome intrinsic chromosomal abnormalities.[27]

Emerging ovarian rejuvenation strategies, such as platelet-rich plasma and stem cell-based therapies, remain experimental with limited clinical validation and unresolved safety concerns.[25] Similarly, targeting meiotic errors at the molecular level remains biologically complex and is not yet clinically feasible.[26]

Lifestyle optimization, including improved metabolic health and reduction of oxidative stress, may support reproductive function, although it cannot reverse chronological aging.[20] Elective oocyte cryopreservation at younger ages represents an effective fertility preservation strategy.[28] Currently, donor oocytes remain the most effective approach to overcome age-related infertility, largely eliminating the impact of oocyte aneuploidy.[30] Future research should prioritize well-designed prospective trials, standardized outcome reporting, and individualized treatment strategies integrating molecular and computational approaches.

DISCUSSION

This review highlights that maternal age remains the most critical determinant of ART outcomes despite technological advancements. The decline in reproductive potential is primarily driven by oocyte aging, characterized by mitochondrial dysfunction, oxidative stress, and increased aneuploidy.[22-28] While ART technologies optimize fertilization and implantation conditions, they cannot reverse intrinsic biological aging processes. The role of preimplantation genetic testing for aneuploidy remains debated; although it reduces miscarriage rates, its impact on cumulative live birth rates is limited, particularly in women with diminished ovarian reserve.[20] Frozen embryo transfer improves endometrial receptivity but does not address embryo-derived chromosomal abnormalities.[10]

This distinction is essential for appropriate patient counselling. Advanced maternal age is also associated with increased obstetric and neonatal risks, including hypertensive disorders, gestational diabetes, preterm birth and stillbirth.[12-20] These findings emphasize the need for multidisciplinary clinical management. Emerging approaches such as AI-based embryo selection and mitochondrial-targeted therapies show promise but lack robust clinical validation.[27,29,30] Further large-scale, standardized studies are required before routine clinical implementation. This review has limitations inherent to narrative methodology, including the absence of formal quality assessment and meta-analysis. Additionally, heterogeneity in study design, patient populations, and ART protocols may influence interpretation of outcomes.

CONCLUSION

Advanced maternal age is a major determinant of reproductive outcomes in ART. The progressive decline in oocyte quality, driven by mitochondrial dysfunction, chromosomal errors, and oxidative stress, leads to reduced implantation, lower live birth rates, and increased miscarriage risk. Although advancements such as vitrification-based FET, extended embryo culture, and preimplantation genetic testing for aneuploidy have improved ART efficiency, they do not fully overcome age-related biological limitations.

Consequently, cumulative success rates remain strongly age-dependent. Advanced maternal age is also associated with increased obstetric and perinatal risks, highlighting the importance of comprehensive counselling, individualized treatment strategies, and close clinical monitoring. Emerging therapies offer promising future directions but are not yet ready for routine clinical application. Early fertility awareness, timely family planning, and consideration of fertility preservation strategies remain essential components of reproductive care.

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