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Anthropometric components of somatotype (endomorphy, mesomorphy, and ectomorphy) according to the Heath-Carter method and body composition components by the Matiegka method in women with infertility

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Infertility in women of advanced reproductive age represents an important clinical and demographic challenge. Female reproductive potential declines significantly after the age of 35 due to progressive ovarian aging, reduced ovarian reserve, and increasing chromosomal abnormalities in oocytes. Consequently, infertility becomes more common, and reproductive outcomes are increasingly influenced by metabolic status, endocrine regulation, and overall systemic health.
Aim – to synthesize current evidence on the relationship between body composition, fat distribution, and reproductive outcomes in women of advanced reproductive age, with particular emphasis on the role of metabolic and endocrine factors.
Special attention is given to the interaction between constitutional body morphology (somatotype), body composition parameters, and fertility, including outcomes of assisted reproductive technologies. In addition, existing gaps in the literature regarding the combined assessment of somatotype according to the Heath-Carter method and body composition based on the Matiegka approach in relation to female infertility are identified. Limited data on the interaction between these constitutional characteristics and stress-related adaptive responses are assessed, particularly under conditions of prolonged psychosocial stress. A conceptual framework integrating somatotype, body composition, metabolic regulation, and adaptive mechanisms in the context of female reproductive function is developed, and the need for further research, especially in populations exposed to chronic stress conditions such as those observed in Ukraine, is substantiated.
Conclusion. Such investigations, especially when conducted in populations exposed to prolonged war-related stress, may provide new insights into the constitutional and adaptive determinants of female reproductive function and contribute to the development of more personalized diagnostic and therapeutic strategies for infertility.
The authors declare no conflict of interest.
Keywords: somatotype, Heath-Carter method, body composition, endomorphy, mesomorphy, ectomorphy, female infertility, anthropometry, reproductive endocrinology.

Антропометричні компоненти соматотипу (ендоморфія, мезоморфія та ектоморфія) за методом Heath-Carter та компоненти складу тіла за методом Matiegka в жінок із безпліддям

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Безпліддя в жінок старшого репродуктивного віку є важливою клінічною та демографічною проблемою. Жіночий репродуктивний потенціал значно знижується після 35 років через прогресуюче старіння яєчників, зниження оваріального резерву та збільшення хромосомних аномалій в ооцитах. Як наслідок, безпліддя стає більш поширеним, а репродуктивні результати дедалі більше залежать від метаболічного статусу, ендокринної регуляції та загального системного здоров'я.
Мета – синтезувати сучасні дані про взаємозв'язок між складом тіла, розподілом жиру та репродуктивними результатами в жінок старшого репродуктивного віку, з особливим акцентом на ролі метаболічних та ендокринних факторів.
Особливу увагу приділено взаємодії між конституційною морфологією тіла (соматотип), параметрами складу тіла та фертильністю, зокрема результати допоміжних репродуктивних технологій. Виявлено існуючі прогалини в літературі щодо комбінованої оцінки соматотипу за методом Heath-Carter та складу тіла на основі підходу Matiegka через жіноче безпліддя. Оцінено обмежені дані про взаємодію між цими конституційними характеристиками та адаптивними реакціями, пов'язаними зі стресом, особливо в умовах тривалого психосоціального стресу. Розроблено концептуальну основу, що інтегрує соматотип, склад тіла, метаболічну регуляцію та адаптивні механізми в контексті жіночої репродуктивної функції, а також обґрунтовано необхідність подальших досліджень, особливо в популяціях, які зазнають таких хронічних стресових умов, як ті, що спостерігаються в Україні.

Висновок. Такі дослідження, особливо проведені в популяціях, які зазнали тривалого стресу, пов'язаного з війною, можуть дати нове розуміння конституційних та адаптивних детермінант жіночої репродуктивної функції та сприяти розробці більш персоналізованих діагностичних та терапевтичних стратегій лікування безпліддя.
Автори заявляють про відсутність конфлікту інтересів.
Ключові слова: соматотип, метод Heath-Carter, склад тіла, ендоморфія, мезоморфія, ектоморфія, жіноче безпліддя, антропометрія, репродуктивна ендокринологія.

Body composition characteristics demonstrate substantial influence on fertility outcomes in women over 35 years, with effects varying by age, body fat distribution, and specific fertility parameters assessed. In late reproductive age women (40–52 years), obesity is associated with 77% lower anti-Müllerian hormone (AMH) levels compared to normal weight women, though antral follicle counts remain similar [58]. For in vitro fertilization outcomes, the impact of obesity differs markedly by age: women under 35 experience compromised embryo quality [44], while women over 35 show significantly reduced clinical pregnancy rates (10% vs 24.5% in normal weight controls, p=0.02) [64]. Cumulative live birth rates decrease progressively with increasing body mass index (BMI), with obese women showing 26% lower odds compared to normal weight women (adjusted odds ratio (OR) 0.74, 95% confidence interval (CI) 0.62–0.87) [50]. The benefit of weight loss depends critically on age and magnitude: women up to 35 years may benefit from modest reduction within one year, whereas those over 38 years require substantial weight loss (⩾5 kg/m^2) in compressed timeframes to offset accelerating age-related decline [50]. Body fat distribution measures often outperform BMI alone in predicting fertility outcomes. Each 0.1 unit increase in waist-hip ratio (WHR) produces a 30% decrease in conception probability per cycle (hazard ratio (HR) 0.706, 95% CI 0.562–0.887) [71], and women with waist circumference >80 cm show lower ovarian sensitivity index independent of BMI [3]. Even eutrophic women with elevated waist circumference demonstrate markedly reduced gestation rates (14.3% vs 38.9%, p=0.002) [10], indicating that central adiposity exerts effects beyond overall body weight. The evidence suggests body composition influences multiple fertility parameters through distinct mechanisms that compound age-related reproductive decline, with central obesity showing particularly strong associations mediated through metabolic dysfunction rather than simple adiposity. The studies varied considerably in design and setting. Fourteen studies were retrospective in design [6,7,9, 12,33,44,50,53,64,71], six were prospective [3,24, 36,47,71], seven were cross-sectional [10,13,20,54, 58,69], and one was a systematic review [38]. The majority were conducted in a fertility clinic or hospital-based settings. Sample sizes ranged from 28 to 14,213 participants, with recruitment periods spanning from the 1990s to 2025.

The aim of this literature review is to synthesize current evidence on the relationship between body composition, fat distribution, and reproductive outcomes in women of advanced reproductive age, with particular emphasis on the role of metabolic and endocrine factors.

Body composition measures assessed across studies. BMI was the most commonly assessed body composition measure, used in 23 of 25 studies [3,6,7, 10,12,13,20,24,31,33,36,44,47,50,53,54,57,58,64,66, 69,71]. BMI was typically categorized according to WHO standards: normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obesity (⩾30 kg/m^2) [3,47,53]. WHR was assessed in nine studies [3,9,10,24,38,57,66,69,71]. Common cutoff values included 0.80 [66,71] and 0.85 [3,10]. Waist circumference (WC) was measured in eight studies [3, 10,38,69,71], with a cutoff of 80 cm frequently used to distinguish central obesity [3,10]. Body fat percentage was assessed using various methods, including bioelectrical impedance analysis (BIA) [10, 24,57] and dual-energy X-ray absorptiometry (DXA) [13]. One study employed a particularly comprehensive assessment of body composition using dual-energy DXA to evaluate the distribution of body fat and the degree of bone mineralization [13]. More sophisticated obesity-related indicators were evaluated in one large population-based study. These included a body shape index (ABSI), weight-adjusted waist index (WWI), body roundness index (BRI), waist-to-height ratio (WHtR), relative fat mass (RFM), and the non-high-density lipoprotein (non-HDL) to high-density lipoprotein cholesterol ratio (NHHR) [69].

Synthesis. The evidence reveals complex, age-dependent relationships between body composition and fertility outcomes that cannot be adequately captured by simple BMI thresholds alone. The interaction between body composition and age creates distinct fertility profiles. In women under 35 years, obesity primarily affects embryo quality parameters – utilization rates, discard rates, and cryopreservation outcomes – while pregnancy rates appear relatively preserved [44]. In contrast, women over 35 years with obesity show no clear differences in embryo quality markers but experience significant reductions in clinical pregnancy rates [64]. This apparent discrepancy may be explained by the greater capacity of younger women to compensate for obesity-related metabolic disturbances through relatively preserved oocyte competence and endometrial receptivity, whereas in older women, obesity may further aggravate the pre-existing decline associated with reproductive aging. Apparent null findings in some studies may reflect methodological limitations rather than a true absence of effect. The study reporting no significant BMI effects in women aged ⩾37 years [33] used relatively broad BMI categories and may have been underpowered to detect modest differences within an already age-compromised population. In contrast, studies demonstrating significant associations used more detailed BMI stratification, including class I, II, and III obesity [53], or analyzed BMI as a continuous variable [50], thereby allowing detection of dose-response relationships that may be obscured in categorical analyses. The predominance of retrospective designs across the included studies introduces the possibility of residual confounding and selection-related bias, as women with higher BMI may differ in unmeasured characteristics such as severity of insulin resistance, duration of obesity exposure, or comorbidity burden. However, the consistency of the observed associations across prospective cohorts [3,71], cross-sectional analyses [54,58], and retrospective studies [50,53] supports the likelihood that these findings reflect underlying biological relationships rather than methodological artifact alone. Synthesizing across studies, several principles emerge for understanding the effects of body composition in women over 35 years. First, different fertility outcomes appear to respond to different aspects of body composition. Ovarian reserve markers, particularly AMH, show strong associations with BMI [54,58]; ovarian response to stimulation appears to be related to both BMI and central adiposity [3,47]; embryo quality appears more sensitive to BMI in younger than in older women [44]; and clinical pregnancy rates show one of the clearest age-dependent interactions with obesity [6,64]. Second, body fat distribution metrics, particularly WHR and WC, often appear to outperform BMI in predicting fertility outcomes [66,69,71], suggesting that the metabolic dysfunction associated with central obesity may be more important than overall adiposity alone. This is particularly evident in women with normal BMI but elevated WC, who show markedly reduced pregnancy rates [10]. Third, the impact of body composition on fertility appears to be non-linear and threshold-dependent. Multiple studies identified specific cut-points – WHR >0.80 [66,71], WC >80 cm [3,10], and body fat mass >16.65 kg [24] – beyond which fertility impairment becomes more pronounced. This supports intervention strategies aimed at achieving clinically meaningful body composition targets rather than non-specific weight reduction alone. Fourth, the adverse fertility consequences of obesity in women over 35 years appear to result from the combined influence on multiple reproductive parameters, including diminished ovarian reserve [54,58], impaired ovarian response to stimulation [3,47], compromised oocyte and embryo quality [53], and reduced implantation potential [64]. The relative contribution of each pathway may vary depending on baseline ovarian reserve, age, and the assisted reproduction protocol used. Taken together, these findings indicate that body composition, particularly central adiposity, exerts clinically meaningful effects on fertility in women over 35 years through multiple interrelated pathways that may act synergistically with age-related reproductive decline.

Infertility in women of advanced reproductive age represents an important clinical and demographic challenge. Epidemiological studies indicate that female fecundity declines markedly after the age of 35 as a consequence of progressive ovarian aging, reduced ovarian reserve, and increasing chromosomal abnormalities in oocytes [48]. Consequently, infertility becomes more prevalent in this age group, and reproductive outcomes are increasingly influenced by metabolic and systemic health factors. Despite the growing number of studies investigating the relationship between body composition and reproductive health, relatively limited research has examined the potential role of constitutional body morphology and somatotype characteristics in fertility disorders among women older than 35 years [31,41].

Theoretical foundations of somatotyping and body component composition. The concept of somatotype describes the constitutional morphology of the human body and reflects the relative development of adipose tissue, musculature, and skeletal structure. The classical somatotype theory was introduced by Sheldon and later refined through anthropometric approaches, most notably the Heath–Carter method, which quantifies the components of endomorphy, mesomorphy, and ectomorphy using standardized anthropometric measurements [21]. Body component composition refers to the proportional distribution of fat mass, muscle mass, and bone mass. These components may be assessed through various methods, including anthropometry, skinfold thickness measurements, and bioelectrical impedance analysis [21,65]. Changes in body composition occur throughout adulthood, and aging in women is frequently associated with gradual increases in adipose tissue and shifts toward more endomorphic body constitution patterns.

Somatotype and body mass component composition in women. Anthropometric and morphological studies have demonstrated that women with different somatotypes exhibit distinct distributions of body composition components, including variations in fat mass, muscle mass, and skeletal dimensions [21,24,31]. Research on adult female populations has shown that constitutional body types differ substantially in metabolic characteristics and patterns of adipose tissue accumulation. Somatotypes associated with greater body mass and higher adiposity often demonstrate increased fat mass relative to muscle and skeletal components [24,31]. These differences in body composition may have important implications for metabolic health and endocrine regulation.

Metabolic disorders, body mass, and female infertility. A large body of evidence has established strong associations between obesity, insulin resistance, dyslipidemia, and polycystic ovary syndrome (PCOS) and female infertility [42,61]. Excess adipose tissue, particularly visceral fat, contributes to reproductive dysfunction through multiple mechanisms, including endocrine imbalance, chronic low-grade inflammation, and disruption of hypothalamic-pituitary-ovarian axis regulation. Increased adiposity has been associated with impaired ovulation, decreased endometrial receptivity, lower implantation rates, and reduced effectiveness of ovarian stimulation in assisted reproductive technologies (ART) [34,42,60,61,65,68]. Both insufficient and excessive body mass have been associated with adverse reproductive outcomes, suggesting that body composition plays an important role in reproductive physiology.

Infertility in women of advanced reproductive age (35+). Female fertility declines significantly after the age of 35 as a result of decreasing ovarian reserve, deterioration in oocyte quality, and increasing rates of chromosomal abnormalities [48]. In addition to reproductive aging, metabolic disorders increasingly contribute to infertility in this age group. Conditions such as obesity, type 2 diabetes mellitus, and arterial hypertension occur more frequently with advancing age and may negatively influence reproductive outcomes [42,48,61].

Integration: possible links between somatotype, body composition, and fertility disorders in women older than 35 years. The available scientific evidence allows the development of a conceptual model linking constitutional morphology with reproductive health: somatotype → body composition → metabolic disturbances → fertility impairment. Somatotypes characterized by increased adiposity may predispose individuals to metabolic disorders such as insulin resistance and PCOS, both of which are established contributors to infertility [34,42, 60,61,65,68]. Morphological studies suggest that body types associated with greater fat accumulation may demonstrate metabolic profiles that adversely influence reproductive endocrine function. Age-related changes in body composition, including increasing adiposity and redistribution of body fat, may therefore represent an additional factor contributing to reduced fertility in women older than 35 years.

Somatotype, body composition, and fertility disorders in women aged 35 and older

Somatotype and body composition influence female reproductive function primarily through adiposity, fat distribution, and associated metabolic and endocrine disturbances rather than through morphological category per se. In women over 35 years, evidence directly focused on somatotype and infertility is limited; however, data on obesity indices, android versus gynoid fat distribution, and conditions such as PCOS provide a mechanistic framework linking endomorphic body habitus and central adiposity to ovulatory dysfunction, reduced fecundability, and treatment-resistant infertility. Age modifies these associations: body fat increasingly redistributes toward android patterns after 30, while the strength of association between relative fat mass and infertility appears to attenuate in older age groups, suggesting that age-related decline in ovarian reserve partly overshadows the contribution of body composition [8,24,28,58]. This suggests age-related ovarian reserve decline overshadows body composition effects, as antral follicle count (AFC) (reflecting primordial pool) remains similar despite lower AMH/inhibin B in obesity [28,58].

Concepts: somatotype and body composition

Heath-Carter somatotype quantifies endomorphy (fatness from skinfolds), mesomorphy (skeletal robustness from breadths/girths), and ectomorphy (linearity from height-weight ratio). Women average higher endomorphy (e.g., 4.1–6.0) and lower ectomorphy (2.0–2.5) than men due to greater subcutaneous fat at equivalent BMI, with dominant categories like mesomorph-endomorph or endomorphic-mesomorph [35].

Endomorphy formula:

$-0.7182+0.1451\times X-0.00068\times X^2+0.0000014\times X^3$,

where X = (sum of skinfolds) × (170.18/height in cm).

Mesomorphy:

0.858 × humerus breadth + 0.601 × femur breadth+ + 0.188 × corrected arm girth + 0.161 × corrected calf girth – 0.131 × height + 4.5 [4].

Somatotype in the Heath–Carter system is described by three components: endomorphy (relative fatness), mesomorphy (musculoskeletal robustness), and ectomorphy (linearity). Women in general tend to present higher endomorphy and lower ectomorphy compared with men, reflecting greater subcutaneous fat mass at similar BMIs, while mesomorphic and endomorphic combinations(«mesomorph-endomorph» or «endomorphic-mesomorph») dominate in female samples [28]. Somatotype categories correlate with but do not uniquely determine underlying fat and lean mass; composition-based analyses show considerable intra-category heterogeneity, underscoring the importance of direct body-composition measures such as BMI, waist circumference, relative fat mass (RFM), and body-shape indices.

Age-related changes in female body shape

Young adult women predominantly exhibit gynoid fat distribution (hips/thighs), shifting progressively toward android (central/abdominal) patterns after age 30, driven by hormonal changes (e.g., estrogen decline), menopause, and lifestyle factors such as reduced physical activity. Population data show that young adult women (in their 20s) are predominantly gynoid, with fat preferentially stored in the hips and thighs, whereas from the age of 30 onward, there is a progressive increase in transitional and android body types and a higher prevalence of overweight and obesity. This age-related «drift» increases WHR, overweight/obesity prevalence, endomorphy, and android/gynoid ratio, especially postmenopause, with a reported 42.1% rise in android fat [18]. This shift reflects both hormonal changes and lifestyle factors, with increased central (abdominal) fat and WHR in older women. Premenopausal women favor gynoid fat; postmenopause, android fat becomes dominant due to low estrogen, with android fat accelerating during the menopause transition [18,26]. Since visceral and android fat are more strongly linked to insulin resistance and systemic inflammation, age-related somatotype drift toward more endomorphic-android patterns enhances exposure to metabolic risk factors that can impair fertility [26].

Body composition and general female fertility

Multiple cohort and cross-sectional studies demonstrate that increased total adiposity is associated with reduced fecundability and higher infertility prevalence, even among eumenorrheic women [37]. Obesity (typically BMI ⩾30 kg/m^2) is linked to lower spontaneous conception rates, decreased cycle fecundity, and diminished success in fertility treatments such as controlled ovarian hyperstimulation and intrauterine insemination (COH-IUI), independent of age and other confounders [49]. Preconception cohorts show a linear decline in fecundability ratio with increasing BMI; BMI >29 kg/m^2 notably reduces the probability of natural conception [15]. Large preconception cohorts have shown a linear decline in fecundability ratios as BMI increases, with notable reductions in the probability of natural conception when BMI exceeds about 29 kg/m^2. Eumenorrheic overweight women have a higher risk of anovulatory infertility, and obesity independently worsens cycle fecundity [46]. Central adiposity appears to be more detrimental than overall adiposity. Indicators that reflect visceral fat accumulation – such as the WHR, the ratio of android to gynoid fat mass, RFM, and the ABSI that accounts for waist circumference in relation to height and BMI – often demonstrate stronger associations with female infertility than BMI alone [8]. These findings support the concept that an endomorphic somatotype with prominent android fat deposition is more closely linked to fertility disorders than peripheral or gynoid fat accumulation. WHR is causally linked to anovulatory infertility (multiplicative random effects inverse-variance weighting (MR IVW) OR=2.099, 95% CI: 1.370–3.216; BMI-adjusted OR=1.582), with WHR>0.85 associated with a higher risk [67]. Subsequently, ABSI and BRI are anthropometric indices that characterize body fat distribution and central adiposity more precisely than BMI, as they incorporate waist circumference and height to better reflect abdominal and visceral fat accumulation. Higher values of these indices indicate greater central adiposity associated with metabolic and reproductive risks, and epidemiological studies show that they may predict infertility more accurately than BMI, with women in the highest quartile of BRI (Q4) demonstrating significantly increased odds of infertility (OR=2.56), the strongest association among indicators such as WC and the lipid accumulation product (LAP) [70]. National Health and Nutrition Examination Survey (NHANES) analyses confirm that RFM strongly predicts female infertility independently, with stronger odds ratios than BMI and WC in reproductive-age women [8]. RFM is a newer anthropometric index designed to estimate body fat percentage using height and waist circumference, and several NHANES-based analyses have evaluated its association with female infertility. Cross-sectional analyses of women aged roughly 20–44 years show that higher RFM is independently associated with an increased odd of a self-reported history of infertility after adjustment for demographic and lifestyle covariates. Dose-response modelling with restricted cubic splines reveals a generally positive, though sometimes non-linear, relationship between RFM and infertility risk, with the lowest risk observed in moderate RFM ranges (for example, around 36–41 in some analyses) [34]. Subgroup analyses suggest that the association between high RFM and infertility is strongest in younger women, particularly those under 35 years of age, whereas the magnitude of association weakens in women beyond this age. Despite the attenuation, high RFM remains an adverse factor across the reproductive age span, indicating that excess central fat is a relevant risk factor for infertility in both younger and older women, albeit with diminishing relative impact as ovarian aging progresses [8].

Somatotype and PCOS-related fertility disorders

PCOS is a leading cause of anovulatory infertility and is strongly associated with obesity, central adiposity, and metabolic syndrome [17]. Recent somatotype analyses using the Heath–Carter method have demonstrated that women with PCOS exhibit significantly higher endomorphy and mesomorphy and significantly lower ectomorphy relative to age-matched controls, with large effect sizes in endomorphy and ectomorphy components. Women with PCOS exhibit higher endomorphy (7.21±0.92 vs. 5.48±0.86; p<0.001, Cohen's d=2.07), higher mesomorphy (4.83±0.93 vs. 4.23±0.87; p<0.001, d=0.74), and lower ectomorphy (2.02±0.58 vs. 2.78±0.64; p<0.001, d=-1.50) [17]. Lean women with PCOS have also been reported to show a somatotype of 4.96–4.38–3.00, compared with 4.17–4.59–2.89 in controls [72]. These women tend to cluster into somatotype categories characterized by high relative fatness and robust musculature, such as endomorphic-mesomorphs, distinguishing them from the leaner, more linear somatotypes seen in healthy controls [72]. PCOS is a major cause of anovulatory infertility and is commonly associated with obesity, central adiposity, and metabolic abnormalities, including insulin resistance and dyslipidemia [17,72]. The combination of endomorphic somatotype, increased central fat, and insulin resistance in PCOS amplifies the risk of chronic anovulation, luteal phase defects, and subfertility. In women older than 35 years, the coexistence of ovarian aging and PCOS-related metabolic dysfunction may further reduce fecundability and may warrant more intensive lifestyle and pharmacologic approaches to restore ovulation and improve treatment outcomes [55,56]. Women with PCOS demonstrated significantly higher body weight, body surface area, and mass-height index. They also exhibited greater hip circumference, chest girth, and shoulder girth. Skinfold thickness analysis revealed pronounced accumulation of adipose tissue in women with PCOS, particularly in the shoulder, breast, and trunk regions. Women with PCOS were predominantly characterized by mesomorphic (54.0%) and endomesomorphic (9.1%) somatotype components and showed increased fat mass according to Mateiko's method [41]. Another study analyzed the relationship between somatometric body parameters, body composition, and endogenous hormone levels in 1,005 healthy adult women. The results demonstrated that even after completion of growth, significant associations persist between body morphology and endocrine regulation. Linear body dimensions and skeletal robustness were positively related to hormones involved in growth processes, while body fat development was closely linked to sex hormone activity and metabolic regulation. These findings indicate that body size, skeletal structure, and fat distribution may serve as anthropometric markers of endocrine function and reproductive capacity in women [27].

Evidence specific to women aged 35 and older

Few studies restrict analysis exclusively to women over 35 when examining somatotype or body composition as determinants of infertility, so the evidence must be interpreted from age-stratified analyses and broader reproductive-age cohorts. NHANES-based studies of relative fat mass and the limited somatotype literature do not provide analyses restricted exclusively to women older than 35 years; instead, they rely on age-stratified data suggesting weaker associations between RFM and infertility in women aged 35 years and older than in younger women, while odds ratios remaining above 1 suggest an additive contribution of adverse body composition on top of ovarian aging [8,17]. In RFM-based studies using NHANES data, interaction analyses show that while high RFM significantly increases infertility odds in women under 35, the association is less pronounced or statistically weaker in women aged 35–44 years, implying that chronological age and related ovarian factors contribute more strongly to infertility in this group [8,34]. Nonetheless, point estimates generally remain above unity for older women with high RFM, suggesting that unfavorable body composition continues to add risk on top of age-related decline. Few studies focus solely on women older than 35 years for somatotype- or body composition-related infertility; evidence from broader cohorts and stratified analyses suggests that android adiposity and obesity become more prevalent after age 30, shifting body composition toward more endomorphic-central patterns and potentially heightening the risk of ovulatory and implantation-related fertility disorders in women over 35 years despite limited age-specific epidemiologic data [26,43]. Age-related changes in body shape described in anthropometric surveys reveal that after age 30, there is both an increase in the prevalence of android body type and a higher proportion of overweight and obese women, reflecting a trend toward more endomorphic and centrally obese somatotypes [26,43,51]. Women older than 35 years with increased endomorphy and central adiposity may therefore represent a high-risk subgroup for ovulatory and implantation-related disorders; in addition, excess body weight is associated with lower clinical pregnancy and live birth rates and higher miscarriage risk in assisted reproduction settings [22,62]. When combined with the known negative effects of android adiposity and obesity on fecundability and ART outcomes, these patterns support the clinical view that women over 35 years with pronounced endomorphy and central fat accumulation represent a high-risk subgroup for ovulatory and implantation-related fertility disorders, even if the available epidemiologic data are not exclusively focused on this age band. Central obesity, reflected by elevated WHR and WC, is associated with reduced fecundity, and obesity is associated with a substantially increased risk of infertility compared with normal weight [42]. Obesity may impair ovulation, oocyte and embryo quality, and endometrial receptivity and implantation [40]. In ART settings, women with obesity often require higher gonadotropin doses and may have fewer retrieved oocytes and lower live birth rates [22,62]. Similar conclusions have been reported in studies showing that higher BMI worsens *in vitro* fertilization (IVF) / intracytoplasmic sperm injection (ICSI) outcomes in older women, with central adiposity remaining particularly relevant despite confounding by age-related ovarian decline [50].

Possible mechanistic links between somatotype, body composition, and fertility

The adverse impact of endomorphic, centrally obese body types on fertility is mediated through several mechanisms. Visceral adipose tissue secretes pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), contributing to chronic low-grade inflammation that impairs hypothalamic-pituitary-ovarian axis function, oocyte quality, and endometrial receptivity [40]. Central obesity also promotes insulin resistance and compensatory hyperinsulinemia, which can exacerbate hyperandrogenism, disrupt folliculogenesis, and lead to chronic anovulation, particularly in PCOS phenotypes. Obesity-related inflammation affects follicular fluid composition, oocyte gene expression, and implantation, including impaired endometrial stromal cell decidualization [19,32]. Central obesity and insulin resistance may increase ovarian androgen production through insulin-sensitive steroidogenesis, reduced sex hormone-binding globulin (SHBG), and increased insulin-like growth factor 1 (IGF-1) activity, thereby contributing to arrested folliculogenesis and anovulation in PCOS [52]. Excess adiposity alters SHBG levels and peripheral aromatization, resulting in disturbed estrogen and androgen balance that may affect follicular recruitment, luteal function, and early pregnancy maintenance [52]. In addition, obesity is associated with altered leptin and adiponectin levels, which have direct and indirect effects on gonadotropin secretion, ovarian steroidogenesis, and implantation, while hepatic steatosis and dyslipidemia may compound endocrine disturbances. In older women, these mechanisms operate on a background of diminished ovarian reserve and oocyte competence, potentially making the reproductive system more vulnerable to metabolic insult while simultaneously limiting the degree to which weight optimization can fully restore fertility potential [59].

Impact on assisted reproductive technology outcome

In women undergoing COH-IUI or *in vitro* fertilization (IVF), body composition appears to influence treatment success beyond chronological age. A prospective study in women with unexplained infertility undergoing COH-IUI found that fat mass >16.65 kg predicted treatment failure (OR 0.4, sensitivity 61.8%, specificity 70.2%) and was more informative than BMI or WHR [24]. A prospective observational study in women with unexplained infertility treated with COH-IUI found that body composition parameters, including higher fat mass, were associated with reduced cycle fecundity, although the cohort was not age-restricted to women over 35 [24]. In IVF settings, obesity has been associated with higher gonadotropin requirements, lower peak estradiol levels, reduced oocyte yield, and lower implantation and live birth rates, with these effects persisting after adjustment for protocol-related factors [1,16]. Every 5-unit increase in BMI has been associated with approximately 5–7% lower clinical pregnancy and live birth rates and about 9% higher miscarriage risk, together with adverse effects on oocyte quality, embryo development, and endometrial receptivity [1]. Given that many ART patients are 35 years or older, these findings are clinically relevant: an endomorphic, centrally obese somatotype may compound age-related decline by impairing ovarian response and endometrial receptivity. However, the relative contribution of somatotype and body composition to ART outcomes, compared with ovarian reserve markers such as AMH and antral follicle count, remains insufficiently quantified in older women [60,69,73]. Identifying an endomorphic, android pattern can help stratify metabolic and reproductive risk, prompting early lifestyle interventions targeting weight reduction, visceral fat loss, and improvement in insulin sensitivity through diet, physical activity, and, in selected cases, pharmacotherapy [30,39]. In patients with PCOS older than 35 years, intensive management of adiposity and metabolic abnormalities may partially restore ovulatory cycles and enhance responsiveness to ovulation induction or IVF, although the absolute benefit remains constrained by age-related ovarian factors [5,14,23,25,63].

Research gaps and future directions. There is a scarcity of prospective, age-stratified studies directly linking somatotype categories (endomorph-mesomorph-ectomorph profiles) to specific infertility diagnoses and treatment outcomes in women aged 35 years and older. Limited prospective studies have linked Heath-Carter somatotype to infertility and treatment-related outcomes in women aged ⩾35 years; most PCOS and broader reproductive-age cohorts lack adequate age stratification [17]. There are no sufficiently characterized datasets focused exclusively on women aged ⩾35 years that relate somatotype profiles directly to infertility diagnoses and outcomes; available broader datasets suggest that endomorphy increases with age, but not specifically in infertility-defined populations [35]. Standardized somatotype assessment remains scarce in late-reproductive-age infertility research [2]. Because AMH and AFC are important predictors of oocyte yield and quality in women aged ⩾35 years, integration of body composition assessment with ovarian reserve markers may help disentangle the relative contributions of adiposity and constitutional morphology to IVF success [11,29,45]. Longitudinal interventional studies focusing on women over 35 with endomorphic, centrally obese somatotypes are needed to quantify the impact of targeted weight and waist reduction on spontaneous pregnancy rates, ovulatory function, and ART success, while accounting for ovarian reserve and comorbidities. In addition, exploration of gene-environment interactions influencing somatotype evolution across the reproductive lifespan could clarify why some women maintain more favorable (less endomorphic, more gynoid) body types despite aging and whether these trajectories are associated with preserved fertility potential [42]. Although numerous studies have examined the relationship between body composition and reproductive health, direct investigations of the association between somatotype and fertility outcomes in women older than 35 years remain limited [31,41]. Future research should therefore include:

• combined assessment of somatotype and body component composition;

• stratification of advanced reproductive age groups (for example, 35–39 and 40–44 years);

• evaluation of reproductive indicators such as ovulatory function, oocyte quality, and outcomes of ART. One possible working hypothesis is that endomorphic somatotypes characterized by higher adipose tissue accumulation may be associated with increased rates of anovulatory infertility and reduced effectiveness of fertility treatment in women older than 35 years [34,42,60,61,65,68].

Conclusions

The available literature suggests a complex interaction between constitutional body morphology, body composition, metabolic status, and reproductive function. These relationships may be summarized as: somatotype → body composition → metabolic risk → fertility outcomes in women aged 35 and older. These findings highlight the clinical importance of evaluating not only BMI but also body composition and constitutional morphology when assessing reproductive health in women of advanced reproductive age. Existing evidence indicates that unfavorable body composition – particularly high overall adiposity and central, android fat distribution – adversely affects female fertility through metabolic and endocrine mechanisms, and this pattern corresponds closely to more endomorphic somatotypes. In women over 35, the same anthropometric risk factors continue to play a role but interact with and are partly overshadowed by age-related declines in ovarian reserve, leading to attenuated but persistent associations between indices like RFM and infertility. Clinically, a comprehensive assessment of body composition and somatotype, alongside ovarian reserve testing, is important for risk stratification, counselling, and designing individualized lifestyle and treatment strategies for older women with fertility disorders.

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