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ORIGINAL RESEARCH

Linking Neonatal Birth Weight with AMH Levels in Umbilical Cord and Maternal Blood: A Prospective Study

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Purpose: We aimed to study the correlation between umbilical cord anti-mullerian hormone (AMH) levels and neonatal birth weight as well as the correlation between maternal and neonatal AMH levels.

Patients and Methods: A prospective observational single center cohort study, conducted at the Carmel Medical Center delivery room from March 2022 to December 2024. Two hundred and one patients and their female newborns were recruited and had umbilical cord and maternal blood AMH levels obtained. Small for gestational age (SGA) was defined as birthweight up to the 10th percentile according to the Hadlock equations. Associations with AMH levels were assessed using linear regression with logarithmic transformation of AMH. Variables significant in univariable analyses ($P < 0.05$) were included in multivariable models. Analyses were performed using IBM SPSS Statistics version 26.

Results: In the SGA group 42 women and their female newborns were included with a mean (SD) birth weight of 2708 (± 218) g, while the appropriate for gestational age (AGA) group included 159 women and their female newborns, with a mean (SD) birth weight of 3362 (±322) g. Median (interquartile range (IQR)) newborn AMH levels were found to be 0.18 (0.07–0.57) in the SGA group, and 0.15 (0.06–0.68) in the AGA group. No differences were found between neonatal AMH levels of the SGA and AGA group (p=0.78). A correlation was found between maternal and neonatal AMH levels (r=0.35, p=0.001), that remained significant in an adjusted multivariate model that included adjustment to age, gravidity, parity, maternal body mass index (BMI), paternal BMI, smoking status and comorbidities.

Conclusion: Female newborns born SGA were found to have comparable levels of AMH as female AGA newborns. A correlation between maternal and neonatal AMH levels was observed, implying AMH genetic predisposition of ovarian reserve, yet more research is needed to conclude clinical applications of this finding.

Keywords: ovarian reserve, neonates, anti-mullerian hormone, small for gestation age, birth weight

Introduction

The anti-Mullerian hormone (AMH) is a glycoprotein belonging to the transforming growth factor-β (TGFβ) family and serves as a key marker for ovarian follicle development and primordial follicle reserve.[1] AMH is produced in the gonads and plays an essential role in fetal reproductive system maturation. In females, AMH is secreted by granulosa cells beginning around the 36th week of gestation, by which time the Mullerian ducts have already become insensitive its effects.[2] In healthy females, AMH levels are low at birth, gradually increase during childhood until puberty, remain relatively stable until through the third decade of life, and subsequently decline until becoming undetectable at menopause.[3] Although AMH measurement is most commonly performed during reproductive age, there is clinical

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interest in AMH assessment during infancy and childhood. In pediatric populations, AMH contributes to the diagnosis of disorders of sexual development, premature ovarian insufficiency, and granulosa cell tumors. In addition, AMH is used as a marker of fertility potential in young girls undergoing gonadotoxic cancer treatments and plays a role in planning fertility preservation strategies and national fertility-sparing programs.[4–6]

Despite its growing clinical relevance, data regarding AMH levels during early life remain limited. Emerging evidence suggests that maternal conditions such as polycystic ovary syndrome (PCOS), metabolic disorders, and cardiovascular disease may influence the intrauterine hormonal environment, potentially affecting fetal endocrine development, including AMH levels.[7,8] In this context, the Barker hypothesis proposes that birth weight reflects not only genetic factors but also intrauterine environmental exposures, which may induce long-term changes in gene expression and increase susceptibility to adult diseases, including reproductive dysfunction.[7] Accordingly, assessment of AMH levels in umbilical cord blood may provide early insight into initial ovarian reserve status. Understanding whether ovarian reserve is compromised from birth is clinically relevant, as it could facilitate early identification of individuals at risk for reduced reproductive potential and enable tailored counseling and follow-up.

To date, only a limited number of studies have examined AMH concentrations in cord blood. Reported AMH levels range from 0.24 to 9.16 ng/mL in female newborns and from 23.23 to 44.31 ng/mL in male newborns.[9–12] Furthermore, newborns of women with PCOS were found to have higher AMH levels compared to newborns of women without PCOS, suggesting that maternal hyperandrogenism and endocrine alterations may influence fetal hormonal and metabolic programming and contribute to PCOS-like phenotypes in female offspring.[11]

The relationship between fetal growth restriction and subsequent ovarian function remains incompletely understood. One study has suggested that women born small for gestational age (SGA) may have smaller ovarian and uterine volumes,[13] while another report found no significant difference.[14] In adolescence, girls born SGA have been found to exhibit lower ovulation rates and higher prevalence of anovulation compared with those born appropriate for gestational age (AGA).[15] However, whether these differences originate during fetal life or emerge later remains unclear.

The present study focused exclusively on female neonates, as AMH in females reflects ovarian follicular development and serves as a surrogate marker of ovarian reserve—the primary outcome of interest. In contrast, in male neonates AMH is produced by Sertoli cells and reflects testicular function rather than gonadal reserve, rendering direct comparisons biologically inappropriate. Consequently, the findings of this study are specific to female offspring and cannot be generalized to male neonates.

Given the limited evidence regarding the impact of birth weight on ovarian reserve, this study aimed to examine the association between AMH levels in female newborns and birth weight status, as well as the correlation between maternal and neonatal AMH levels. By exploring these relationships, we sought to clarify whether intrauterine growth and maternal endocrine milieu influence early ovarian reserve.

Materials and Methods

This was a prospective observational cohort study conducted between March 2022 and December 2024 at the delivery room of Carmel Medical Center, a tertiary teaching hospital affiliated with the Technion–Israel Institute of Technology. The center has an annual delivery rate of approximately 3,800 births and provides obstetric care for both low- and high-risk pregnancies. The study was approved by the Carmel Medical Center Institutional Review Board on August 24, 2021 (protocol number CMC-0080-21) and was conducted in accordance with the Declaration of Helsinki. The study is reported in compliance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies.

Pregnant women at term (37–42 gestational weeks) with a singleton female fetus expected to give birth within three days were recruited consecutively upon admission to the delivery room of Carmel Medical Center. This included women in active labor as well as women admitted for scheduled induction of labor or planned cesarean delivery. Neonatal sex was determined postnatally at birth based on clinical examination. Recruitment was performed prospectively at the time of admission, and all eligible women who agreed to participate and provided informed consent were included. Excluded were individuals over 40 years old, women with a diagnosis of PCOS, women carrying a multi-gestational pregnancy, preterm delivery or a male-sex fetus. Informed consent was obtained prospectively from all participants prior to

enrollment. Recruitment and consent procedures were conducted by the principal investigator and co-investigators, who provided verbal and written explanations of the study in the participants' native language. Each participant was assigned a unique study identification number to ensure anonymity. Data were collected and stored in a coded manner, with access restricted to the principal investigator only. All personal identifiers were removed, and confidentiality was maintained in accordance with institutional and ethical guidelines.

Maternal blood samples were obtained during pregnancy prior to delivery, and umbilical cord blood samples were collected up to 30 minutes after birth. Blood samples were processed according to standardized laboratory procedures and centrifuged shortly after collection, and serum was stored under appropriate conditions until analysis.

AMH concentrations in both maternal and cord blood samples were measured using the Elecsys® AMH Plus electrochemiluminescence immunoassay (Roche Diagnostics, USA), according to the manufacturer's instructions. The assay demonstrates high analytical sensitivity and specificity for human AMH, with reported intra- and inter-assay coefficients of variation below 4%, indicating good reproducibility. All analyses were performed in a single laboratory to minimize inter-assay variability.

At the time of recruitment, the following demographic and clinical data were collected and considered as potential confounders: maternal age, maternal and paternal body mass index (BMI), ethnicity, history of systemic disease and chronic medications, smoking status, age at menarche, parity, and gestational age. Further data were extracted from patients' charts following delivery including newborn weight.

Clinical and demographic data were collected prospectively using a structured, study-specific data collection form developed for this study. The form included predefined variables related to maternal characteristics, pregnancy course, delivery details, and neonatal outcomes. To ensure consistency and reliability, data collection was performed exclusively by the primary investigator using uniform definitions for all variables. Laboratory measurements were performed using validated commercial assays, as described above.

The association between AMH levels, fetal weight, maternal AMH and feto-maternal characteristics were analyzed after the completion of data gathering.

SGA was defined as a birth weight at or below the 10th percentile for gestational age, based on the Hadlock fetal growth reference equations, which are widely used in obstetric practice. Neonates with birth weight above the 10th percentile were classified as AGA.

Missing data were infrequent due to the prospective study design. Analyses were performed using a complete-case approach, and participants with missing data for a given variable were excluded only from analyses involving that specific variable.

Outcomes

The primary outcome was the association between neonatal AMH levels and neonatal birth weight. Secondary outcomes included the association between maternal and neonatal AMH levels and descriptive data on normal female neonatal AMH concentrations.

Statistical Analysis

The primary outcome of the study was neonatal AMH concentration. The primary exposure variable was birth weight status, categorized as SGA versus AGA. In addition, maternal AMH levels were examined as an independent variable in correlation analyses with neonatal AMH. The distribution of AMH levels was assessed using visual inspection of histograms. As AMH values were not normally distributed, AMH concentrations were log-transformed prior to statistical analyses. Associations between exposure variables and neonatal AMH levels were first evaluated using univariable linear regression models. Variables associated with neonatal AMH in univariable analyses were subsequently included in multivariable linear regression models to estimate adjusted associations.

Continuous variables are presented as means and standard deviation (SD) or as medians and interquartile range (IQR). Categorical variables are presented as percentages. Correlation coefficients with 95% confidence intervals (CI) are presented for unadjusted associations, while multivariable analyses are reported as β coefficients with 95% CIs.

The sample size required to detect a difference of 0.5 with 80% power, with an enrollment ratio of 1:4 was calculated to be 195 (39 in the SGA group and 156 in the AGA group). A 1:4 enrollment ratio between SGA and AGA newborns was selected to ensure adequate representation of the SGA group for comparative analyses, given the expected prevalence of SGA births of approximately 10% in the general obstetric population, while maintaining feasibility and statistical power in this prospective study. Although the sample size calculation indicated a minimum of 195 participants, a total of 201 eligible mother–newborn dyads were ultimately enrolled and included in the final analysis.

No formal correction for multiple comparisons was applied, as the analyses were hypothesis-driven and focused on a limited number of predefined outcomes.

All Analyses Were Performed Using IBM Statistics Version 26 (SPSS)

Results

During the study period, 201 women were recruited, including 42 mother–newborn pairs in the neonatal SGA group and 159 in the neonatal AGA group. Baseline characteristics of the study population according to neonatal birth weight category are presented in Table 1. Mean (SD) maternal BMI was lower in the SGA group compared with the AGA group (26.1 ± 5.1 vs. 28.2 ± 5.2, $P = 0.02$), while paternal BMI did not differ between groups ($P = 0.99$).

Table 1 Characteristics of Included Women and Female Newborns

		SGA (n=42)	AGA (n=159)	P value
Age, years – mean (SD)		31.9 (4.5)	32.1 (4.8)	0.79
Gestational age at birth, weeks – mean (SD)		39.4 (1.0)	39.5 (1.1)	0.77
Gravidity, n – median (IQR)		2 (1–3)	2 (1–3)	0.16
Parity, n – median (IQR)		1 (0–2)	1 (0–2)	0.32
BMI (mother), mean (SD)		26.1 (5.1)	28.2 (5.2)	0.02
BMI (father), mean (SD)		26.4 (4.3)	26.4 (4.2)	0.99
Maternal birth weight, grams – mean (SD)		3110.7 (577.2)	3233.9 (548.2)	0.29
Maternal menarche age, years – mean (SD)		13.1 (1.6)	12.8 (1.5)	0.52
Comorbidities	Hypothyroidism, n (%)	4 (10)	8 (5)	0.25
	Anemia, n (%)	2 (5)	1 (0.6)	
	Asthma, n (%)	2 (5)	4 (3)	
	Endometriosis (%)	0 (0)	3 (2)	
	Other, n (%)	2 (5)	9 (6)	
Gestational diabetes	A1	2 (5)	13 (8)	0.73
	A2	1 (2)	4 (3)	
Past smoking status positive n (%)		7 (17)	34 (21)	0.5
Maternal AMH level, median (IQR)		0.58 (0.33–1.42)	0.66 (0.31–1.07)	0.90
Female newborn birth weight, grams – mean (SD)		2708.2 (218.1)	3362.4 (322.5)	<0.001
Newborn AMH level, median (IQR)		0.18 (0.07–0.57)	0.15 (0.06–0.68)	0.78

Notes: Data are presented as mean (standard deviation), median (interquartile range), or number (percentage), as appropriate.

Abbreviations: SGA indicates small for gestational age; AGA, appropriate for gestational age; BMI, body mass index.

In the SGA group, mean (SD) neonatal birth weight was 2708 ± 218 g, compared with 3362 ± 322 g in the AGA group ($P< 0.001$). Median (IQR) neonatal AMH levels were 0.18 (0.07–0.57) in the SGA group and 0.15 (0.06–0.68) in the AGA group, with no significant difference between groups ($P = 0.78$). Median (IQR) maternal AMH levels were 0.58 (0.33–1.42) in the SGA group and 0.66 (0.31–1.07) in the AGA group ($P = 0.78$). Other than neonatal birth weight and maternal BMI, no significant differences were observed in demographic or clinical characteristics between groups (Table 1).

Primary Outcome

Neonatal birth weight was not associated with neonatal AMH levels in univariable analysis (β=−0.002, p=0.975). The estimated effect size was negligible (B = -3.94×10^{-6}, 95% CI −0.000 to 0.000). Consistently, no significant difference in neonatal AMH levels was observed between SGA and AGA female neonates (β = 0.021, p = 0.782). The estimated effect size was small and not statistically significant (B = 0.034, 95% CI −0.209 to 0.277).

Secondary Outcomes

Maternal AMH levels were not associated with neonatal birth weight (β = 0.036, p = 0.620). In contrast, a moderate positive association was observed between maternal and neonatal AMH levels (β = 0.243, p = 0.001). The unstandardized regression coefficient indicated a positive association (B = 0.348, 95% CI 0.139–0.556). This association remained statistically significant in multivariable linear regression models with log-transformed neonatal AMH as the dependent variable, after adjustment for maternal age, gravidity, parity, maternal and paternal BMI, smoking status, and comorbidities (Figure 1). Similar associations were observed when analyses were stratified by neonatal birth weight category. The scatterplot indicated no association between neonatal AMH levels and maternal age (Figure 2). Consistently, the boxplot demonstrated no significant differences in neonatal AMH levels across parity groups (Figure 3). Neonatal AMH levels demonstrated a right-skewed distribution, with approximately 70% of neonates having AMH concentrations ≤0.5. The overall median neonatal AMH level was 0.15 (IQR 0.07–0.65). The distribution of neonatal AMH levels is illustrated in Figure 4.

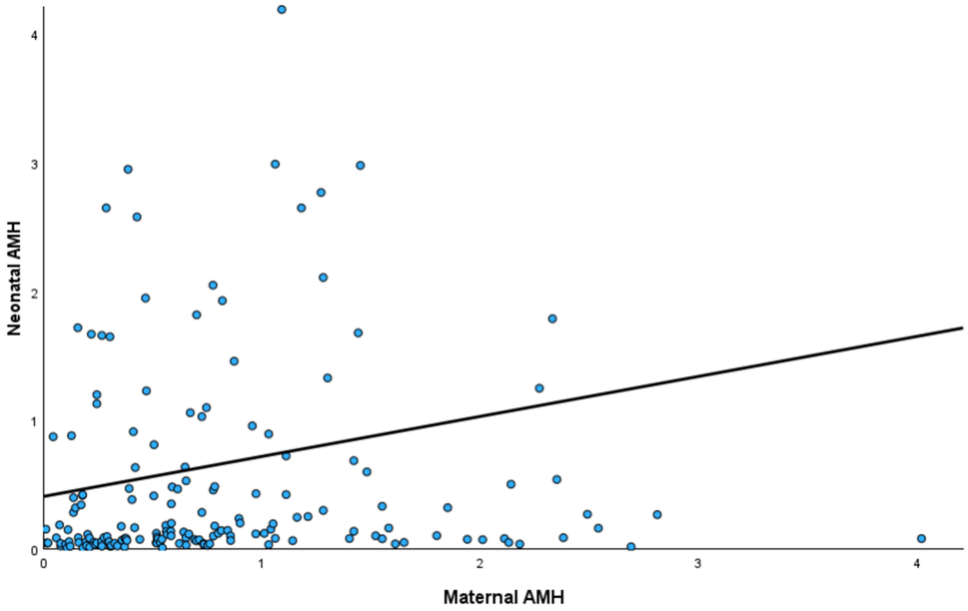

Figure 1 Correlation between maternal and neonatal AMH levels. Graphical representation demonstrating a positive association between maternal and neonatal AMH concentrations.

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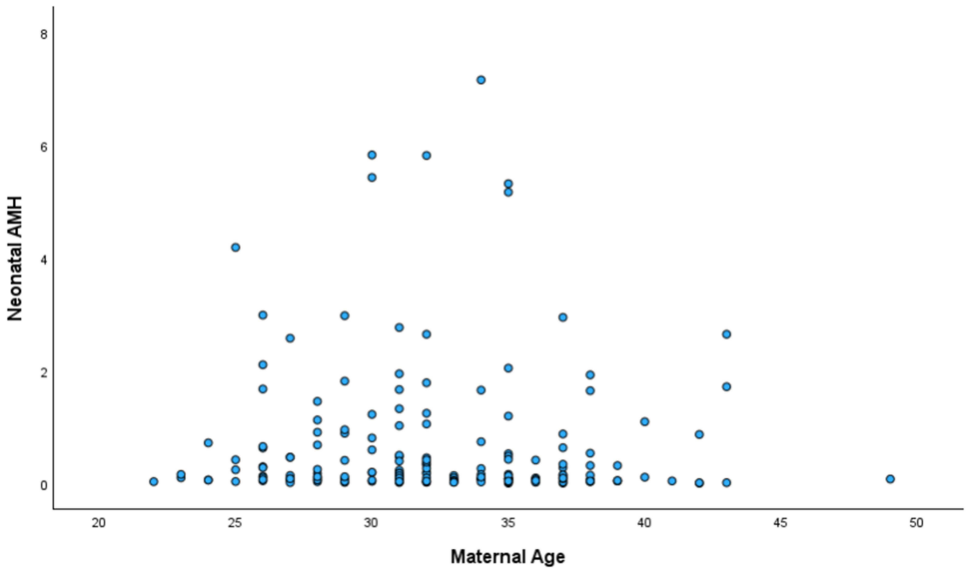

Figure 2 Relationship between neonatal AMH levels and maternal age. Graphical exploration demonstrated no meaningful association between maternal age and neonatal AMH concentrations.

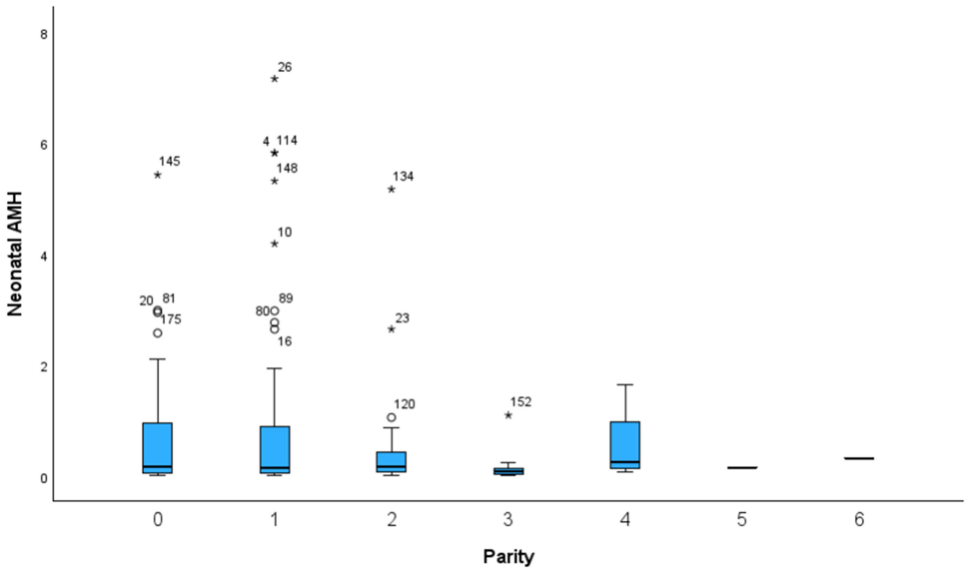

Figure 3 Distribution of neonatal AMH levels according to maternal parity. Boxplot showing neonatal AMH concentrations stratified by maternal parity. Neonatal AMH levels were comparable across parity groups. Circles indicate mild outliers and asterisks indicate extreme outliers. Numbers correspond to individual observations.

Discussion

The present study evaluated whether fetal growth status is associated with ovarian reserve at birth and explored the relationship between maternal and neonatal AMH levels. Two principal findings emerged. First, neonatal AMH concentrations were not associated with birth weight and did not differ between SGA and AGA female newborns. Second, maternal AMH levels were positively correlated with neonatal AMH levels, and this association remained significant after multivariable adjustment.

Neonatal AMH and Fetal Growth

SGA reflects impaired fetal growth and is associated with increased perinatal morbidity as well as long-term metabolic and cardiovascular consequences.[16] However, SGA represents a heterogeneous condition influenced by placental,

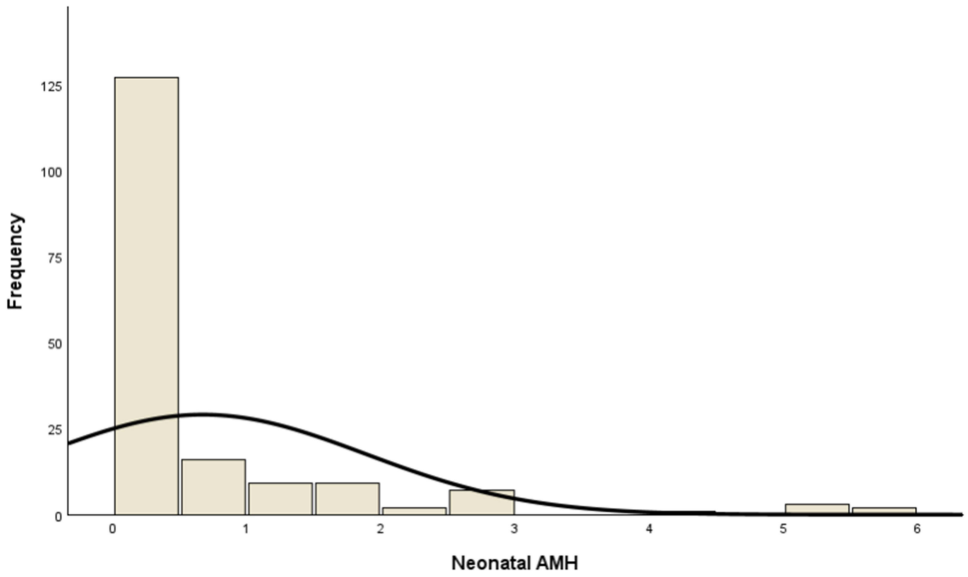

Figure 4 Distribution of female neonatal AMH concentrations. Neonatal AMH levels demonstrated a right-skewed distribution, with the majority of values concentrated at lower concentrations.

maternal, genetic, infectious, and structural factors, all of which may act as potential confounders.[17] Although mode of delivery has been shown to influence certain neonatal hormonal and stress-related parameters,[18] evidence regarding its effect on neonatal anti-Müllerian hormone levels is lacking.

Impaired intrauterine growth and low birth weight have been linked to adverse health outcomes later in life, including metabolic disorders and reproductive dysfunction. Several studies have suggested that low birth weight may be associated with earlier reproductive aging, including earlier menopause.[16,19–23] These observations align with the Barker hypothesis, which proposes that intrauterine conditions may influence long-term physiologic trajectories.[16]

Despite these theoretical considerations, our findings suggest that fetal growth restriction does not translate into measurable differences in ovarian reserve at birth, as reflected by neonatal AMH levels. These results are consistent with prior studies demonstrating no association between birth weight and AMH concentrations in childhood or adulthood. Kerkhof et al found that neither preterm birth nor SGA birth affected AMH levels in young adult women.[24] Similarly, Lima et al reported no significant differences in AMH levels among women aged 34–35 years who were born SGA, AGA, or large for gestational age.[25]

AMH has also been evaluated in prepubertal populations. In a cohort of girls aged 3–10 years, AMH concentrations were similar between those born SGA and controls born AGA, suggesting that the follicular pool is not compromised by SGA birth.[26] In contrast, other studies have suggested potential long-term reproductive consequences associated with SGA birth. Ibáñez et al reported reduced ovulation rates and increased anovulation among adolescent girls born SGA.[15] However, these studies did not assess neonatal AMH levels, making it difficult to determine whether the observed reproductive differences reflect intrinsic ovarian alterations or postnatal influences.

The absence of differences in neonatal AMH levels between SGA and AGA newborns may be explained by the developmental physiology of AMH. AMH is secreted by granulosa cells of early-growing follicles, and its expression begins relatively late in gestation.[4] Consequently, factors affecting fetal growth may not directly influence folliculogenesis or AMH secretion at birth.

Collectively, these findings suggest that being born SGA does not necessarily indicate compromised ovarian reserve at birth. While this does not exclude the possibility of long-term reproductive differences, it may provide reassurance regarding immediate ovarian function.

Maternal and Neonatal AMH Association

A significant positive correlation was observed between maternal and neonatal AMH levels. This finding persisted after adjustment for potential confounders, suggesting a biologically meaningful relationship.

Peptide hormones such as AMH are not believed to cross the placenta readily,[27] indicating that the observed association is unlikely to reflect direct hormonal transfer. Instead, shared genetic or intrauterine regulatory mechanisms may explain this relationship. Genetic factors are known to play a substantial role in determining AMH concentrations, with variants in the AMH and AMHR2 genes associated with circulating AMH levels and reproductive lifespan traits.[28] These heritable influences may contribute to intergenerational similarities in AMH concentrations.

In addition to genetic determinants, the intrauterine environment may modulate neonatal ovarian development. Establishment of the primordial follicle pool occurs during fetal life, and maternal metabolic, hormonal, and lifestyle factors may influence this process. Prior studies have demonstrated associations between maternal characteristics and offspring AMH levels, supporting the concept that prenatal exposures may shape early ovarian reserve markers.[24–26,29]

Studies examining pregnancies complicated by PCOS have reported elevated neonatal AMH levels, suggesting that maternal endocrine milieu may influence fetal ovarian physiology.[12,27] In contrast, the present study excluded women with PCOS and focused specifically on fetal growth parameters. Our findings therefore suggest that, in the absence of overt maternal endocrine pathology, maternal hormonal characteristics may have a greater influence on neonatal AMH concentrations than birth weight alone.

Interpretation of this association should consider the timing of maternal AMH measurement. Although AMH is relatively stable compared with other reproductive hormones, pregnancy-related physiological changes may influence circulating levels.

Clinical Interpretation of Neonatal AMH

While AMH is widely used as a marker of ovarian reserve in adult populations, its role in neonatal and pediatric settings remains incompletely defined.

Interpretation of the observed association between maternal and neonatal AMH levels should also consider the timing of maternal AMH measurement. In the present study, maternal AMH was assessed during pregnancy, prior to delivery. Although AMH is considered relatively stable compared with other reproductive hormones, pregnancy-related physiological changes may influence circulating concentrations and should be acknowledged when interpreting maternal–offspring associations.

Moreover, clinical interpretation of AMH measurements is complicated by inter-assay variability, lack of standardized thresholds, and population-specific differences influenced by age, ethnicity, and metabolic factors.[30] These considerations warrant cautious interpretation of both maternal and neonatal AMH concentrations.

Strengths and Limitations

A major strength of this study is its prospective design, performed in a single center with strict inclusion criteria ensuring a homogeneous study population. Additionally, the use of standardized AMH assays in a single laboratory minimizes inter-assay variability and enhances the reliability of the findings.

However, several limitations must be acknowledged. Although the sample size was sufficient to detect moderate differences in AMH levels, it may have been underpowered to identify subtle effects of low birth weight, particularly in subgroup analyses. This study only assessed AMH levels at birth, and long-term follow-up is needed to determine whether differences in ovarian reserve that might emerge later in life. While we controlled for multiple confounding factors, unmeasured variables such as maternal nutrition and placental function could still influence the results. No data was available about the possible causes for fetal SGA such as placental abnormalities or genetic predisposition. Lastly, as AMH levels are known to decrease during pregnancy, it is possible that the specific timing of maternal AMH measurement is not a true reflection of the maternal ovarian reserve.

Longitudinal follow-up studies are needed to determine whether neonatal AMH levels are predictive of future ovarian reserve, pubertal development, or reproductive outcomes later in life.

Conclusions

Our findings suggest that neonatal AMH levels are not significantly influenced by birth weight status at birth while maternal and neonatal AMH levels are correlated. These results may provide novel insights into early ovarian reserve and maternal–neonatal endocrine relationships however they should be considered preliminary and interpreted with caution. Further large-scale, longitudinal studies are needed to establish the clinical relevance of neonatal AMH levels and to determine whether these early-life measurements predict long-term ovarian reserve or reproductive outcomes.

Attestation Statements

Data regarding any of the subjects in the study has not been previously published. Data will be made available to the editors of the journal for review or query upon request. The STROBE checklist for this study design was followed.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethical Statement

The study was approved by the Carmel Medical Center Institutional Review Board on August 24, 2021 (protocol number CMC-0080-21) and was conducted in accordance with the Declaration of Helsinki. A written and signed informed consent form was obtained from all study participants before recruitment.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflict of interest.

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