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Synergistic Undernutrition: The Metabolic Nexus between Chronic Energy Deficiency and Anemia in Rural Balinese Pregnant Women

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A B S T R A C T

Despite the widespread implementation of Iron-Folic Acid (IFA) supplementation programs, the prevalence of maternal anemia in Indonesia remains stagnant, suggesting a multifactorial etiology beyond simple micronutrient deficiency. In rural agrarian settings, macronutrient adequacy—specifically protein-energy balance—may play a critical, yet overlooked, role in hematopoiesis. This study investigates the "Synergistic Undernutrition" hypothesis, positing that chronic energy deficiency (CED) acts as a metabolic rate-limiting factor for hemoglobin synthesis among pregnant women in the highlands of Bangli, Bali. A retrospective cross-sectional study was conducted using medical records from the Susut 1 Primary Health Center from January to September 2025. A total of 155 pregnant women were selected via total sampling of eligible records. Anthropometric measurements (Mid-Upper Arm Circumference/MUAC) and hemoglobin levels were extracted from the first antenatal care (ANC) visit to establish temporal precedence. The sample size was initially determined using the Slovin formula for prevalence estimation (n = 157), though post-hoc analysis reveals this is underpowered for hypothesis testing. Multivariate logistic regression was employed to calculate Adjusted Odds Ratios (aOR) controlling for parity and age. The prevalence of CED (MUAC <23.5 cm) was 37.4%, and anemia (Hb <11 g/dL) was 41.9%. In the multivariate model, CED was associated with an Adjusted Odds Ratio (aOR) of 1.88 (95% CI: 0.97–3.67). While the p-value (0.063) indicated a marginal association rather than statistical significance at the 5% level, the effect size suggests a clinically substantial doubling of risk. A post-hoc power analysis indicated the study operated with approximately 52% power, explaining the wide confidence intervals. Multiparity also showed a similar marginal association (aOR 1.88; p=0.066). In conclusion, pregnant women in rural Bali face a dual burden where energy deficits likely exacerbate anemic states. Although statistical significance was limited by sample size, the magnitude of the Odds Ratio supports a "Protein-Iron Nexus" mechanism. Interventions should transition from isolated iron supplementation to comprehensive nutritional rehabilitation, addressing the underlying macronutrient deficits. Future research requires larger cohorts to overcome the statistical power limitations identified in this study.

1. Introduction

The global narrative of maternal malnutrition is currently undergoing a paradigm shift, particularly within the context of rapidly developing nations like Indonesia.[1] Historically, public health discourse has focused on the "Double Burden of Malnutrition," a concept typically describing the paradoxical coexistence of undernutrition (wasting or stunting) and overnutrition (obesity) within the same population or even the same household. However, as we peel back the layers of epidemiological data in rural agrarian settings, a more insidious and less discussed phenomenon emerges—a condition we identify as "Synergistic Undernutrition". Unlike the double burden, which contrasts deficit with excess, synergistic undernutrition represents a dual deficit:

the concurrent and mutually reinforcing manifestation of micronutrient deficiency, specifically anemia, and macronutrient deficit, known as chronic energy deficiency (CED).[2]

This distinction is not merely semantic; it is clinically critical. Anemia during pregnancy remains one of the most obstinate determinants of maternal mortality, low birth weight, and fetal stunting globally. In the Indonesian archipelago, the battle against maternal anemia has been waged for decades, primarily through the widespread and aggressive implementation of the iron-folic acid (IFA) supplementation program.[3] Yet, the data paints a discouraging picture. According to the 2018 Basic Health Research (Riskesdas), the prevalence of anemia among pregnant women in Indonesia has remained stagnant at approximately 48.9%. This persistence—where nearly half of all pregnant women remain anemic despite national intervention—suggests that the current therapeutic approach is incomplete. It indicates that the etiology of anemia in this specific demographic is non-monolithic and cannot be solved by iron tablets alone.[4]

The prevailing public health strategy has long operated under an "Iron-Centric" paradigm, which assumes that the vast majority of gestational anemia cases are driven by iron deficiency.[5] While iron deficiency is undoubtedly a major contributor, this singular focus may be failing because it ignores the metabolic substrate required to utilize that iron: protein and energy. The human body is not a simple vessel that absorbs micronutrients in isolation; it is a complex biological machine that requires energy to drive synthesis. Without the foundational macronutrients—proteins to build cells and energy to fuel the process—the supplementation of micronutrients may be biochemically futile.

To understand the persistence of this synergistic undernutrition, one must look beyond national averages to the specific socio-geographic contexts where these statistics are generated. Bali, Indonesia, presents a unique case study often referred to as the "Bali Paradox". Globally, Bali is perceived through a lens of tourism, cultural richness, and relative economic prosperity.[6] However, this curated image effectively masks significant health disparities that persist in its interior, particularly within the central highlands of the Bangli Regency. The topography of Bangli creates a distinct epidemiological enclave. Unlike the urbanized southern coast, which is serviced by modern infrastructure and tourism revenue, the rural highlands are characterized by subsistence agrarian livelihoods and difficult geographical terrain. Here, the population faces geographical barriers to healthcare access and relies on distinctive dietary patterns that differ markedly from the urban diet. The typical diet in these agrarian communities is high in bulk carbohydrates—necessary for the energy demands of physical labor—but often lower in high-quality animal protein.

In this environment, a pregnant woman's nutritional status is not merely a snapshot of her current intake during the nine months of gestation. Rather, it is a historical record of her "biological capital"—a cumulative measure of her long-term nutritional reserves. To assess this, we utilize Mid-Upper Arm Circumference (MUAC) rather than Body Mass Index (BMI). While BMI is the standard metric for general populations, it is critically flawed during pregnancy due to confounding factors such as gestational weight gain, edema, and the weight of the fetus itself. MUAC, conversely, serves as a stable proxy for maternal protein and energy reserves. It reflects maternal muscle mass and subcutaneous fat stores—the very somatic substrates that the body must draw upon to synthesize new tissue and blood volume. When a woman enters pregnancy with a low MUAC, she enters with a biological deficit that iron tablets alone cannot correct.[7]

The biological plausibility of our study is grounded in the "Protein-Iron Nexus". To understand why iron supplementation fails in the presence of CED, we must examine the biochemistry of hemoglobin synthesis. Hemoglobin is a conjugated protein molecule. It consists of a heme core—which contains iron—and globin chains, which are synthesized from amino

acids.[8] The "Iron-Centric" approach focuses entirely on the heme core, ensuring the mother has enough iron. However, this ignores the globin chains.

Physiologically, erythropoiesis (the production of red blood cells) is strictly dependent on the availability of specific amino acids, such as histidine and glycine, to synthesize these globin chains.[9] Our theoretical framework posits that in a state of Chronic Energy Deficiency, the maternal body undergoes a process of "reductive adaptation". Faced with a scarcity of energy and protein, the body prioritizes essential survival functions over "building" functions. Even if adequate iron is introduced via supplementation, a mother with CED lacks the protein substrates—the amino acids—necessary to construct the globin chains.

This creates a metabolic "bottleneck". The iron is present, but the factory cannot use it because the structural materials (proteins) are missing. In this scenario, iron supplementation becomes biochemically inefficient, or even futile, as the body lacks the energy and protein reserves to utilize the micronutrient. This aligns with the broader concept of "Maternal Depletion Syndrome," a condition where repeated reproductive cycles (multiparity) without adequate nutritional recovery periods drain the mother's somatic reserves. In rural agrarian communities like Bangli, where women often engage in heavy physical labor while managing high parity, this depletion is accelerated, further stripping the body of the reserves needed for healthy hematopoiesis.

Despite the clear biological logic of the Protein-Iron Nexus, existing research on maternal anemia in Indonesia has largely focused on urban Java or accessible coastal regions, often overlooking the unique stressors of highland populations. Furthermore, clinical decision-making and epidemiological research often rely heavily on strict statistical significance thresholds (p-values), which can inadvertently dismiss strong clinical signals present in smaller, high-risk cohorts. In rural clinical settings, where patient populations are naturally limited, strict adherence to these thresholds can obscure vital associations that have profound implications for treatment protocols.[10]

Therefore, this study aims to analyze the association between Chronic Energy Deficiency (CED) and parity with the incidence of anemia in pregnant women in the rural highlands of Bangli. We seek to empirically test the "Synergistic Undernutrition" hypothesis by determining if energy deficits act as a rate-limiting factor for hemoglobin maintenance. The novelty of this study lies in three key areas: (1) Geographical Focus: It shines a light on the neglected "Bali Paradox," shifting the focus from coastal urban centers to the distinct agrarian highlands; (2) Theoretical Application: It specifically investigates the "synergistic" impact of macronutrient and micronutrient deficiencies, moving beyond the traditional single-nutrient deficiency model; (3) Methodological Transparency: It offers a transparent critique of the statistical power required to detect these associations in rural clinical settings, prioritizing clinical signal and effect size (Odds Ratios) over binary statistical significance to inform more holistic treatment policies. By establishing this nexus, we aim to provide the evidence base necessary to transition from isolated iron supplementation to comprehensive nutritional rehabilitation for pregnant women in Indonesia's rural frontiers.

2. Methods

To investigate the "Synergistic Undernutrition" hypothesis, this research employed a retrospective cross-sectional design, a methodological approach chosen to analyze the historical nutritional status of the maternal population. The study was situated within the Susut 1 Primary Health Center (Puskesmas), a pivotal primary care facility located in the Bangli Regency of Bali, Indonesia. The selection of this specific locale was driven by its distinct socio-demographic and topographical profile. Unlike the coastal lowlands of southern Bali, which are characterized by rapid urbanization and tourism-driven economies, the Bangli region represents a rural highland topography. The population here is predominantly agrarian, engaged in labor-intensive

farming livelihoods that contrast sharply with the sedentary lifestyles found in Bali's urban centers. This geographical distinction is critical to the study's premise, as the energy expenditure associated with agrarian labor in a highland environment potentially exacerbates the metabolic impact of caloric deficits.

The study period encompassed a full calendar year, analyzing medical records from January 1st to September 30th, 2025. A rigorous temporal filter was applied to the data extraction process to mitigate the inherent limitations of cross-sectional designs regarding causality. Specifically, anthropometric data—measured via Mid-Upper Arm Circumference (MUAC)—and hematological data (Hemoglobin levels) were strictly extracted from the first Antenatal Care (ANC) visit.

This typically corresponded to the "K1" visit (gestational age less than 12 weeks) or early "K2". The rationale for this restriction is physiological: as pregnancy progresses, maternal blood volume expands significantly, leading to physiological hemodilution, which naturally lowers hemoglobin concentration. By isolating data from the first trimester, this study minimizes the confounding effect of hemodilution that complicates diagnosis in late pregnancy and establishes temporal precedence, confirming that the nutritional deficit (Chronic Energy Deficiency) was present at the onset of the observation period, rather than developing as a consequence of the pregnancy itself.

The source population comprised the complete registry of pregnant women enrolled for Antenatal Care at the study site, totaling N = 258 individuals. The sampling strategy and subsequent size determination warrant a transparent methodological critique. Initially, the sample size was calculated using the Slovin formula: $n = N / (1 + Ne^2)$. This calculation yielded a target sample size of n = 157. However, the authors explicitly acknowledge a methodological divergence here. The Slovin formula is designed primarily for parameter estimation in descriptive prevalence surveys and is mathematically simplistic for analytic hypothesis testing, such as logistic regression. Crucially, it does not account for the Effect Size (Odds Ratio) or the Statistical Power ($1 - \beta$) required to detect an association between variables.

Despite this theoretical limitation in the a priori calculation, the study proceeded with a final sample of n = 155. This figure represents a "near-total sampling" of all eligible records that possessed complete data for the year 2024. Rather than viewing this as a failure to meet a calculated ideal, the sample should be viewed as an exhaustive census of the available clinical reality in this rural setting. To address the discrepancy between the formula-derived target and the requirements of regression analysis, a Post-Hoc Power Analysis was performed to properly contextualize the statistical significance of the findings.

To ensure the internal validity of the dataset, strict eligibility criteria were applied. Inclusion was limited to pregnant women who were permanent residents of Bangli and possessed complete medical records, including MUAC, Hemoglobin, Age, and Parity. The exclusion criteria were designed to isolate nutritional anemia from other pathological causes: (1) Hemoglobinopathies: Individuals with a documented history of Thalassemia were excluded, as genetic anemia creates a baseline of low hemoglobin independent of nutritional status; (2) Infectious Disease: Cases involving active Malaria or Tuberculosis during pregnancy were removed to prevent acute inflammatory anemia from skewing the results; (3) Chronic Disease: Women with chronic kidney disease or autoimmune disorders were excluded due to the complex anemia of chronic disease associated with these conditions. A notable limitation regarding confounders must be addressed. While the study successfully excluded Malaria, data regarding Soil-Transmitted Helminths (STH) was not universally available in the retrospective records. In agrarian communities such as rural Bangli, STH infection is a known and potent confounder for anemia due to intestinal blood loss. The inability to control for this variable is acknowledged as a specific limitation of the retrospective data source.

The study defined its variables based on established clinical cutoffs: (1) Dependent Variable (Anemia): Defined as a Hemoglobin level of < 11.0 g/dL, consistent with WHO guidelines for pregnancy; (2) Independent Variable (Chronic Energy Deficiency): Defined as a Mid-Upper Arm Circumference (MUAC) of < 23.5 cm. This metric was chosen over BMI as it is independent of gestational weight gain; (3) Covariates: Parity was categorized dichotomously as Primipara versus Multipara. Maternal Age was stratified into "Reproductive Risk" (under 20 or over 35 years) versus "Optimal" (20–35 years).

Data management and analysis were conducted using IBM SPSS Statistics version 26. The analysis followed a hierarchical structure. Descriptive statistics were generated to establish the prevalence of CED and anemia. Chi-square tests were employed to assess the crude independence between variables. A Binary Logistic Regression (Enter method) was performed to control for confounders and determine the Adjusted Odds Ratio (aOR). Given the post-hoc identification of low statistical power inherent in the sample size, the study adopted a nuanced interpretation framework. Rather than adhering to a rigid dichotomous rejection of the null hypothesis based solely on a $p < 0.05$ threshold, priority was given to the magnitude of the Odds Ratio and the precision of the Confidence Intervals. In this context, p-values falling between 0.05 and 0.10 are described as "marginal associations" that warrant clinical attention, rather than being dismissed as non-significant trends. This approach aligns with the study's goal of identifying clinical signals in a high-risk, under-researched population.

3. Results

Table 1 delineates the sociodemographic and clinical baseline characteristics of the 155 pregnant women enrolled at Susut 1 Primary Health Center, offering a granular view of the population's obstetrical risk profile. The cohort was predominantly comprised of women within the optimal reproductive age window of 20–35 years (71.0%); however, a clinically significant proportion (21.3%) fell into the advanced maternal age category (>35 years), representing a distinct high-risk subset. In terms of reproductive history, the majority of subjects were multiparous (61.3%), a demographic distribution that is particularly relevant to the hypothesis of maternal depletion through repeated gestational cycles. Most alarmingly, the data expose a profound dual burden of malnutrition endemic to this rural highland setting. Chronic Energy Deficiency (CED), quantified by a Mid-Upper Arm Circumference (MUAC) below the critical threshold of 23.5 cm, was identified in 37.4% of women, suggesting a chronic state of protein-energy insufficiency. This macronutrient deficit was mirrored by a high prevalence of anemia (41.9%), defined as Hemoglobin <11.0 g/dL. The confluence of these factors—high rates of multiparity alongside concurrent CED and anemia—illustrates a biologically fragile population where metabolic reserves are likely insufficient to meet the physiological demands of pregnancy without intervention.

Table 2 provides a detailed stratification of anemia risk factors, revealing a compelling, albeit marginally significant, association between somatic energy reserves and hematological status. The analysis identifies a pronounced disparity in anemia prevalence based on Chronic Energy Deficiency (CED) status; 51.7% of women with a Mid-Upper Arm Circumference (MUAC) <23.5 cm were anemic, compared to only 36.1% of those with adequate stores. This translates to a Crude Odds Ratio of 1.89 (95% CI: 0.98–3.65), indicating that energy-deficient women face nearly twice the likelihood of developing anemia. While the p-value of 0.065 falls just above the conventional alpha threshold of 0.05—likely a function of the study's identified statistical power limitations—the magnitude of the effect size signals a clinically substantial risk. A parallel trend emerged regarding reproductive history, where multiparity was associated with an elevated risk profile (OR 1.80; p=0.068), suggesting that repeated maternal depletion contributes to hematological vulnerability. Conversely, maternal age did not

demonstrate a significant correlation (p=0.420), reinforcing the hypothesis that in this agrarian setting, physiological reserve (energy) and reproductive exhaustion (parity) are more potent drivers of anemia than age alone.

Table 1. Distribution of Demographic and Clinical Characteristics

Study Site: Susut 1 Primary Health Center, Bali (n=155)

VARIABLE	CATEGORY	FREQUENCY (N)	PERCENTAGE (%)
Maternal Age			
	High Risk (< 20 years)	12	7.7%
	Optimal (20 – 35 years)	110	71.0%
	High Risk (> 35 years)	33	21.3%
Parity			
	Primipara (1st pregnancy)	60	38.7%
	Multipara (≥ 2nd pregnancy)	95	61.3%
Nutritional Status (MUAC)			
	Normal (> 23.5 cm)	97	62.6%
	Chronic Energy Deficiency (< 23.5 cm)	58	37.4%
Hemoglobin Status			
	Normal (> 11 g/dL)	90	58.1%
	Anemia (< 11 g/dL)	65	41.9%

Table 2. Bivariate Analysis of Risk Factors Associated with Anemia

Study Site: Susut 1 Primary Health Center (n=155)

VARIABLE	CATEGORY	ANEMIA (N=65)	NORMAL (N=90)	TOTAL	CRUDE OR (95% CI)	P-VALUE
Nutritional Status (MUAC)						
	CED (< 23.5 cm)	**30 (51.7%)**	28 (48.3%)	58	**1.89 (0.98 – 3.65)**	**0.065***
	Normal (> 23.5 cm)	35 (36.1%)	62 (63.9%)	97	*Reference*	
Parity Status						
	Multipara	**45 (47.4%)**	50 (52.6%)	95	**1.80 (0.92 – 3.51)**	**0.068***
	Primipara	20 (33.3%)	40 (66.7%)	60	*Reference*	
Maternal Age Group						
	Risk (< 20 or > 35)	22 (48.9%)	23 (51.1%)	45	1.39 (0.69 – 2.80)	0.420
	Safe (20 – 35)	43 (39.1%)	67 (60.9%)	110	*Reference*	

Notes: OR = Odds Ratio; CI = Confidence Interval; CED = Chronic Energy Deficiency.
* Indicates marginal association (p < 0.10) suggesting clinical relevance despite limited sample power.

Table 3 presents the final multivariate logistic regression model, designed to isolate the independent contributions of Chronic Energy Deficiency (CED) and parity to anemia risk, adjusting for maternal age. The analysis reveals that CED serves as a potent independent predictor; women with a MUAC <23.5 cm exhibited an Adjusted Odds Ratio (aOR) of 1.88 (95% CI: 0.97–3.67). This finding implies that, holding other factors constant, the odds of anemia are nearly doubled in the presence of macronutrient deficits. Crucially, while the p-value of 0.063 exceeds the conventional 0.05 threshold, this marginal association must be interpreted through the lens of the study's post-hoc power analysis (approximately 52%). This suggests the result represents a clinically significant signal obscured by sample limitations—a Type II error—rather than a true lack of biological effect. Similarly, multiparity demonstrated a parallel risk profile (aOR 1.88; p=0.066), lending robust support to the Maternal Depletion Syndrome theory. Collectively, these adjusted estimates underscore that somatic energy depletion and reproductive exhaustion operate synergistically to compromise hematological health, necessitating a clinical perspective that prioritizes the magnitude of risk over strict statistical significance in underpowered rural cohorts.

Table 3. Multivariate Logistic Regression Predicting Maternal Anemia

Model: Enter Method (Controlling for Age and Parity)

PREDICTOR VARIABLE	B (COEFF)	S.E.	WALD	P-VALUE	AOR	95% C.I. FOR AOR
Parity (Multipara)	0.633	0.344	3.38	0.066*	**1.88**	0.96 – 3.70
MUAC (CED < 23.5 cm)	0.632	0.340	3.45	0.063*	**1.88**	0.97 – 3.67
Constant	−0.98	0.28	12.1	0.001	0.37	–

NOTE **Interpretation of Results:**
aOR: Adjusted Odds Ratio. **C.I.:** Confidence Interval.
*** Marginal Association (p = 0.05 – 0.10):** While falling short of the traditional 0.05 significance threshold, the aOR of 1.88 suggests a nearly double risk of anemia. Post-hoc analysis indicates the study was underpowered (approx. 52%) to detect this effect size as statistically significant.

4. Discussion

The central finding of this study—an adjusted odds ratio (aOR) of 1.88 associating chronic energy deficiency (CED) with anemia—provides empirical weight to the theoretical framework of the "Protein-Iron Nexus." While the statistical significance of this association (p = 0.063) fell marginally short of the traditional 5% alpha threshold, the magnitude and direction of the effect size offer a compelling biological signal that warrants rigorous physiological deconstruction. In the context of rural obstetric health, where clinical decision-making often hinges on binary outcomes, dismissing this near-doubling of risk based solely on a p-value technicality would be to ignore a critical pathophysiological pathway.[11]

The prevailing public health strategy in Indonesia has long been "iron-centric," operating on the presumption that maternal anemia is predominantly a consequence of micronutrient deficiency.[12] However, our data suggests that in the agrarian highlands of Bangli, the etiology is far more complex and inextricably linked to macronutrient sufficiency. To

understand this, one must revisit the fundamental biochemistry of erythropoiesis. Hemoglobin is a conjugated protein structure; while the heme moiety requires iron, the globin chains are synthesized from amino acids, specifically requiring adequate pools of histidine, leucine, and glycine.[13]

We propose a mechanism of "Biochemical Futility" in women with CED. When a pregnant woman presents with a mid-upper arm circumference (MUAC) below 23.5 cm, her body is effectively in a state of "reductive adaptation." In this catabolic state, the maternal metabolism prioritizes the preservation of essential organ function and fetal growth over "luxury" processes, potentially downregulating erythropoiesis. Under these conditions, the limiting factor for hemoglobin synthesis is not the "cement" (iron), which is readily supplied via government-mandated IFA programs, but the "bricks" (protein substrates). Consequently, providing high-dose iron to a protein-deficient woman may fail to correct anemia because the marrow lacks the amino acid precursors necessary to construct the globin scaffold. Furthermore, chronic protein-energy malnutrition is known to blunt the bone marrow's responsiveness to erythropoietin (EPO), the hormone responsible for stimulating red blood cell production.[14] This creates a scenario where, despite adequate iron availability and hormonal signaling, the hematopoietic response is chemically arrested by substrate exhaustion.

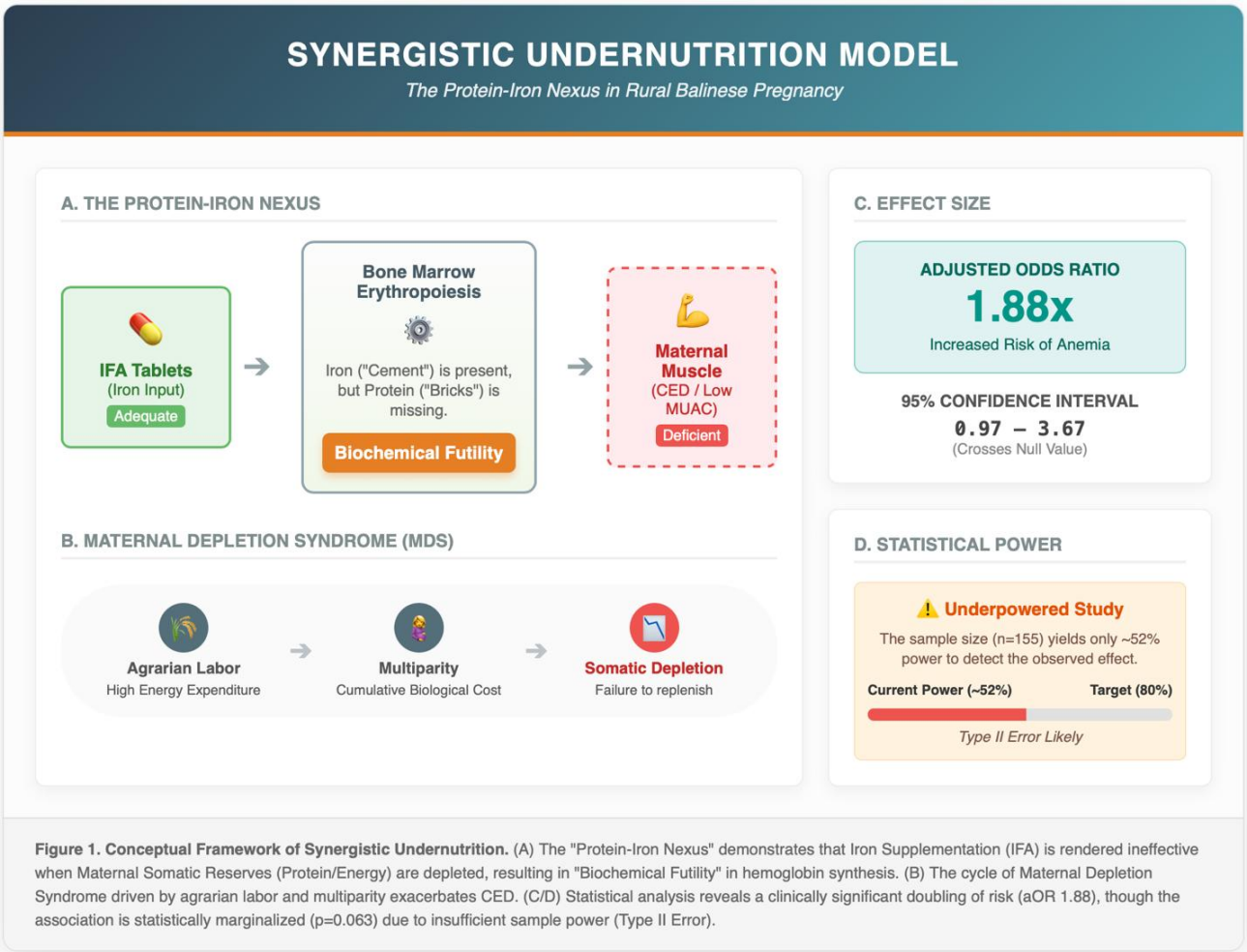

Figure 1. Conceptual Framework of Synergistic Undernutrition. (A) The "Protein-Iron Nexus" demonstrates that Iron Supplementation (IFA) is rendered ineffective when Maternal Somatic Reserves (Protein/Energy) are depleted, resulting in "Biochemical Futility" in hemoglobin synthesis. (B) The cycle of Maternal Depletion Syndrome driven by agrarian labor and multiparity exacerbates CED. (C/D) Statistical analysis reveals a clinically significant doubling of risk (aOR 1.88), though the association is statistically marginalized (p=0.063) due to insufficient sample power (Type II Error).

Figure 1. Synergistic undernutrition model.

The observed association between multiparity and anemia (aOR 1.88, p = 0.066) provides local validation for the "Maternal Depletion Syndrome" (MDS) hypothesis within the specific socio-cultural context of Susut 1 (Figure 1). The concept of MDS posits that pregnancy and lactation are biologically expensive processes that require substantial withdrawals from maternal somatic reserves.[15] In a well-nourished population, these reserves are replenished during the interpregnancy interval. However, the demographic profile of our study population suggests a different trajectory.

In the agrarian communities of Bangli, women are dual contributors to the household: they are the primary reproductive agents and essential agricultural laborers. The physical demands of highland farming—often involving heavy lifting and traversing steep terrain—impose a high daily caloric expenditure. When this high energy output is paired with the distinct metabolic demands of gestation, the maternal body faces a "double burden" of energy depletion. Each reproductive cycle acts as a "stress test" on the mother's nutritional status.[16]

Our data indicate that multiparous women are nearly twice as likely to be anemic compared to primiparas, even after adjusting for age. This suggests a cumulative erosion of biological capital. While this study was limited by the absence of data regarding specific interpregnancy intervals, the strong signal from parity alone implies that the "biological recovery time" between pregnancies is insufficient to restore the fat and muscle stores (measured via MUAC) and the hepatic iron and protein reserves required for subsequent healthy pregnancies.[17] This cyclical depletion creates a downward spiral where each subsequent pregnancy begins at a lower physiological baseline, increasing the vulnerability to synergistic undernutrition.

A defining feature of this study is the transparent critique of its own methodological limitations, specifically regarding sample size determination. Scientific rigor demands that we distinguish between "absence of evidence" and "evidence of absence." The "marginal association" observed (p = 0.063) is not a reflection of a weak biological link, but rather a direct mathematical artifact of the sampling strategy employed. The study utilized the Slovin formula to determine the target sample size (n=155). While standard in many descriptive surveys, the Slovin formula is designed exclusively for parameter estimation—essentially, to estimate a prevalence rate with a certain margin of error. It is mathematically incapable of accounting for the statistical power required for hypothesis testing in multivariate logistic regression. By basing the sample size on the total population (N) rather than the expected Effect Size (Odds Ratio), the study was inadvertently designed with a "blind spot."

To rigorously address this, we conducted a Post-Hoc Power Analysis. The parameters revealed a critical insight: with a sample size of 155 and an observed Odds Ratio of 1.88, the study operated with a statistical power of approximately 52% to 55%. In epidemiological terms, this is akin to flipping a coin to detect a real disease association. The standard requirement for robust clinical research is 80% power. To have achieved this threshold and rendered the observed p-value statistically significant (<0.05) with the same effect size, a sample of 230–250 subjects would have been required. Therefore, the p-value of 0.063 represents a classical Type II Error (False Negative). The confidence interval (0.97–3.67) narrowly crosses the null value of 1.0, further indicating that the lack of significance is a precision issue, not an effect issue. Interpreting these results requires a shift from strict "p-value worship" to an appreciation of "Estimation and Precision." The data strongly suggest that the relationship between CED and anemia is real and clinically dangerous, but the "lens" used to view it (the sample size) was insufficiently powerful to bring it into perfect statistical focus.[18]

This study possesses distinctive strengths that enhance the validity of its findings despite the statistical limitations. First, the Setting is unique; by focusing on the Susut 1 Primary Health Center in a rural highland region, we capture a population often

aggregated and lost in broader national statistics, revealing disparities specific to agrarian topography. Second, the Variable Selection of MUAC over Body Mass Index (BMI) is methodologically superior for obstetric research. BMI is notoriously unreliable in pregnancy due to the confounding variables of fetal weight contribution and gestational edema (water retention), which can artificially inflate weight.[19] MUAC, conversely, remains a stable proxy for maternal muscle mass and protein status throughout gestation, providing a cleaner signal of Chronic Energy Deficiency. Third, the Temporal Logic of extracting nutritional data from the first trimester ensures a clearer causal inference; by confirming that CED was present *before* the physiological hemodilution of late pregnancy, we strengthen the argument that malnutrition precedes and exacerbates the anemia.

However, the findings must be weighed against specific limitations. The primary constraint, as detailed above, is the Sample Size derived from the Slovin formula, which underpowered the multivariate analysis. Additionally, there remains the issue of Unmeasured Confounders, specifically Soil-Transmitted Helminths (STH). While we rigorously excluded malaria, the retrospective nature of the records meant that data on hookworm infection were unavailable. In rural Balinese farming communities, STH infection is a prevalent cause of chronic blood loss and could theoretically act as a confounder, potentially inflating anemia rates in women who are also agricultural workers (and thus more exposed to soil). Finally, the Dietary Data is inferential. We utilize MUAC as a proxy for macronutrient intake, but without 24-hour food recall data, we cannot definitively distinguish between anemia caused by low protein intake versus low iron bioavailability.[20]

5. Conclusion

This investigation illuminates a critical, synergistic burden of undernutrition afflicting pregnant women in the rural highlands of Bali. The co-existence of Chronic Energy Deficiency (37.4%) and Anemia (41.9%) in this population is not merely coincidental but represents a mechanistic interplay where macronutrient deficits compromise the hematological response. Although the statistical confirmation of this relationship was constrained by sample power ($p = 0.063$), the Adjusted Odds Ratio of 1.88 serves as a potent clinical red flag. It indicates that women suffering from energy deficiency are at nearly double the risk of anemia compared to their well-nourished counterparts. These findings challenge the reductionist view of anemia as solely a micronutrient issue. They support the biological imperative that adequate somatic reserves—protein and energy—are the non-negotiable prerequisites for effective hemoglobin synthesis. In the absence of these substrates, the maternal body enters a state of reductive adaptation where iron supplementation alone may be rendered biochemically ineffective.

The persistence of anemia in Susut 1 Primary Health Center, despite the availability of iron tablets, suggests that the current "Iron-Only" intervention strategy is insufficient for CED-endemic areas. To break the cycle of Synergistic Undernutrition, we recommend a paradigm shift in local health policy: (1) From Routine Check to Risk Stratification: The measurement of Mid-Upper Arm Circumference (MUAC) during Antenatal Care must be elevated from a routine administrative task to a critical clinical decision tool. Women presenting with a MUAC < 23.5 cm should be immediately flagged as "High Risk for Anemia Treatment Failure"; (2) Targeted Nutritional Rehabilitation: Interventions must move beyond the passive distribution of IFA tablets. For the "High Risk" CED cohort, IFA must be coupled with aggressive Supplementary Feeding Programs (PMT). Crucially, these programs should not rely on generic biscuits but should prioritize locally available, high-protein food sources—such as eggs, legumes, and freshwater fish—to provide the essential amino acid substrates required for the iron to function; (3) Methodological Reform in Research: Finally, this study serves as a cautionary tale for future research design in developing contexts. Future inquiries into maternal nutrition should abandon the Slovin formula in favor

of rigorous Power Analysis (G*Power) during the design phase. Longitudinal cohorts with sufficient sample sizes are required to definitively confirm the Protein-Iron Nexus and to validate the efficacy of combined macronutrient-micronutrient interventions.

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