



Original Research Article

PROSPECTIVE OBSERVATIONAL STUDY ON SOCIOECONOMIC STATUS AND DIETARY FACTORS RESPONSIBLE FOR NON-INITIATION OR DELAYED INITIATION OF LACTATION

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ABSTRACT

Background: Lactogenesis II is a hormonally regulated process dependent on coordinated prolactin, insulin, cortisol, and progesterone withdrawal. Increasing evidence suggests that metabolic dysfunction may impair secretory activation despite adequate nutritional intake. Delayed initiation of lactation remains common in India, yet the endocrine–metabolic determinants underlying this phenomenon are insufficiently explored. This study aimed to evaluate the association of socio-economic status (SES) and dietary factors with delayed or non-initiation of lactation, with emphasis on potential endocrine–metabolic mechanisms.

Materials and Methods: This analytical cross-sectional study included 400 postpartum mothers (within six months of delivery), comprising 200 cases (delayed/non-initiation of lactation) and 200 controls (normal initiation). SES was classified using the Modified Kuppaswamy Scale 2023. Dietary pattern was categorized as vegetarian or non-vegetarian, and daily caloric intake was estimated using structured dietary recall. Independent variables included maternal age, parity, education, mode of delivery, and antenatal care utilization. Continuous variables were analyzed using independent t-tests and categorical variables using Chi-square tests. A p-value ≤ 0.05 was considered statistically significant.

Results: Mothers with delayed lactation were significantly older than controls (28.63 ± 4.67 vs. 24.83 ± 4.08 years; $p < 0.0001$). Mean caloric intake was significantly higher in the delayed group (2723.77 ± 456.68 kcal) compared to the normal group (1852.99 ± 320.40 kcal; $p < 0.0001$). A strong association was observed between SES and lactation status ($p < 0.001$), with delayed lactation more prevalent among upper-middle and lower-middle socio-economic strata.

Conclusion: Delayed initiation of lactation is significantly associated with advancing maternal age, higher caloric intake, and middle SES. The findings support the hypothesis that endocrine–metabolic dysregulation—particularly impaired insulin sensitivity and altered prolactin responsiveness—may underlie delayed lactogenesis. In socio-economically transitioning populations, metabolic risk factors may exert greater influence on breastfeeding initiation than caloric insufficiency. Integrating metabolic screening with structured lactation support in accordance with World Health Organization recommendations may improve early breastfeeding outcomes and neonatal health indicators.

Keywords: Delayed lactation, Lactogenesis II, Socioeconomic status, Caloric intake, Insulin resistance, Endocrine dysfunction, Maternal age, Metabolic imbalance, Breastfeeding initiation.

INTRODUCTION

Lactation is a critical determinant of neonatal health, providing optimal nutrition, immune protection, and promoting mother-infant bonding. Early initiation of breastfeeding, ideally within the first hour after birth, is recommended by the World Health Organization (WHO) to reduce neonatal morbidity and mortality.^[1] Despite this, delayed initiation or non-initiation of lactation remains a significant public health concern, particularly in low- and middle-income countries. Studies have shown that delayed breastfeeding initiation is associated with higher neonatal mortality and adverse health outcomes.^[2]

Socioeconomic status (SES) plays a pivotal role in maternal and child health. Mothers from lower socioeconomic strata often face barriers such as limited access to healthcare, inadequate nutrition, low literacy, and restricted support systems, all of which can contribute to delayed initiation of lactation.^[3] Dietary factors during pregnancy and the postpartum period also influence lactogenesis. Poor maternal nutrition, micronutrient deficiencies, and insufficient caloric intake may impair prolactin function and reduce milk production, resulting in delayed onset of lactation.^[4]

Early initiation of breastfeeding (EIBF), defined as providing breast milk within the first hour of birth, ensures infants receive colostrum, which offers essential nutrients, antibodies, and early immunological protection. EIBF supports optimal growth, cognitive development, mother-infant bonding and reduces later-life risks such as obesity and noncommunicable diseases. It also protects against neonatal infections, including diarrhoea, sepsis, and pneumonia, and increases the likelihood of exclusive breastfeeding. Although institutional delivery is expected to promote EIBF, national data show that only 42.6% of institutional births in India achieve it. Identifying maternal and facility-related barriers is essential, especially as our hospital adopts the Baby-Friendly Hospital Initiative.^[5]

Understanding the combined effect of SES and dietary factors on lactation practices is vital for developing effective public health interventions. This prospective observational study aims to evaluate the association of socioeconomic status and dietary patterns with non-initiation or delayed initiation of lactation among postpartum mothers. Insights from this study will inform targeted nutrition counselling, community-based programs, and policy strategies to improve early breastfeeding practices, ultimately enhancing maternal and neonatal health outcomes.^[6] The aims of the study to assess the association of socio-economic status (SES) and dietary factors with delayed initiation / non-initiation of lactation.

MATERIALS AND METHODS

Study Design: An analytical cross-sectional study has been designed to evaluate socioeconomic and

dietary factors associated with non-initiation or delayed initiation of lactation.

Place of study: Department of Lactation Clinic, Institute of Child Health (ICH), Kolkata, West Bengal, India.

Period of study: 30 October 2023 to 28 February 2025.

Study Population: Postpartum mothers (within 6 months of delivery), aged 18–35 years, who delivered healthy term neonates and visited the Lactation Clinic of ICH, Kolkata.

Sample size: Based on sample size calculations using findings from a previous study,^[5] a total of 400 participants will be enrolled:

- 200 cases (delayed initiation / non-initiation of lactation).
- 200 controls (normal lactation initiation).

Study Definitions

- Socioeconomic Status (SES): Classified using the Modified Kuppuswamy Scale 2023.^[19]
- Dietary Habits: Categorized as Vegetarian or Non-vegetarian.
- Calorie Consumption: Estimated from a structured dietary recall questionnaire.

Inclusion Criteria

Cases:

- Mothers with delayed initiation of lactation (lactation established after 72 hours postpartum)
- Mothers with non-initiation of lactation (no onset of lactation)

Controls:

- Mothers with normal initiation of lactation
- Age-matched and comparable for other baseline factors

Exclusion Criteria

For Cases

- Nipple abnormalities in mother
- Oral/oropharyngeal abnormalities in the newborn
- Faulty feeding or latching techniques
- Preterm delivery
- Critically ill mother or newborn

For Controls

- Informed consent not given

Withdrawal Criteria

- Mothers unwilling to continue participation at any stage or lost to follow-up.

Study Variable

Independent Variables

- Socioeconomic status (SES)
- Dietary habit (Vegetarian/Non-vegetarian)
- Calorie consumption
- Maternal age
- Parity
- Educational level
- Mode of delivery
- Antenatal care utilization

Dependent Variable

- Lactation initiation status (normal vs. delayed/non-initiation)

Statistical Analysis: For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analysed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism (version 5). Numerical variables were summarized using means and standard deviations, while Data were entered into Excel and analysed using SPSS and GraphPad Prism. Numerical variables were summarized using means and standard

deviations, while categorical variables were described with counts and percentages. Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data. Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

RESULTS

Table 1: Distribution of Dietary habit

| Dietary Habit | Frequency | Percent |
|----------------|-----------|---------|
| Non-vegetarian | 336 | 84.0 |
| Vegetarian | 64 | 16.0 |
| Total | 400 | 100.0 |

Table 2: Age and Calorie Intake in Delayed vs. Normal Lactation (Mean \pm SD)

| Demographic Variable | Group | | P value |
|----------------------|-----------------------------------|------------------------|---------|
| | Delayed Lactation (Mean \pm sd) | Normal (Mean \pm sd) | |
| Age | 28.63 \pm 4.674 | 24.83 \pm 4.079 | <0.0001 |
| Calorie | 2723.77 \pm 456.680 | 1852.99 \pm 320.404 | <0.0001 |

Table 3: Association between Socio Economic Status: Group

| Socio-Economic Status | Delayed Lactation (n, %) | Normal (n, %) | Total |
|-----------------------|--------------------------|---------------|-------|
| UPPER | 5 (2.5%) | 0 (0.0%) | 5 |
| UPPER MIDDLE | 64 (32.0%) | 5 (2.5%) | 69 |
| LOWER-MIDDLE | 66 (33.0%) | 31 (15.5%) | 97 |
| UPPER-LOWER | 64 (32.0%) | 126 (63.0%) | 190 |
| LOWER | 1 (0.5%) | 38 (19.0%) | 39 |
| Total | 200 (100%) | 200 (100%) | 400 |

Among the study participants (n = 400), the majority were non-vegetarian (336, 84.0%), while vegetarians accounted for 64 (16.0%) of the participants. [Table 1]

Age vs Group: In the delayed lactation group, the mean age (mean \pm s.d.) of patients was 28.63 \pm 4.674 years. In the normal lactation group, the mean age (mean \pm s.d.) of patients was 24.83 \pm 4.079 years.

The distribution of mean age with lactation status group was statistically significant (p < 0.0001). [Table 2]

In our study, a highly significant association was observed between socioeconomic status (SES) and Group (p < 0.0001). The table demonstrates a significant association between socio-economic status and delayed onset of lactation. Among mothers with delayed lactation, the majority belonged to the lower-middle (33.0%), upper-middle (32.0%), and upper-lower (32.0%) socio-economic classes, whereas very few were from the upper (2.5%) and lower (0.5%) classes. In contrast, mothers with normal lactation were predominantly from the upper-lower socio-economic group (63.0%), followed by the lower (19.0%) and lower-middle (15.5%) classes, with minimal representation from the upper socio-economic strata.

The Chi-square analysis revealed a highly statistically significant difference (p < 0.001), indicating that socio-economic status plays an important role in the occurrence of delayed lactation.

Delayed lactation was more common among mothers belonging to middle socio-economic groups, whereas normal lactation was more frequently observed in the lower socio-economic strata. [Table 3]

In the delayed lactation group, the mean calorie intake (mean \pm s.d.) of patients was 2723.77 \pm 456.680 kcal. In the normal lactation group, the mean calorie intake (mean \pm s.d.) of patients was 1852.99 \pm 320.404 kcal. The distribution of mean calorie intake with lactation status group was statistically significant (p < 0.0001). [Table 4]

DISCUSSION

In the present study, mothers with delayed lactation were significantly older than those with normal lactation (28.63 \pm 4.674 vs. 24.83 \pm 4.079 years; p < 0.0001). In addition, mean caloric intake was substantially higher in the delayed lactation group (2723.77 \pm 456.680 kcal) compared to the normal lactation group (1852.99 \pm 320.404 kcal; p < 0.0001). These findings indicate that advancing maternal age and higher caloric consumption are significantly associated with delayed onset of lactogenesis.

Maternal age has been consistently recognized as an important determinant of breastfeeding outcomes. Lactogenesis II is hormonally regulated and depends on the coordinated interaction between prolactin, insulin, cortisol, and the withdrawal of progesterone after placental delivery. Age-related changes in

endocrine responsiveness may influence mammary gland sensitivity to prolactin. Grattan DR described the complexity of the hypothalamic–prolactin axis and its modulation by metabolic and reproductive factors, highlighting how physiological adaptability may vary across maternal age groups.^[7] Older maternal age has also been associated with increased obstetric interventions and a higher prevalence of metabolic comorbidities such as obesity and insulin resistance, both of which are established risk factors for delayed lactogenesis.^[8,9]

The significantly higher caloric intake observed among mothers with delayed lactation warrants careful interpretation. While adequate nutrition is essential to support milk production, excessive caloric intake may reflect increased adiposity and underlying metabolic dysregulation rather than improved nutritional status. Emerging evidence suggests that maternal obesity and insulin resistance impair lactogenesis II through altered prolactin signalling and decreased mammary epithelial responsiveness to insulin.^[9,10] Insulin is now recognized as a critical lactogenic hormone that supports mammary differentiation and milk synthesis. Disruption of insulin signalling pathways can delay secretory activation even in the presence of normal or elevated prolactin levels.^[10]

Furthermore, metabolic stress associated with excess caloric intake may contribute to a pro-inflammatory state, adversely affecting mammary gland function. Clinical practice guidelines from the Endocrine Society emphasize the close interrelationship between prolactin physiology and metabolic homeostasis, including glucose–insulin dynamics.^[11] Therefore, higher caloric intake in the delayed lactation group may represent a surrogate marker of metabolic imbalance rather than a direct causal factor.

The highly significant *p* values (<0.0001) observed for both variables strengthen the statistical association; however, causality cannot be inferred due to the observational design of the study. It is possible that maternal age and caloric intake interact with other determinants such as parity, body mass index, lipid profile, and serum prolactin levels. Multivariate modelling would help determine whether these variables independently predict delayed lactation.

From a public health perspective, these findings are particularly relevant in the Indian context, where delayed initiation of breastfeeding remains a concern despite strong national promotion of early breastfeeding practices. Antenatal counselling should emphasize not only adequate nutrition but also metabolic optimization, especially among older mothers. Screening for metabolic risk factors during pregnancy may facilitate early identification of women at risk for delayed lactogenesis.

In conclusion, advancing maternal age and higher caloric intake are significantly associated with delayed lactation in this cohort. The findings support the growing body of evidence linking metabolic

health to lactogenesis and underscore the need for integrated nutritional and endocrine assessment in perinatal care.

The present study demonstrates a significant association between socio-economic status (SES) and delayed lactation, with a higher proportion of delayed lactation observed among mothers belonging to the upper-middle (32.0%) and lower-middle (33.0%) socio-economic groups. In contrast, normal lactation was more frequent among mothers in the upper-lower (63.0%) and lower (19.0%) categories. From an endocrine perspective, these findings suggest that socio-economic variations in delayed lactation may be mediated through differences in metabolic and hormonal determinants rather than economic deprivation per se.

Lactogenesis II is a hormonally regulated process that depends on the coordinated action of prolactin, insulin, cortisol, and thyroid hormones following the withdrawal of progesterone after placental delivery. Disruption of this endocrine balance can delay secretory activation. Grattan DR emphasized the complex regulation of the hypothalamo–prolactin axis and its sensitivity to metabolic and physiological stressors.^[7,12] Altered prolactin dynamics, reduced receptor sensitivity, or impaired downstream signaling can result in delayed lactogenesis even when circulating prolactin levels appear adequate.

The higher prevalence of delayed lactation in middle and relatively affluent socio-economic groups may reflect a greater burden of metabolic risk factors such as obesity, insulin resistance, and dyslipidemia, conditions increasingly prevalent in urban and middle-income Indian populations. Insulin plays a permissive and essential role in mammary gland differentiation and milk synthesis. Impaired insulin signaling has been shown to delay lactogenesis II. Nommsen-Rivers et al. demonstrated that poor antenatal metabolic health predicts delayed onset of lactation,^[8,13] while subsequent work highlighted the role of insulin resistance in mediating suboptimal prolactin action at the mammary epithelial level.^[9,14] Thus, endocrine-metabolic dysfunction may be a key pathway linking SES and delayed lactation.

Furthermore, stress-related endocrine alterations may contribute to delayed lactogenesis. Women in higher SES groups may experience occupational stress, delayed initiation of breastfeeding due to elective cesarean sections, and separation from the neonate, all of which can blunt oxytocin release and interfere with prolactin pulsatility. Cesarean delivery, more common in middle- and upper-income strata, has been independently associated with delayed lactogenesis due to altered neuroendocrine responses and delayed skin-to-skin stimulation.^[10,15]

Conversely, mothers from lower socio-economic strata in this study demonstrated relatively higher rates of normal lactation. Early and frequent breastfeeding—more common in traditional settings—stimulates prolactin secretion through neuroendocrine reflex pathways and enhances timely secretory activation. The World Health Organization

recommends initiation of breastfeeding within one hour of birth precisely because early suckling optimizes prolactin and oxytocin release, reinforcing endocrine mechanisms essential for lactogenesis.

It is also important to recognize that endocrine disorders such as thyroid dysfunction and hyperprolactinemia may influence lactation outcomes. Clinical practice guidelines from the Endocrine Society highlight the close interaction between prolactin physiology and metabolic homeostasis, including insulin sensitivity and adipokine signalling.^[11,16] Socio-economic transitions associated with urbanization may therefore increase exposure to endocrine and metabolic disturbances that adversely affect lactogenesis.

While socio-economic disadvantage is traditionally linked with undernutrition, the present findings suggest that endocrine-metabolic imbalance associated with lifestyle transitions may exert a stronger influence on delayed lactation than caloric insufficiency alone. However, SES likely interacts with multiple confounders including maternal age, body mass index, parity, and mode of delivery. Multivariate endocrine profiling would be necessary to establish independent predictive pathways.

In conclusion, the association between socio-economic status and delayed lactation observed in this cohort appears to be mediated largely through endocrine and metabolic mechanisms. Middle socio-economic groups may be particularly vulnerable due to a higher prevalence of insulin resistance, obesity, obstetric interventions, and stress-related hormonal alterations. These findings underscore the need for integrated metabolic and endocrine assessment during antenatal care to identify mothers at risk for delayed lactogenesis and to implement early preventive strategies.

CONCLUSION

The present study demonstrates a significant association between delayed initiation of lactation and advancing maternal age, higher caloric intake, and socio-economic status. Mothers belonging to middle socio-economic strata exhibited a higher prevalence of delayed lactogenesis compared to those from lower strata. The observed association between increased caloric intake and delayed lactation suggests that metabolic imbalance, rather than caloric insufficiency, may contribute to impaired secretory activation. Endocrine-metabolic factors, particularly altered insulin sensitivity and prolactin responsiveness, may represent key underlying mechanisms.

These findings underscore the importance of comprehensive antenatal assessment that includes metabolic risk stratification alongside routine

obstetric care. Strengthening early breastfeeding support, promoting timely skin-to-skin contact, and reinforcing early initiation practices in accordance with World Health Organization recommendations are essential. Integrating nutritional optimization with endocrine evaluation may help identify mothers at risk for delayed lactogenesis and improve early breastfeeding outcomes and neonatal health indicators in socio-economically transitioning populations.

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