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Parental Biomedical Manifestations and Socioeconomic Status Influence on Newborn Telomere Length

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Abstract

***Background*:** Human biology, even inside the womb, is not safe from the environment and can leave parental epigenetic marks on the newborn**.** However, multiple risk factors that influence the telomere biology of the fetus are not fully known. This study aimed to determine the influence of parents' socioeconomic status (SES) and its related biomedical manifestations on newborn telomere length.

***Methods*:** A total of 204 parent-newborn pairs were recruited in this study. Blood samples were obtained after informed consent, and demographic and environmental risk factors such as socioeconomic status, education and exposure to endemic disease were recorded. The Leukocyte Telomere Length (TL) quantification was done using qPCR and T/S ratio was calculated. For the association between paternal and newborn TL and different variables, multivariate regression analysis was utilized. The results with $p<0.05$ were considered Statistically significant.

***Results*:** The mean age (years) of mothers and fathers was 27 ± 5.12, 34 ± 6.36. Parents with an increase in age have a positive association with newborns TL (T/S ratio) (2.31 $\pm$ 1.45) (p=0.034) *(*mother: β 0.032, p=.009, father: $\beta=0$.043, p=0.099). The low SES and blue color occupational group showed significantly smaller TL in parents-newborns (1.5 $\pm$ 1.14, 1.41 $\pm$ 1.08, 1.95 $\pm$ 1.36) ($p<0.05$). Moreover, parents with environmentally imposed non-communicable diseases like GDM/Diabetes/hypertension had shorter LTL (1.54 $\pm$ 1.37, 1.32 ± 1.1) than their newborns (2.36 $\pm$ 1.43) (p=0.048) (Hypertension: β = -0.785, p = 0.045, COVID: β = -0.791, p=0.069). Most diseases were seen in the high SES (94.1%, 60.8%) followed by the upper middle, low middle and low SES, respectively.

***Conclusion*:** The parents' socioeconomic status and its related biomedical manifestations can reprogram the telomere biology of the newborn.

Keywords: Telomere, newborn, socioeconomic status, reprogramming.

1. INTRODUCTION

The parental genes are transmitted to the newborn along with their epigenetic changes. Since DNA is continuously replicating, a complex interplay between genes and the environment in utero can manipulate telomere biology, which can be assessed by studying the sequences of markers of aging [1]. However, how these factors genetically influence the telomere biology of the growing fetus is not fully known.

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The womb is the haven for the fetus. It is supposed to guard it against physical insult, socioeconomic status or nutrient supply insults by filtering through placental barriers. These perinatal factors like genetics and lifestyle play a role in determining Telomere length (TL) and its biology, which may differ in telomere length expression among different cells [2].

Prenatal stress and environmental influences can cause telomere shortening, profoundly affecting aging and overall quality of life [3]. Factors such as reactive oxygen species (ROS), inflammation, education, physical activity, and smoking can induce DNA damage and accelerate telomere attrition. Adverse conditions during

pregnancy, including psychosocial stress, low socioeconomic status, lack of social support, and childhood trauma, create an unfavorable intrauterine environment [4].

According to the literature, telomeres and telomerase are strongly associated with the risk of various diseases, including upper respiratory tract infections in children [5], malignant tumors [6], cardiovascular disease [7], chronic obstructive pulmonary disease (COPD) [8], diabetes [9], and metabolic disorders [10]. Although telomere biology plays a vital role in the development of age-related diseases and premature aging, these findings may not be universally generalizable to the broader population [11].

The research data regarding the impact of environmentally induced factors, viral diseases, or ailments such as COVID-19 on Leukocyte telomere length is scarce in the region of Pakistan.

Following our previous research [12], this study builds on the knowledge of the association between newborn TL reprogramming and parental socioeconomic status and its related biomedical manifestations. Thus, this study is designed to determine the influence of SES and its related biomedical manifestations of parents on newborn TL.

2. METHODOLOGY

A total of 204 parent-newborn pairs (n = 612) were enrolled in the study after obtaining ethical approval from the Ethics Review Committee of Ziauddin University and Hospitals (Ref No. 3950721SFBC). Between September 2021 and June 2022, samples were collected with informed consent from all parents. The recruited females were aged 18–35 years, and males were 18–45 years. The demographics and disease status of parents and newborns were documented through hospital records and questionnaire responses.

Socioeconomic status (SES) was categorized as low (< $98), lower middle ($99–735), upper middle (>$735–1961), and high (> $1961) based on income, using the World Bank classification [13, 14] (exchange rate: Rs. 204/USD as of July 1, 2022). Endemic non-communicable diseases (Gestational Diabetes Mellitus (GDM)/Diabetes, Hypertension) and viral diseases (COVID-19, Dengue) were also recorded.

After receiving informed consent from both parents, 5 mL of venous blood was collected from each parent into EDTA tubes. For newborns, 5 mL of venous blood was drawn from the umbilical cord into EDTA tubes. DNA was extracted using the Qiagen DNA Blood Mini Kit (catalog number 51306, Germany). Leukocyte telomere length (LTL) was measured by qPCR using the Cawthon multiplex method [15], with beta-globin (a single-copy gene) used as the reference. The qPCR reaction, programming, and calculation of the telomere/single-copy gene (T/S) ratio were performed according to previously published methods [16].

SPSS and GraphPad Prism Software were used for data analysis. Qualitative variables were reported as frequencies and percentages, and quantitative variables as means ± standard deviations (SD). Multivariate regression analysis was applied to examine associations between parental-newborn TL (T/S ratio) and various variables. A p-value < 0.05 was considered statistically significant.

3. RESULTS

A total of 204 parent-newborn pairs were included, with the mean (± SD) maternal and paternal ages being 27 ± 5.12 years and 34 ± 6.36 years, respectively.

Parents younger than 25 years exhibited longer telomere lengths (T/S ratio: 1.53 ± 1.18 for mothers, 1.73 ± 1.14 for fathers) compared to those older than 30 years. However, newborns of younger parents had shorter telomere lengths (1.85 ± 1.39) compared to newborns of older parents (2.46 ± 1.2) ($p < 0.05$).

Demographic analysis showed longer newborn TL across all SES categories (low: 1.95 ± 1.36, lower middle: 2.12 ± 2.09, upper middle: 2.76 ± 2.17, high: 2.55 ± 2.21). Among mothers, those from the upper-middle SES had longer TL (2.21 ± 2.12), whereas fathers from the low SES group had longer TL (1.75 ± 1.08) compared to those from the high SES group ($p = 0.000$).

Parents with postsecondary education demonstrated longer TL (mothers: 1.88 ± 1.35; fathers: 1.69 ± 1.11), and their newborns also exhibited longer TL (2.62 ± 1.44) ($p = 0.08$). White-collar occupational groups showed significantly longer TL in parents and newborns (2.35 ± 1.20, 1.86 ± 1.24, and 1.04 ± 1.01, respectively) ($p = 0.001$) (Table **1**).

Parents with non-communicable diseases such as GDM/Diabetes had shorter LTLs (1.54 ± 1.37 for mothers, 1.32 ± 1.10 for fathers) compared to their newborns (2.36 ± 1.43) ($p = 0.048$) (Table **1**). A significant difference ($p = 0.045$) was observed in viral disease (COVID-19) comparisons, where mothers had shorter LTLs (0.98 ± 0.81) compared to fathers and newborns (Table **1**).

Regression analysis revealed a positive association between maternal ($\beta = 0.032$, $p = 0.009$) and paternal age ($\beta = 0.043$, $p = 0.099$) and newborn LTL. SES also showed a positive trend ($\beta = 0.256$, $p = 0.059$). Conversely, the presence of hypertension ($\beta = -0.785$, $p = 0.045$) and COVID-19 ($\beta = -0.791$, $p = 0.069$) was negatively associated with newborn TL.

Table 1: Mean difference between T/S ratio and environmental influence on parents and their newborns.

Variables		Mother (n=204)		Father (n=204)		Newborn (n=204)	p-value
		n (%)	TL (T/S Ratio) (Mean+ SD)	*n* (%)	TL (T/S Ratio) (Mean+ SD)	TL (T/S Ratio) (Mean+ SD)	
Age	<20	21 (10)	1.93± 1.18	4 (2)	1.73 ± 1.14	1.85 ± 1.39	0.034*
	20-30	119 (58)	1.72 ± 1.30	53 (28)	1.57 ±1.12	2.31 ± 1.45	
	>30	64(32)	1.34± 1.02	133(70)	1.42 + 1.13	2.46 + 1.2	
Socioeconomic Status	Low	51(25)	1.76± 1.11	51(25)	1.75± 1.08	1.95 ± 1.36	0.00*
	Lower middle	51(25)	1.29± 2.3	51(25)	1.73 ±1.95	2.12 ±2.09	
	Upper middle	51(25)	2.21 ±2.12	50(24)	1.69± 1.39	2.76± 2.17	
	High	51(25)	1.73 ± 1.37	52(26)	1.70 ±1.12	2.55 ± 2.21	
Education	Presecondary and secondary	121(59)	1.55 ± 1.20	107(56)	1.38 ±1.08	1.92 + 1.41	0.080
	Postsecondary	83(41)	1.88 ± 1.35	83(44)	1.69 +1.11	2.62 ± 1.44	
Occupation	Blue Collar	184(90)	1.75 ± 1.26	147(77)	1.66 ± 1.09	2.04 ± 1.47	0.001*
	White Collar	20(10)	1.86 ± 1.24	43(33)	1.04 ± 1.01	2.35 ± 1.20	
Diseases	Non-communicable Diseases	174(85)	1.54 ± 1.37	178 (87)	1.32±1.1	2.32± 1.43	0.048*
	Viral Diseases	30(15)	0.98 ± 0.81	26 (13)	1.18±0.98	2.2 ± 1.47	0.045*

**P value 0.05 significance level*
Non-communicable Diseases: GDM/Diabetes, Hypertension
Viral diseases: COVID, Dengue

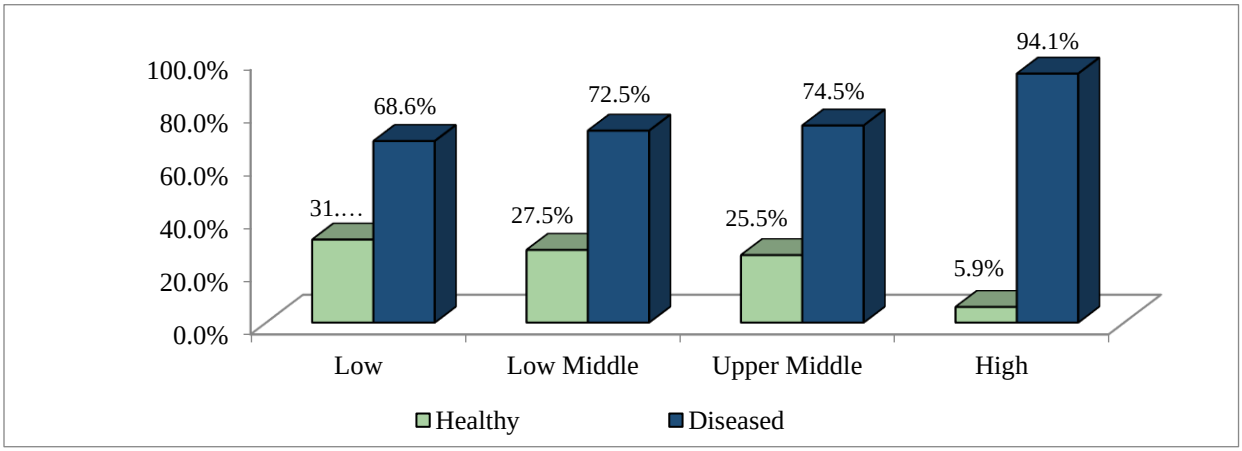

Figure 1: Disease status of mother with respect to SES. High SES shows high prevalence of disease than healthy mothers.

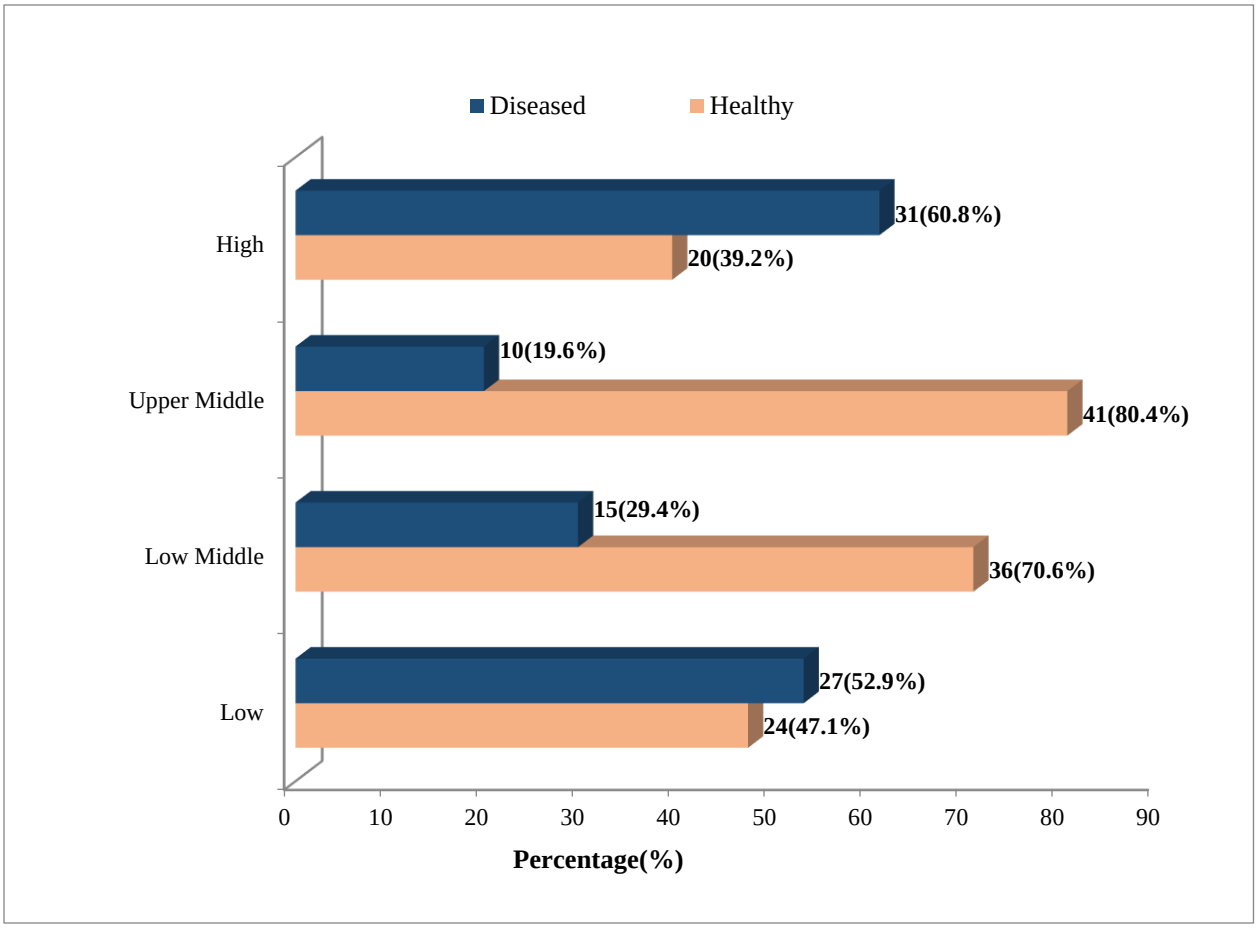

Figure 2: Disease status of a father with respect to SES. High SES shows higher prevalence of disease than healthy fathers.

The distribution of maternal diseases across SES categories revealed the highest prevalence among the high SES group (94.1%), followed by the upper-middle (74.5%), lower-middle (72.5%), and low SES groups (68.6%) (Fig. **1**). A similar trend was seen in fathers, with the highest prevalence in the high SES group (60.8%), followed by low (52.9%), lower-middle (29.4%), and upper-middle SES (19.6%) groups (Fig. **2**).

4. DISCUSSION

Despite extensive data on accelerated telomere shortening due to adverse effects of social status, environmental pollutants, and occupational exposures, the relationship between these factors and telomere length remains inconsistent [16]. This study investigates telomere length (TL) and the effects of age, socioeconomic status (SES), occupation, environmentally induced diseases, and viral infections, highlighting how these insults impact newborn telomere genetics and suggest cellular aging due to oxidative stress responses [7, 17].

In the current study, the disease statuses of parents were assessed, showing the highest prevalence among the high SES group (94.1% for mothers and 60.8% for fathers with at least one disease), followed by other SES categories. This finding contrasts with a previous study, which indicated that participants with high SES were more likely to maintain physical inactivity and continue smoking (OR 2.08 [1.14–3.80]), behaviors that may contribute to the development of non-communicable diseases [18]. Another study reported that higher life-course SES in both men and women is associated with an increased risk of overweight and obesity. Furthermore, in middle-income countries (China, Ghana, India, Mexico, South Africa, and Russia), higher SES is linked to greater odds of diabetes and hypertension in men [19]. These findings signal an alarming trend for lower-middle-income countries, emphasizing the need to investigate the emerging health risks associated with higher SES.

Moreover, among all SES categories, parents from the upper-middle SES group had newborns with longer telomere lengths (2.76 ± 2.17) ($p = 0.00$). This

observation is supported by our previous study, which also showed a statistically significant association ($p < 0.05$) between newborn TL and parents' upper-middle SES [12, 20]. However, other studies have reported mixed results, showing either shorter or longer TL in relation to SES [21]. Additionally, shorter TL in fathers from upper-middle and high SES groups, as observed in this study, is consistent with findings from other research [22], suggesting an increased risk of developing diseases earlier in life [23, 24].

Thus, telomere length detection at birth or early in life may serve as a valuable indicator, revealing associations with future disease risk and aging processes that begin early and continue into adulthood.

In this study, we found a significant effect of maternal age on the telomeres of the newborn, with a negative association ($\beta = -0.144$, $p = 0.049$). Although many researchers have studied maternal age at last childbirth in relation to maternal longevity and found a positive association [25, 26], similar to the current study, Fagan *et al*. observed that women who delivered a child after the age of thirty-three were 2–3 times more likely to have newborn telomere lengths in the second (4947.62–5217.28 bp) and third tertiles (5217.29–7715.65 bp) compared to the first tertile (4484.6–4947.61 bp) [27]. Therefore, maternal age at childbirth may be considered a marker for longevity [28].

Among diseases (GDM/Diabetes and Hypertension), shorter TL (1.54 ± 1.37, 1.32 ± 1.1) ($p = 0.048$) in parents compared to their newborns (2.32 ± 1.43) was observed in this study. The effect of disease on TL may be due to increased oxidative stress, causing impaired glucose regulation in dysglycemia, especially in GDM. These findings suggest that diseases associated with shorter LTL cause reduced replicative potential and impaired repair ability [29]. Furthermore, a relationship with other diseases like COVID-19 was considered a major risk factor leading to adverse pregnancy outcomes. Current research, besides highlighting TL as a biomarker for biological aging or DNA damage, is exploring the modulating role of genetic and environmental factors on telomere length, linking it to cardiovascular disease, diabetes, and cancer [30-32].

The low socioeconomic population is more vulnerable to hazardous exposures due to unhealthy lifestyles, poor eating habits, lack of access to quality food, tobacco consumption, and insufficient physical activity [12]. Diabetic fathers had shorter TL compared to their age-matched peers with hypertension. The exact underlying mechanisms are not fully elucidated, but oxidative stress and metabolic toxins are contributing factors. These findings emphasize that fetal programming of telomere length can significantly contribute to health disparities and the lifespan of the affected population and their offspring.

The small sample size limits the significance of the results; however, this limitation can be addressed through cohort studies to identify all relevant risk factors more effectively.

5. CONCLUSION

Parental environmental stressors, particularly socioeconomic status (SES) and biomedical factors such as hypertension, can negatively impact newborns' telomere genetics. These influences may contribute to altered telomere length, potentially affecting cellular aging and disease susceptibility in early life, highlighting the significance of parental health in shaping genetic inheritance.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest.

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AUTHOR CONTRIBUTIONS

SF contributed to the study design, performed experiments, analyzed the data, and wrote the manuscript.

SB gave the main concept of the study, supervised the whole research and revised the manuscript.

The authors have read and agreed to the published version of the manuscript.

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