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The Impact of External Social and Lifestyle Factors on Psychological Status, Stress, and Premature Ageing: An Observational Study.

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Abstract:
Background: The pathogenesis of premature biological ageing and chronic stress is increasingly linked not only to genetic predispositions but to modifiable external social and lifestyle factors. This study investigates the correlation between specific lifestyle variables (sleep, diet, physical activity), social determinants (isolation, socioeconomic status, work environment), and their collective impact on psychological status, perceived stress, and markers of premature ageing.
Methods: A cross-sectional observational study was conducted among 500 urban adults aged 30-55. Lifestyle and social factors were assessed using validated self-report questionnaires, alongside the Perceived Stress Scale (PSS) and the General Health Questionnaire (GHQ-12) for psychological status. Biological ageing was estimated using non-invasive phenotypic markers (e.g., skin elasticity scores) and self-reported health metrics. Data were analyzed using multivariate logistic regression models.
Results: High social isolation was strongly correlated with elevated PSS scores ($p < 0.05$) and poorer psychological status on the GHQ-12 ($P<0.05$). Individuals in the lowest quartile for sleep quality exhibited a 3.2-fold increase in phenotypic markers of premature ageing compared to those in the highest quartile. Work-related stress emerged as a significant mediator between socioeconomic status and accelerated ageing markers.
Conclusion: Adverse social environments and poor lifestyle choices significantly contribute to chronic psychological stress, acting as catalysts in the pathogenesis of premature ageing. Interventions targeting social connectivity and sleep hygiene are critical for mitigating these effects.
Key words: Psychological Status, Stress, Perceived Stress Scale (PSS) and the General Health Questionnaire (GHQ-12)

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Introduction:

The modern urban environment presents a complex matrix of social and lifestyle factors that profoundly influence human biology and psychology. While chronological ageing is an inevitable biological process, "premature ageing"—where biological age exceeds chronological age—is increasingly recognized as a pathological state driven by chronic physiological and psychological stress. The pathogenesis of this accelerated decline is deeply intertwined with external variables, moving the focus of gerontological research beyond pure genetics toward modifiable environmental factors.[1]

Chronic psychological stress has been established as a primary driver of cellular ageing, notably through mechanisms such as telomere shortening, chronic low-grade inflammation ("inflammageing"), and oxidative stress. However, the specific social and lifestyle precursors that induce this chronic stress state require detailed quantification.[2]

This study aims to address the critical gap in understanding how specific external factors—namely social isolation, high-pressure work environments, sleep deprivation, and sedentary behaviors—cumulatively affect psychological status and contribute to the premature ageing phenotype. We hypothesize that individuals reporting higher levels of social isolation and poorer lifestyle metrics will exhibit significantly higher levels of perceived stress and an increased presence of premature ageing markers.[3]

2. Methodology

1. Study Design and Participants

This cross-sectional observational study recruited 500 adults (aged 30-55 years) residing in a major metropolitan area. Recruitment was conducted via stratified random sampling using digital community boards and local health clinics.

- **Inclusion Criteria:** Adults aged 30-55, residing in the urban center for at least 5 years, fluent in the primary language of the assessment tools.
- **Exclusion Criteria:** Individuals with diagnosed severe psychiatric disorders (e.g., schizophrenia), known genetic progeria syndromes, or those currently undergoing treatments known to significantly alter biological ageing (e.g., chemotherapy).

2 Measures and Instruments

Assessment of External Social Factors:

- **Social Isolation:** Measured using the Lubben Social Network Scale (LSNS-6), assessing family and friend networks.
- **Work Environment:** Assessed via the Job Content Questionnaire (JCQ), focusing on psychological demands and decision latitude.
- **Socioeconomic Status (SES):** Calculated using a composite index of education level, household income, and occupation.

Assessment of Lifestyle Factors:

- **Sleep Quality:** The Pittsburgh Sleep Quality Index (PSQI) was utilized.
- **Physical Activity:** Measured using the International Physical Activity Questionnaire (IPAQ).
- **Dietary Habits:** Assessed via a validated Food Frequency Questionnaire (FFQ) focusing on the intake of ultra-processed foods versus whole foods.

Assessment of Psychological Status and Stress:

- **Perceived Stress:** The Perceived Stress Scale (PSS-10) quantified the degree to which situations in life are appraised as stressful.
- **General Psychological Status:** The General Health Questionnaire (GHQ-12) was used to screen for general (non-psychotic) psychiatric morbidity and emotional distress.

Assessment of Premature Ageing Markers:

Due to the observational field nature of this study, non-invasive phenotypic and self-reported markers were utilized as proxies for biological age, including:

- Standardized dermatological assessments of skin elasticity and wrinkling (evaluated against chronological age norms).
- Self-reported functional mobility assessments.
- Frequency of medically diagnosed age-related comorbidities (e.g., early-onset hypertension, pre-diabetes).

3. Statistical Analysis

Data were analyzed using SPSS Version 28. Descriptive statistics summarized demographic characteristics. Bivariate correlations (Pearson's r) were initially calculated to identify potential relationships. To control for confounding variables (age, gender, BMI), multivariate logistic regression models were employed to ascertain the independent effects of social and lifestyle factors on stress, psychological status, and ageing markers. Significance was set at . $\alpha=0.05$

Observations and Results

Demographics and Baseline Characteristics

The final cohort consisted of 482 participants (after excluding incomplete responses), with a mean age of 42.5 ± 7.1 years. The sample was 54% female. Detailed demographics and baseline lifestyle characteristics are summarized in Table 1.

Table 1: Baseline Demographic and Lifestyle Characteristics of the Study Population (N = 482)

Characteristic	**Total Sample (N = 482)**
Age (years), Mean ± SD	42.5 ± 7.1
Gender, n (%)	
Female	260 (54.0%)
Male	222 (46.0%)
Socioeconomic Status (SES), n (%)	
Low	115 (23.9%)
Middle	245 (50.8%)
High	122 (25.3%)
Poor Sleep Quality (PSQI > 5), n (%)	185 (38.4%)
High Social Isolation (LSNS-6 < 12), n (%)	134 (27.8%)
Sedentary Lifestyle (IPAQ), n (%)	201 (41.7%)

Impact of Social Factors on Psychological Status

Analysis revealed a robust correlation between social connectivity, work environment, and psychological well-being (Table 2). Participants scoring below 12 on the LSNS-6 (indicating high social isolation) had significantly higher mean PSS scores (22.4 ±4,1) compared to socially integrated peers (14.2 ±38) ($p<0.001$). High job strain (high demand/low control) was similarly predictive of poorer psychological status on the GHQ-12.[4]

Table 2: Bivariate Correlations between Social/Lifestyle Factors and Psychological/Ageing Metrics

Variable	Perceived Stress (PSS-10)	General Psych Status (GHQ-12)	Phenotypic Ageing Score
Social Isolation (LSNS-6)	r = -0.45	r = -0.38	r = -0.29
Job Strain (JCQ)	r = 0.52	r = 0.47	r = 0.34
Sleep Quality (PSQI)	r = 0.61	r = 0.55	r = 0.68
Sedentary Behavior (IPAQ)	r = 0.31	r = 0.28	r = 0.42

*Note: For the LSNS-6,lower scores indicate higher isolation, hence the negative correlation with stress metrics $p<0.005$, $p<0.001$ **

Lifestyle Factors and Premature Ageing Markers

Sleep quality emerged as the most critical lifestyle factor associated with premature ageing. As detailed in our regression model (Table 3), individuals with a PSQI score >5 (indicating poor sleep quality) were significantly more likely to exhibit advanced phenotypic ageing markers compared to chronological peers.

Table 3: Multivariate Logistic Regression Analysis for Predictors of Advanced Phenotypic Ageing

Predictor Variable	Odds Ratio (OR)	95% CI	p-value
Poor Sleep Quality (PSQI > 5)	3.24	2.15 - 4.88	< 0.001
High Social Isolation (LSNS-6 < 12)	2.41	1.62 - 3.59	< 0.01
High Job Strain (JCQ)	1.88	1.21 - 2.92	0.02
Low Socioeconomic Status (SES)	1.65	1.10 - 2.47	0.04
Sedentary Lifestyle (IPAQ)	1.42	1.05 - 1.92	0.05

Note: Adjusted for confounding variables including Age, Gender, and BMI.

Sedentary behavior (as measured by the IPAQ) showed a moderate correlation with an increased incidence of early-onset comorbidities, though this effect was partially mitigated when controlling for dietary factors.

The Interplay of Stress and Ageing

A mediation analysis indicated that perceived stress (PSS scores) significantly mediated the relationship between external social factors (SES, isolation) and markers of premature ageing. For example, the effect of low SES on skin elasticity scores was reduced by 40% when PSS scores were included in the model, suggesting that the physiological damage of low SES is largely driven by the psychological stress it induces.[5]

Discussion

The primary objective of this observational study was to quantify the extent to which specific external social and lifestyle factors contribute to chronic psychological stress and, subsequently, the pathogenesis of premature ageing. Our findings robustly support the hypothesis that adverse social environments and suboptimal lifestyle habits are not merely peripheral influences but act as central drivers in the acceleration of biological ageing processes.[6] By isolating these factors within a relatively young, urban demographic (30-55 years), we observe the early pathogenesis of stress-induced physiological decline before the onset of typical geriatric comorbidities.

The Biological Implications of Social Isolation

Our analysis revealed that social isolation (as measured by the LSNS-6) is one of the strongest social predictors of elevated perceived stress (PSS-10) and poorer general psychological status (GHQ-12). This aligns with the "social baseline theory," which posits that humans are evolutionarily adapted to function within social networks, and isolation signals an environmental threat to the nervous system.[7-9]

Biologically, the chronic hyper-arousal observed in our socially isolated participants likely translates to sustained dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. Prolonged elevation of glucocorticoids (primarily cortisol) is a known catalyst for accelerated cellular ageing. Previous in vitro and cohort studies have demonstrated that chronic cortisol exposure accelerates telomere attrition and inhibits telomerase activity. While our study utilized phenotypic proxies rather than cellular assays, the strong correlation between isolation, stress, and visible ageing markers (such as decreased skin elasticity and early onset comorbidities) strongly suggests this physiological cascade is occurring within our sample. This highlights social isolation not merely as a psychological burden, but as a systemic physiological toxin.[10-12]

Work-Related Stress and Allostatic Overload

The significant correlation between high job strain (high demand/low control, per the JCQ) and premature ageing markers provides real-world evidence of "allostatic overload." Allostatic load refers to the cumulative wear and tear on biological systems resulting from chronic overactivity or underactivity of systems that adapt to environmental challenges.

Our multivariate regression (Table 3) showed that high job strain increased the odds of exhibiting advanced phenotypic ageing by nearly two-fold (OR = 1.88). The modern work environment often requires sustained

psychological vigilance without physical exertion, leading to chronic sympathetic nervous system activation. This state promotes low-grade systemic inflammation, often termed "inflammageing." Inflammageing is a cornerstone in the pathogenesis of structural tissue degradation and metabolic dysfunction, which manifests as the premature ageing phenotype observed in our cohort. Furthermore, our mediation analysis suggested that the negative impact of lower Socioeconomic Status (SES) on ageing was largely driven by the specific types of high-strain, low-control environments these individuals are disproportionately exposed to.[13-16]

Sleep as a Primary Mediator of Cellular Homeostasis

Perhaps the most striking finding in our study was the overwhelming impact of sleep quality. Poor sleep (PSQI > 5) emerged as the single most significant lifestyle predictor of advanced phenotypic ageing (OR = 3.24).[17]

The biological mechanisms underpinning this are likely multifaceted. Sleep is a critical period for cellular repair, the clearing of metabolic waste (particularly neurotoxic waste via the glymphatic system), and immune system regulation. Chronic sleep deprivation disrupts circadian rhythms, which regulate a vast array of metabolic and hormonal processes. Disrupted circadian rhythms have been shown in animal models to accelerate oxidative stress and impair DNA repair mechanisms—both of which are hallmarks of biological ageing.

Our findings suggest that poor sleep may act synergistically with social stress. For instance, individuals experiencing high job strain or social isolation may suffer from subsequent insomnia or fragmented sleep, thereby removing the biological "recovery period" necessary to heal the cellular damage caused by daytime stress. This creates a vicious cycle where psychological stress degrades sleep, and poor sleep diminishes psychological resilience, rapidly accelerating the ageing process.[18-19]

Sedentary Behavior and Metabolic Ageing

While less impactful than sleep or severe social isolation, sedentary behavior (IPAQ) showed a moderate correlation with premature ageing ($p = 0.05$). Lack of physical activity contributes directly to metabolic dysregulation, including insulin resistance and loss of muscle mass (sarcopenia), which are central features of the ageing phenotype. The fact that this effect was partially mitigated when controlling for diet suggests that the synergistic effect of poor diet and lack of movement is a primary driver of the early-onset comorbidities noted in our sedentary participants.[20]

Limitations and Future Directions

While the findings are robust, several limitations must be acknowledged.

1. **Cross-Sectional Design:** As an observational, cross-sectional study, we cannot definitively establish causality. Bidirectional relationships are highly likely; for example, while poor sleep may cause psychological distress, existing psychological distress undoubtedly causes poor sleep. Longitudinal studies following cohorts over decades are required to parse the exact temporal sequence of pathogenesis.
2. **Self-Report Bias:** Reliance on self-reported questionnaires (PSQI, IPAQ,

LSNS-6) introduces the potential for recall bias or social desirability bias.

3. **Phenotypic Proxies:** We relied on phenotypic markers and health histories as proxies for biological age. Future studies should incorporate direct cellular assays, such as epigenetic clocks (e.g., Horvath's clock) or leukocyte telomere length, to provide a more precise quantification of biological ageing at the molecular level.

Despite these limitations, this study provides compelling evidence that the "exposome"—the totality of environmental exposures including social and lifestyle factors—is a critical determinant of how rapidly we age.

Conclusion

External social environments and lifestyle choices are not merely correlative factors but act as active agents in the pathogenesis of premature ageing and psychological distress. Chronic stress serves as the primary biological transducer, converting social isolation and poor lifestyle habits—particularly sleep deprivation—into accelerated physical and mental decline. Public health strategies must prioritize interventions that foster social connectivity and improve lifestyle hygiene as critical preventative measures against the rising tide of stress-induced premature ageing in urban populations.

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