Original Article

Premature Cardiovascular Disease in Brazil: What Explains This Phenomenon?

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Abstract

Background: **Hospitalization rates for acute myocardial infarction (AMI) have been rising among young Brazilian adults, despite the overall decline in cardiovascular mortality. However, the contribution of modifiable risk factors remains insufficiently characterized.**

Objectives: **To assess the temporal trend of AMI hospitalizations among individuals aged 35 to 44 years in Brazil (2008–2024) and to identify potential risk factors correlated with this trend.**

Methods: **This ecological time-series study used data from SIH-SUS and prevalence estimates from Vigitel (2008–2023, excluding 2022). Pearson and Spearman correlations, multiple linear regression, and annual percent change (APC) were employed. The significance level was set at 5% ($p < 0.05$).**

Results: **The rate of AMI hospitalizations increased 82.3% (16.49 to 30.07/100,000; APC: +3.20%/year; 95% confidence interval: 2.65–3.75; $p < 0.001$), accelerating from +2.17%/year (2008–2016) to +7.48%/year (2020–2023). Obesity showed the strongest correlation (Pearson $r = 0.872$; Spearman $\rho = 0.946$; $p < 0.001$). In the regression model ($R^2 = 0.906$; $p < 0.001$), obesity ($\beta = 1.10$; $p = 0.001$) and abusive alcohol consumption ($\beta = 0.53$; $p = 0.037$) were significant predictors. Smoking declined 37% without a concomitant reduction in AMI.**

Conclusions: **Obesity was the risk factor most strongly correlated with the increase in AMI among young Brazilian adults, representing the main hypothesis to be confirmed in individual-level studies.**

Keywords: **Myocardial Infarction; Heart Disease Risk Factors; Epidemiology; Obesity; Brazil.**

Introduction

Cardiovascular diseases remain the leading cause of morbidity and mortality in Brazil and worldwide.[1,2] Although a global decline in cardiovascular mortality rates has been observed in older age groups, recent data have indicated an upward trend in the incidence of acute myocardial infarction (AMI) among young adults.[3-5]

The 35-to-44-year age group warrants particular attention, as it corresponds to the period of peak economic productivity. Premature cardiovascular disease in this population has a disproportionately high socioeconomic impact.[6,7]

At the same time, the profile of cardiovascular risk factors in Brazil is undergoing changes. The prevalence of obesity among individuals aged 35 to 44 years increased by 77.6% between 2008 and 2023. The prevalence of diabetes increased by 61.8%, and abusive alcohol consumption increased by 28.0%. Conversely, smoking rates declined by 37.0%.[8]

The relative contribution of each modifiable risk factor to the rise in infarction rates within this age group has not been adequately quantified in Brazilian literature, representing a significant scientific gap in the evidence base for informing public prevention policies.

The primary objective of this study was to assess the temporal trend in AMI hospitalization rates among individuals aged 35 to 44 years in Brazil between 2008 and 2024, as shown in the Central Illustration. Secondary objectives included quantifying correlations between risk factors and hospitalization rates, exploring the relative contribution of each factor, and characterizing the annual percent change (APC).

Methods

This was an ecological time-series study reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines

Data on hospitalizations for AMI were obtained from the Hospital Information System of the Brazilian Unified Health System (SIH-SUS),[9] considering admissions recorded

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Central Illustration: Premature Cardiovascular Disease in Brazil: What Explains This Phenomenon?

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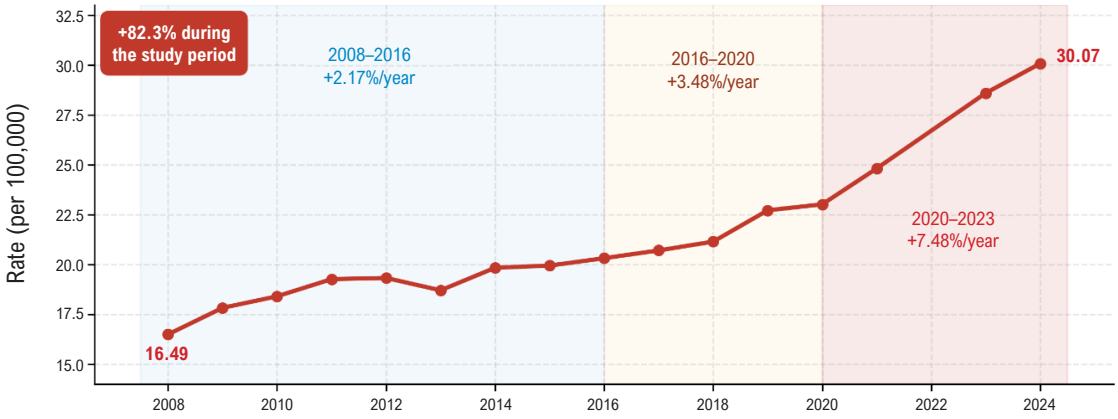
Premature cardiovascular disease in Brazil (35 to 44 years), 2008–2024: summary of key findings – ecological study

A. Temporal trend – AMI hospitalization rates (35 to 44 years)



B. Risk factors: which increased and which decreased (2008–2023)

Increasing			Decreasing	
▲ Obesity	+77.6%	(Spearman ρ = 0.946)	▼ Smoking	−37.0% (Spearman ρ = −0.706)
▲ Diabetes	+61.8%	(Spearman ρ = 0.697)	▼ Hypertension	−10.0% (Spearman ρ = −0.504)
▲ Alcohol	+28.0%	(Spearman ρ = 0.695)		

The increase in metabolic risk factors occurred alongside a decline in smoking.

C. Key findings (hypothesis-generating correlations)

Obesity: the strongest correlate
Highest regression coefficient (standardized β = 1.280) and highest correlation (ρ = 0.946).

Alcohol: second correlated factor
β = 0.53; p = 0.037 (less robust evidence, not confirmed by bootstrap analysis).

Smoking paradox
Smoking declined 37% whereas AMI increased by 82% during the same period.

Regression model
$R^2 = 0.906$
Adjusted $R^2 = 0.854$
$F(5, 9) = 17.41$; $p < 0.001$
n = 15 observations

HYPOTHESIS

The co-variation of metabolic factors parallels the increase in premature AMI (ecological association; non-causal)

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Premature cardiovascular disease in Brazil: Summary of key findings. Panel A – Temporal trend of the AMI hospitalization rate (2008–2024) with annual percentage change by subperiod. Panel B – Rising and declining risk factors. Panel C – Multiple linear regression. AMI: acute myocardial infarction. Source: SIH-SUS/DATASUS and IBGE population estimates.

under codes I21 and I22 of the International Classification of Diseases, 10th Revision (ICD-10),[10] for the 35-to-44-year age group. Information on the prevalence of risk factors was extracted from the Surveillance System for Risk and Protective Factors for Chronic Diseases by Telephone Survey (Vigitel),[8] which is conducted annually by the Ministry of Health in Brazilian state capitals and the Federal District. The data cover the period from 2008 to 2023, with the exception of 2022, when the survey was not published. Population data used to calculate the rates were obtained from estimates by the Brazilian Institute of Geography and Statistics (IBGE).[11]

The dependent variable was the rate of hospitalization for AMI per 100,000 inhabitants in the 35-to-44-year age group, calculated using the following formula: rate = (number of admissions ÷ estimated population) × 100,000. The use of rates, rather than absolute numbers, aims to control for the effect of population growth on the time series. The independent variables were the prevalence of the following risk factors, expressed as percentages: obesity (body mass index ≥ 30 kg/m²), self-reported diabetes mellitus, self-reported hypertension, current smoking, and abusive alcohol consumption (defined as intake of 5 or more drinks for men or 4 or more drinks for women on a single occasion within the preceding 30 days). It should be noted that current smoking and abusive alcohol consumption are self-reported measures with limitations, as they do not include biochemical validation (e.g., cotinine levels), measures of intensity (pack-years), or chronicity, capturing only episodic consumption. Furthermore, they are subject to underreporting due to social desirability bias. These limitations should be considered when interpreting the respective coefficients.

The temporal trend of each variable was assessed by calculating the APC, obtained by regressing the natural logarithm of the values against the year, with 95% confidence intervals (CI) and significance testing. To quantify bivariate correlations, Pearson (r) and Spearman (ρ) coefficients were calculated, along with their respective p values and 95% CI derived via Fisher's Z transformation. The Pearson coefficient (r) was used to assess the linear relationship between continuous variables with an approximately normal distribution, whereas the Spearman coefficient (ρ) was used as a nonparametric corroborative measure, given that it is robust against deviations from normality, the presence of outliers, and non-linear monotonic relationships. The simultaneous use of both methods aimed to evaluate the robustness of the findings. A full correlation matrix was generated to identify multicollinearity among the independent variables. To examine the independent effect of each factor, a multiple linear regression model was fitted using the ordinary least squares method, including the five factors as simultaneous predictors. Both unstandardized (β) and standardized coefficients were reported. Diagnostic checks included normality of residuals (Shapiro–Wilk test), autocorrelation (Durbin–Watson), multicollinearity (variance inflation factor [VIF]), and influential points (Cook's distance). Bootstrap confidence intervals (1,000 resamples) and leave-one-out cross-validation were employed to assess the robustness of the coefficients. Sensitivity analysis was conducted excluding the year 2023, which was identified as an influential point. The significance level was set at 5% ($p < 0.05$). All analyses were performed using Python 3 with the pandas, scipy, statsmodels, and matplotlib packages.

Given that this study is based exclusively on publicly accessible secondary data that are aggregated and contain no individual identifiers, it is exempt from Research Ethics Committee review, in accordance with Resolution Number 466/2012 of the Brazilian National Health Council.[12]

Results

The AMI hospitalization rate in the 35-to-44-year age group showed a continuous upward trajectory throughout the analyzed period, rising from 16.49/100,000 inhabitants in 2008 to 30.07/100,000 in 2024, representing a cumulative increase of 82.3%. The overall APC was +3.20%/year (95% CI: 2.65–3.75), a statistically significant trend. Analysis by subperiod revealed a progressive acceleration of this growth, rising from +2.17%/year between 2008 and 2016 to +7.48%/year between 2020 and 2023, indicating an intensification of the trend in the most recent period of the series (Table 1, Figure 1).

During the same period, cardiovascular risk factors exhibited divergent temporal trajectories. Obesity showed the greatest relative expansion, with a 77.6% increase in prevalence, followed by diabetes (+61.8%) and abusive alcohol consumption (+28.0%). Conversely, smoking declined by 37.0%, and hypertension showed a slight reduction (−10.0%). All these trends were statistically significant. Figure 4C details the respective APCs.

In the bivariate correlation analysis, obesity stood out as the factor most strongly correlated with the AMI hospitalization rate, with concordant coefficients indicating a very strong correlation (Pearson $r = 0.872$; Spearman $\rho = 0.946$), alone explaining approximately 76% of the variance in the rates ($R^2 = 0.761$). Abusive alcohol consumption and diabetes also covaried positively with the rates, showing strong to very strong correlations, whereas smoking showed a negative correlation, and hypertension was the only factor with no statistically significant correlation over the entire period. Complete agreement was observed between the Pearson and Spearman coefficients for the five factors evaluated. The exclusion of 2023, which was identified as an influential data point, preserved the significance of four of the five factors according to the Spearman coefficient, reinforcing the stability of the observed correlations (Table 2; Figures 2 and 3).

In the multiple linear regression model, which included all five factors simultaneously, the set of variables explained a substantial portion of the variability in hospitalization rates ($R^2 = 0.906$; adjusted $R^2 = 0.854$), with the model showing overall significance. In this model, obesity remained the most robust correlate, displaying the highest standardized coefficient, followed by abusive alcohol consumption; the other factors did not remain statistically significant. The reversal of the signs for the diabetes and smoking coefficients warrants attention; this finding is consistent with the high multicollinearity between obesity and diabetes (obesity VIF = 7.79), which limits the independent estimation of effects. Diagnostic checks indicated that the model assumptions were

Table 1 – Time trend of hospitalization rates for AMI and prevalence of cardiovascular risk factors in the population aged 35 to 44 years, Brazil, 2008–2024

Year	Hospitalizations	Population age 35 to 44	Rate (per 100,000)	Obesity (%)	Smoking (%)	Alcohol (%)	Diabetes (%)	Hypertension (%)
2008	4,368	26,478,822	16.49	15.2	16.5	19.3	3.4	21.1
2009	4,771	26,778,822	17.82	15.6	14.8	20.0	3.3	20.9
2010	4,984	27,080,202	18.40	16.6	15.1	19.8	3.4	18.4
2011	5,280	27,420,022	19.26	19.6	13.9	18.2	3.3	19.6
2012	5,380	27,848,356	19.32	19.7	12.9	20.0	3.9	19.3
2013	5,304	28,369,851	18.70	20.1	11.2	17.5	3.6	18.3
2014	5,745	28,964,160	19.83	22.0	9.9	18.0	3.9	19.5
2015	5,901	29,600,138	19.94	23.6	10.4	19.4	5.0	18.9
2016	6,141	30,238,319	20.31	22.5	10.0	21.2	5.2	19.1
2017	6,385	30,846,512	20.70	22.3	11.7	22.2	4.1	17.3
2018	6,642	31,421,585	21.14	23.2	9.1	21.7	3.6	16.9
2019	7,263	31,974,888	22.71	22.8	9.7	20.9	3.6	17.0
2020	7,472	32,467,898	23.01	24.7	9.6	21.5	3.9	18.5
2021	8,148	32,835,926	24.81	25.5	11.6	20.0	4.9	18.6
2023	9,482	33,164,068	28.59	27.0	10.4	24.7	5.5	19.0
2024	9,964	33,134,523	30.07	—	—	—	—	—

Sources: SIH-SUS (hospitalizations); IBGE (population); Vigitel (risk factors). Vigitel was not published in 2022. Risk factor data were unavailable for 2024.

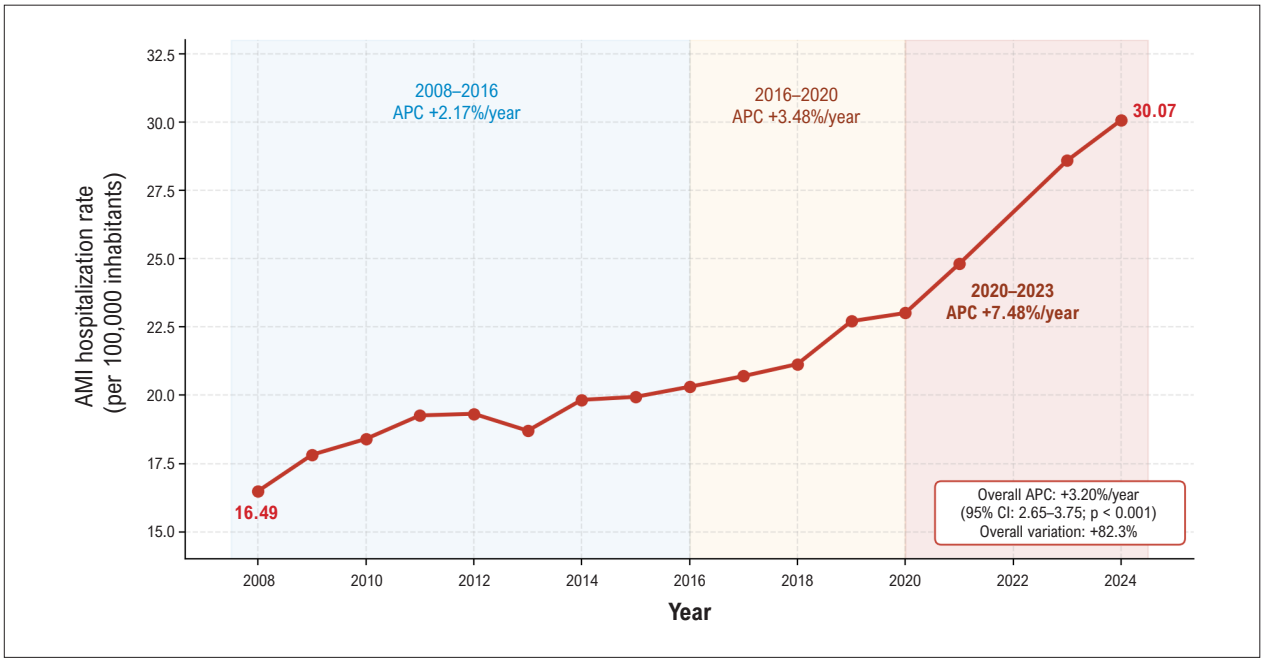

Figure 1 – *Time trend of AMI hospitalization rates (per 100,000 inhabitants) in the population aged 35 to 44 years, Brazil, 2008–2024. AMI: acute myocardial infarction; APC: annual percent change; CI: confidence interval. Source: SIH-SUS/DATASUS and IBGE population estimates.*

Table 2 – Bivariate correlations between cardiovascular risk factors and hospitalization rates for AMI in the population aged 35 to 44 years, Brazil, 2008–2023 (n = 15)

Risk factor	Pearson (r)	p (Pearson)	Spearman (ρ)	p (Spearman)	R²	Classification	Agreement
Obesity	+0.872*	<0.001	+0.946*	<0.001	0.761	Very strong (+)	Complete
Alcohol	+0.711*	0.003	+0.695*	0.004	0.505	Very strong (+)	Complete
Diabetes	+0.666*	0.007	+0.697*	0.004	0.444	Strong (+)	Complete
Smoking	−0.590*	0.021	−0.706*	0.003	0.348	Strong (−)	Complete
Hypertension	−0.402	0.138	−0.504	0.056	0.161	Moderate (−)	Complete (ns)

** p < 0.05. R² = Pearson coefficient of determination. ns = not significant. Classification: |r| < 0.30 weak; 0.30 to 0.50 moderate; 0.50 to 0.70 strong; > 0.70 very strong. Source: SIH-SUS/DATASUS and Vigitel/MS.*

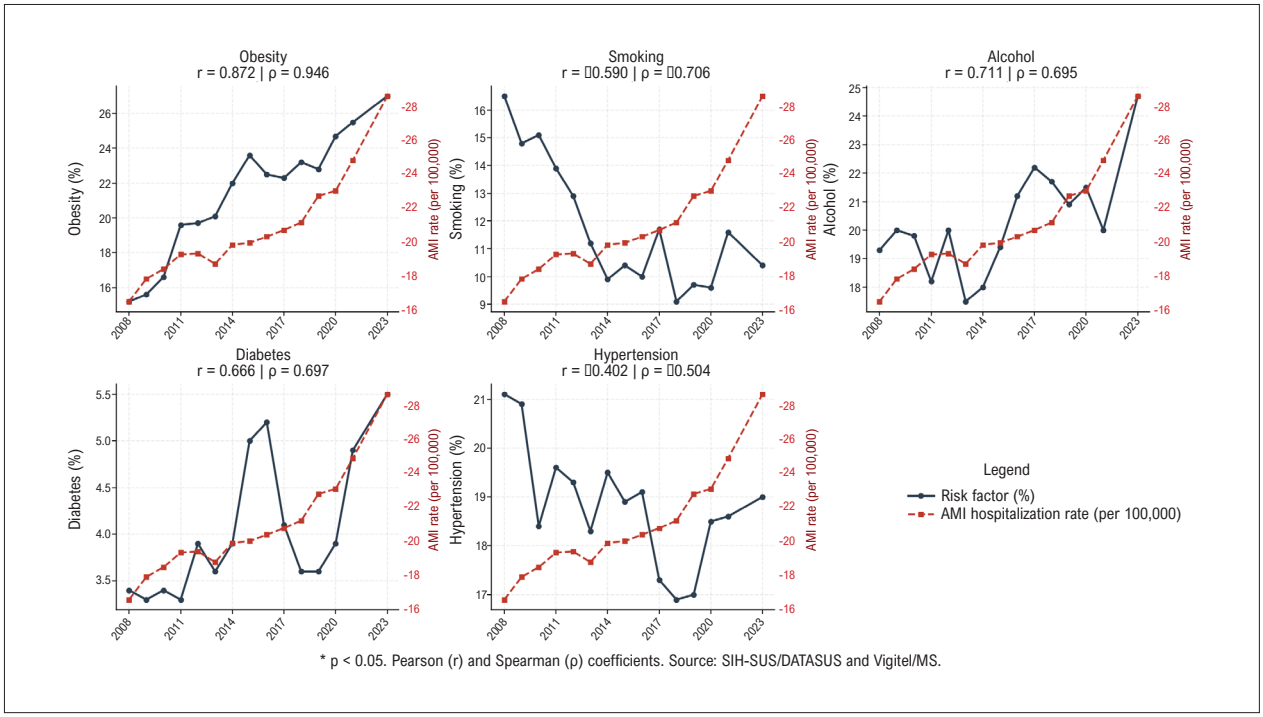

Figure 2 – *Temporal evolution of cardiovascular risk factors and AMI hospitalization rates in the population aged 35 to 44 years, Brazil, 2008–2023. AMI: acute myocardial infarction. Source: SIH-SUS/DATASUS and Vigitel/MS.*

adequately satisfied, including normality of residuals and the absence of significant autocorrelation; among the factors assessed, only obesity maintained a bootstrap confidence interval that did not include zero (Table 3; Figure 4).

Discussion

As this is an ecological study, these findings should be interpreted as generating population-level hypotheses, rather than providing evidence of individual-level causality. The main finding of this study was that obesity was the factor most strongly correlated with the increase in AMI hospitalization rates among the Brazilian population aged 35 to 44 years, showing a near-perfect correlation (Spearman $\rho = 0.946$; $p < 0.001$) and the highest standardized coefficient in the regression model (standardized $\beta = 1.280$; $p = 0.001$). This finding was supported by three converging statistical approaches: agreement between Pearson and Spearman coefficients, bootstrap confidence intervals that did not cross zero, and the maintenance of statistical significance in a sensitivity analysis excluding the year 2023. Even though the ecological design precludes individual-level inference, the correlation observed at the population level is biologically plausible considering evidence from individual-level studies. The cohort study by Khan et al. described a dose-response relationship between body mass index and

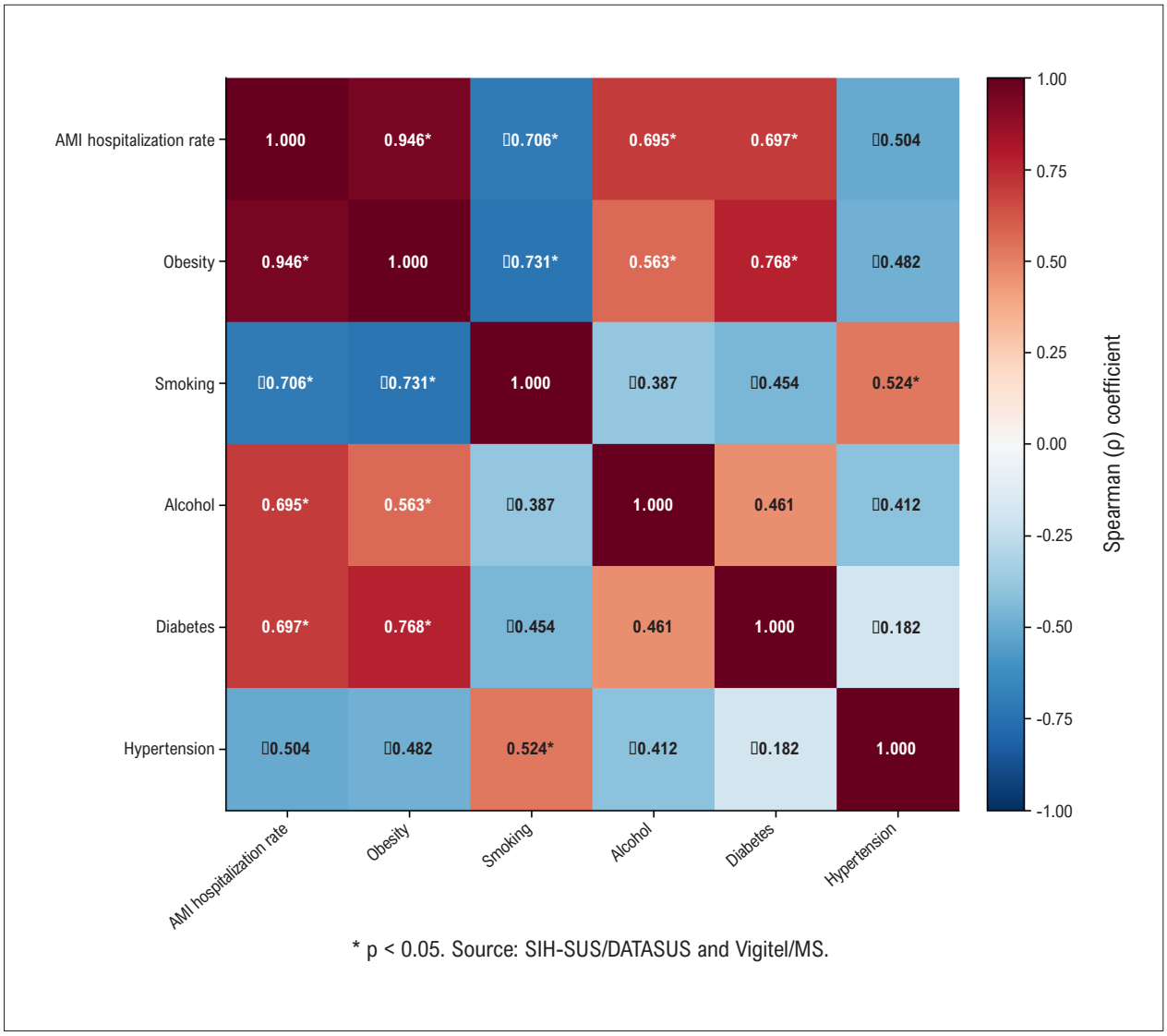

Figure 3 – *Spearman correlation matrix between cardiovascular risk factors and AMI hospitalization rates in the population aged 35 to 44 years, Brazil, 2008–2023 (n = 15). AMI: acute myocardial infarction. Source: SIH-SUS/DATASUS and Vigitel/MS.*

Table 3 – Multiple linear regression coefficients: predictors of hospitalization rates for AMI in the population aged 35 to 44 years, Brazil, 2008–2023 (n = 15)

Variable	β	Standardized β	SE	t	p	95% CI	VIF
Intercept	−21.94	—	9.89	−2.218	0.054	[−44.3; +0.4]	—
Obesity	+1.10	+1.280	0.25	+4.496	0.001	[+0.5; +1.7]	7.8
Smoking	+0.57	+0.432	0.29	+1.928	0.086	[−0.1; +1.2]	4.8
Alcohol	+0.53	+0.320	0.22	+2.438	0.037	[+0.04; +1.0]	1.7
Diabetes	−0.85	−0.208	0.76	−1.122	0.291	[−2.6; +0.9]	3.3
Hypertension	+0.27	+0.110	0.39	+0.698	0.503	[−0.6; +1.2]	2.4

CI: confidence interval; SE: standard error; VIF: variance inflation factor. $R^2 = 0.906$; adjusted $R^2 = 0.854$; $F(5, 9) = 17.41$ ($p < 0.001$). Shapiro–Wilk $W = 0.949$ ($p = 0.504$). Durbin–Watson $d = 1.930$. Bootstrap (1,000 resamples): Only obesity had a confidence interval that did not cross zero. Leave-one-out cross-validation: $Q^2 = 0.705$. VIF > 5 indicates multicollinearity. Source: SIH-SUS/DATASUS and Vigitel/MS.

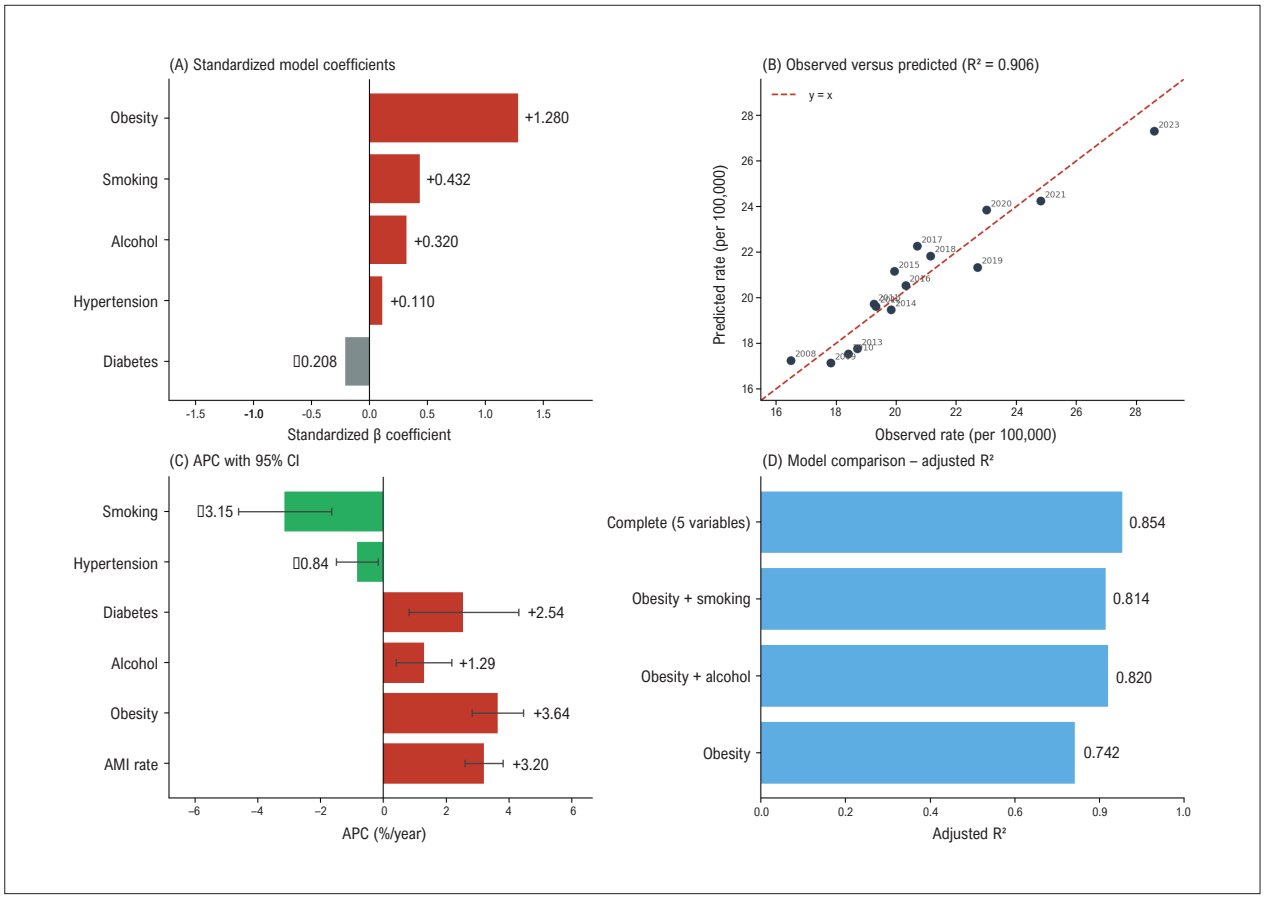

Figure 4 – *Multiple linear regression: (A) standardized model coefficients; (B) observed versus predicted values; (C) annual percent change (APC) with 95% CI; (D) model comparison by adjusted R². AMI: acute myocardial infarction; APC: annual percent change. Source: Prepared by the authors using data from SIH-SUS/DATASUS and Vigitel/MS.*

lifetime cardiovascular risk, showing a significantly higher lifetime cumulative risk of cardiovascular disease among obese individuals compared to those with normal weight.[13] Similarly, the INTERHEART study, conducted by Yusuf et al. across 52 countries with 27,000 participants, identified the waist-to-hip ratio, a marker of visceral adiposity, as one of the risk factors with the highest population-attributable fractions for myocardial infarction.[14] These individual-level studies make the metabolic hypothesis suggested by the temporal co-variation described here biologically plausible; however, as they do not share the same aggregated unit of analysis, they do not confirm this ecological finding.

The magnitude of the correlation between obesity and premature AMI observed in this study ($R^2 = 0.761$, corresponding to 76% of the variance in hospitalization rates) can be understood in light of pathophysiological mechanisms that have been well established in the literature. Ouchi et al. described how visceral adipose tissue functions as an active endocrine organ, secreting pro-inflammatory adipokines (TNF-α, IL-6, resistin) that foster an environment characterized by chronic low-grade inflammation, endothelial dysfunction, and accelerated atherosclerosis.[15] Koliaki et al. complement this perspective by demonstrating that obesity not only constitutes a direct risk factor for coronary artery disease but also potentiates other factors such as dyslipidemia, insulin resistance, and hypertension, thereby exerting a cumulative direct and indirect effect on cardiovascular risk.[16] In the Brazilian context, the 77.6% increase in obesity prevalence among adults aged 35 to 44 during the study period (from 15.2% in 2008 to 27.0% in 2023; APC of +3.64%/year) closely paralleled the rise in AMI rates, reinforcing the biological plausibility of the ecological correlation observed here. A similar pattern of temporal co-variation has previously been described in another Brazilian ecological study using the same data sources (Vigitel and DATASUS), in which the prevalence of obesity, overweight, and diabetes showed a progressive upward trend in an aggregated time-series analysis.[17] At the population level, data from Cuba illustrated the same direction of co-variation. The reduction in population body weight during the 1991–1995 economic crisis was accompanied by a parallel decline in cardiovascular disease mortality, with both indicators reverting during the subsequent period of weight regain.[18] Because they share the same aggregated unit of analysis, these ecological studies serve as the most methodologically comparable benchmarks and reinforce the coherence of the metabolic hypothesis,

even though they do not replace the need for confirmation through individual-level studies.

This scenario reflects the rapid nutritional transition Brazil has undergone over the past two decades, characterized by a significant rise in the consumption of ultra-processed foods, increasing physical inactivity, and socioeconomic inequalities that limit access to healthy foods, especially among young adults of working age.[8] The 35-to-44-year age group represents a generation that has been exposed since childhood to an increasingly obesogenic environment, featuring high availability of ultra-processed foods and intensive marketing of sugary and alcoholic beverages. This prolonged exposure to multiple risk factors from an early age may be contributing to increasingly early cardiovascular manifestations, as evidenced by the progressive acceleration in AMI hospitalization rates from +2.17% per year (2008–2016) to +7.48% per year (2020–2023).

Abusive alcohol consumption emerged as the factor with the second-strongest correlation in the regression model ($\beta = 0.53$; $p = 0.037$), although the evidence was less robust than that for obesity, as the finding was not confirmed by bootstrap analysis. At the individual level, the biological plausibility of this correlation is supported by consistent evidence in the literature. Piano described the multiple mechanisms by which excessive alcohol consumption affects the cardiovascular system, including cardiomyopathy, arrhythmias, hypertension, and a prothrombotic state.[19] Mukamal et al. demonstrated that binge drinking doubles the risk of mortality following AMI (hazard ratio = 2.0; 95% CI: 1.3–3.0),[20] and Mostofsky et al., in a systematic review and dose-response meta-analysis, confirmed that high levels of acute alcohol consumption (6–9 drinks) increase the risk of cardiovascular events within the following 24 hours (relative risk of 1.3 to 2.3).[21] The 28% increase in the prevalence of binge drinking among young Brazilian adults during the study period, with an acceleration after 2021, may reflect post-pandemic behavioral changes and the growing cultural normalization of excessive consumption within this age range.

Although diabetes mellitus showed a significant positive bivariate correlation with AMI hospitalization rates ($r = 0.666$; $\rho = 0.697$; $p < 0.01$), it did not reach significance in the multiple regression model, displaying a paradoxical negative coefficient ($\beta = -0.85$; $p = 0.291$). This finding is explained by high multicollinearity with obesity (VIF = 7.79; correlation between the two $\rho = 0.768$), reflecting the well-known pathophysiological overlap among the components of metabolic syndrome. Mongraw-Chaffin et al. demonstrated that metabolically healthy obesity is a transient state; nearly half of individuals with metabolically healthy obesity progressed to metabolic syndrome during follow-up, with a progressive increase in cardiovascular risk.[22] Bays et al. described how adiposopathy (i.e., adipose tissue dysfunction) plays a central role linking obesity, dyslipidemia, insulin resistance, and atherogenesis, framing metabolic syndrome as the manifestation of a single underlying pathophysiological process.[23] In this context, the inability to separate the independent effects of obesity and diabetes given the sample size of n = 15 observations does not invalidate the importance of diabetes as a risk factor; rather, it reinforces the synergistic nature of metabolic syndrome as a central component of the metabolic hypothesis raised in this study.

A particularly relevant finding of this study was the smoking paradox. The 37% reduction in smoking prevalence (APC of −3.15%/year; $p < 0.001$), resulting from decades of successful anti-smoking public policies, was not accompanied by a corresponding reduction in AMI rates. On the contrary, rates nearly doubled during the same period. In the regression analysis, smoking showed a paradoxical positive coefficient ($\beta = +0.57$; $p = 0.086$); this may be explained either by statistical suppression resulting from multicollinearity with obesity ($\rho = -0.731$) or by the fact that the reduction in smoking was insufficient to offset the simultaneous rise in multiple metabolic factors, namely, obesity (+77.6%), diabetes (+61.8%), and abusive alcohol consumption (+28.0%). This finding aligns with population-level observations by Wilmot et al., who documented a stagnation in the decline of coronary mortality among young adults in the United States, especially women, alongside a concomitant rise in obesity and diabetes in this age group.[5] Andersson and Vasan predicted that the upward trend in risk factors among young adults in developed countries foreshadows a new cardiovascular disease epidemic as this population ages,[6] and the data from the present study suggest that this is already taking place in Brazil. Taken together, the dissociation between declining smoking rates and rising AMI rates constitutes, in itself, a population-level signal that is compatible with the hypothesis that the accumulation of metabolic factors has come to outweigh smoking as a correlate of premature coronary disease. This hypothesis requires verification in individual-level studies.

Hypertension did not reach statistical significance for the entire period (Pearson $r = -0.402$; $p = 0.138$; Spearman $\rho = -0.504$; $p = 0.056$), although the p value for the Spearman coefficient was close to the significance threshold. This result can be explained by the observed biphasic temporal pattern: a downward trend from 2008 to 2017 (21.1% to 17.3%), followed by a rebound up to 2023 (19.0%). This dynamic, which is possibly linked to fluctuations in access to and adherence to antihypertensive treatment within primary health care, obscures the linear correlation when the full period is analyzed. In other words, because hypertension prevalence first declined (2008–2017) and then rose again (up to 2023), while the AMI rate increased almost monotonically throughout the interval, the lack of a unidirectional trend in hypertension causes the correlation coefficients (which are particularly sensitive to monotonic relationships) to approach zero for the overall period, even though a positive relationship might exist within each subperiod. This biphasic behavior illustrates how a purely aggregated analysis can mask co-variations that would be evident over shorter timeframes. Notably, in a sensitivity analysis excluding 2023, the correlation for hypertension became significant according to the Spearman coefficient ($\rho = -0.587$; $p = 0.027$), suggesting that this factor warrants close monitoring in future studies covering longer time horizons.

The acceleration in AMI rates in the post-2020 period (APC of +7.48%/year, compared to +3.48%/year in the immediately preceding period) is a finding that temporally coincides with the COVID-19 pandemic and prompts an exploratory hypothesis to be tested in studies using individual-level data. It should be noted, however, that this study lacks information on prior SARS-CoV-2 infection, vaccination coverage, potential changes in diagnostic criteria, or variations in hospital access over the period; thus, any link to the pandemic remains speculative. Nevertheless, mechanisms described in individual-level studies lend biological plausibility to this hypothesis. In a large United States cohort, Xie et al. observed that prior SARS-CoV-2 infection was associated with a 63% increase in the risk of cardiovascular events (hazard ratio = 1.63; 95% CI: 1.59–1.68) during the first year, including coronary artery disease, regardless of the severity of the acute infection.[24] In addition to the direct effects of the virus (myocardial injury, endothelial dysfunction, and prothrombotic state), the pandemic may have had additional indirect effects, including reduced access to services for the prevention and control of chronic risk factors; adverse behavioral changes during social isolation (sedentary lifestyle, weight gain, increased alcohol consumption); interruption of treatments for hypertension and diabetes; and increased psychosocial stress. Rallidis et al. reported that young adults with AMI exhibit a risk factor profile similar to that of older patients, with an emphasis on smoking, dyslipidemia, and family history, which may have rendered this population particularly susceptible to the pandemic's combined effects on lifestyle.[7]

Despite the care taken in separating and analyzing the results, certain limitations must be considered when interpreting the findings, which are essentially exploratory and hypothesis-generating in nature. The ecological design, based on aggregated population data, does not allow for the establishment of causal relationships at the individual level, posing a risk of ecological fallacy. Multicollinearity between obesity and diabetes (VIF = 7.79) compromises the independent estimation of their effects within the multiple regression model, although bivariate correlation methods confirmed the correlation of each factor individually. It is also worth noting that the limited number of observations (n = 15 years) relative to the five factors included simultaneously results in a ratio of approximately three observations per variable, which is well below the 10-to-15 ratio typically recommended for multiple regression models. This implies low statistical power and potentially unstable estimates, further reinforcing the exploratory nature of the modeling. As a mitigating factor, however, a sensitivity analysis using a parsimonious model, restricted to obesity and abusive alcohol consumption (two predictors for fifteen observations), identified these two factors as the dominant correlates and maintained high explanatory power (R^2 = 0.846), suggesting that the main findings do not rely solely on the over-parameterization of the full model. The year 2023 was identified as an influential point (Cook's distance = 0.943), although sensitivity analysis confirmed the robustness of the main findings. Data on risk factor prevalence were derived from Vigitel; these estimates are self-reported, lacking biochemical or clinical validation, and restricted to Brazilian state capitals and the Federal District, which limits national representativeness and extrapolation to the country as a whole, particularly to areas outside state capitals. Additionally, changes in telephone coverage between 2008 and 2023 altered the sampling frame, potentially compromising the comparability of obesity, hypertension, and diabetes prevalence rates over time. The operational definitions of certain factors are also limited. Smoking status is based solely on self-reported current smoking, without assessment of intensity (pack-years) or validation via cotinine, and abusive alcohol consumption captures binge-drinking episodes (5 or more drinks for men or 4 or more for women on a single occasion within the preceding 30 days) rather than chronic consumption patterns. These self-reported factors are also subject to underreporting due to social desirability bias. These limitations must be considered when interpreting the coefficients for smoking and alcohol, including the paradoxical pattern observed with smoking. Furthermore, the model did not incorporate known relevant variables, such as dyslipidemia, physical activity, dietary patterns, illicit drug use, socioeconomic inequality, environmental pollution, and family history, which means that residual confounding cannot be ruled out. Hospitalization data are limited to SIH-SUS, which covers approximately 80% to 85% of the Brazilian population; given that the economically active 35-to-44-year age group tends to have higher rates of supplementary health insurance coverage, the calculated rates may underestimate or otherwise bias the estimated magnitude of the phenomenon in this population. Finally, given the limited number of observations, some of the observed correlations may be due to chance, particularly the coefficients showing paradoxical signs (such as the inverted relationship between smoking and diabetes in the multiple regression model); therefore, they should be interpreted with particular caution.

Notwithstanding these limitations, the findings of this study have significant implications for cardiovascular public health policies in Brazil. Identifying obesity as the factor most strongly correlated with rising rates of AMI in young adults reinforces the importance of monitoring and investigating comprehensive primary prevention strategies geared toward this economically active age group. Specifically, the study results support strategies such as the regulation of ultra-processed foods; Camargo et al. estimated the effectiveness of taxation scenarios ranging from 10% to 50% for ultra-processed foods in reducing the prevalence of overweight and chronic diseases in Brazil.[25] Other strategies include the effective implementation of front-of-package labeling, in accordance with Brazilian Health Regulatory Agency (Anvisa) regulations analyzed by Mais et al., as a tool to promote healthier food choices;[26] early screening for metabolic factors (obesity, diabetes, dyslipidemia) in primary care, focusing on the 30-to-44-year age group; and more restrictive policies regarding abusive alcohol consumption among young adults, given that, according to De Boni et al., 53% of the Brazilian population supports restrictions on alcohol advertising.[27] The acceleration in premature AMI rates observed in the

post-pandemic period highlights the need for continuous monitoring and confirmatory individual-level studies, as well as coordination between the health and public policy sectors.

Conclusions

In this ecological time-series study, the sustained and progressively accelerating increase in hospitalizations for AMI among young Brazilian adults consistently co-varied with increases in the prevalence of metabolic risk factors. Obesity emerged as the factor most strongly correlated with this trend, making it the leading candidate for an explanatory hypothesis to be tested in studies using individual-level data. Abusive alcohol consumption emerged as a secondary correlate, albeit with less robust evidence. The concurrent reduction in smoking was not accompanied by a decline in hospitalization rates, a pattern suggesting that the gains achieved through tobacco control may have been offset by the rise in metabolic factors. As this analysis relies on aggregated data, these findings do not establish individual-level causality; however, they generate the hypothesis that the metabolic transition plays a central role in early-onset cardiovascular disease in this population, reinforcing the importance of continuous epidemiological monitoring and confirmatory individual-level studies to quantify the contribution of each risk factor and to more reliably inform cardiovascular prevention strategies geared toward young adults.

Author Contributions

Conception and design of the research, acquisition of data, analysis and interpretation of the data and statistical analysis: Pinho AR, Barros MC; writing of the manuscript: Pinho AR, Barros MC; writing of the manuscript: Pinho AR, Barros MC; critical revision of the manuscript for intellectual content: Barros MC.

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Use of Artificial Intelligence

During the preparation of this work, the authors used Claude for correcting grammar errors and checking text formatting. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

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