



Cardiovascular Mortality and Structural Inequalities in Europe

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Abstract

Background: Cardiovascular Diseases (CVD) remain the leading cause of mortality in Europe despite substantial improvements in prevention and treatment. Persistent inequalities across the continent suggest that cardiovascular mortality may be influenced not only by individual behavioral risk factors, but also by broader structural, socioeconomic, infectious-disease, and psychosocial conditions. This study examined long-term associations between circulatory-system mortality and selected epidemiological and structural indicators across European countries between 1990 and 2019.

Methods: A longitudinal ecological study was conducted using country-level data from European countries for 1990, 2000, 2010, and 2019. The dependent variable was age-standardized mortality from diseases of the circulatory system (SDR per 100,000 population). Independent variables included tuberculosis incidence, alcohol poisoning mortality, suicide mortality, smoking prevalence, and Gross National Income (GNI) per capita. Multivariable linear regression models were constructed for each study year to identify independent predictors of circulatory-system mortality. Comparative East-West analyses were additionally performed to assess regional inequalities across Europe.

Results: The multivariable regression models demonstrated high explanatory power across all study years (adjusted R^2 approximately 0.71-0.82). Tuberculosis incidence consistently remained the strongest positive independent predictor of circulatory-system mortality, while GNI per capita demonstrated a stable inverse association. Alcohol poisoning mortality was positively associated with cardiovascular mortality in most study years. Smoking prevalence showed positive but statistically unstable associations after multivariable adjustment, whereas suicide mortality did not remain independently associated with circulatory-system mortality. Comparative analyses demonstrated persistent East-West disparities throughout the study period. Eastern and post-socialist European countries generally exhibited substantially higher cardiovascular mortality, higher tuberculosis incidence, higher alcohol-related mortality, and lower national income compared with Western European countries. These patterns persisted despite overall declines in circulatory-system mortality across Europe between 1990 and 2019.

Conclusions: Cardiovascular mortality in Europe appears to be strongly associated with broader structural, infectious, socioeconomic, and psychosocial conditions rather than solely with individual behavioral risk factors. The coexistence of elevated circulatory-system mortality, tuberculosis burden, alcohol-related mortality, and socioeconomic disadvantage in Eastern Europe supports structural and syndemic interpretations of cardiovascular inequalities. Reducing cardiovascular mortality inequalities in Europe may therefore require not only conventional risk-factor prevention, but also broader policies addressing social inequality, psychosocial stress, healthcare-system resilience, and structural vulnerability.

Keywords: Cardiovascular Mortality; Psychosocial Stress; Alcohol-related Mortality; Socioeconomic Inequality; Europe

Introduction

Cardiovascular diseases remain the leading cause of mortality in Europe despite substantial advances in prevention, treatment, and life expectancy during recent decades. Although most European countries have experienced major declines in circulatory-system mortality, pronounced inequalities persist between Western and Eastern Europe [1]. Countries in Eastern and post-socialist Europe continue to demonstrate substantially higher cardiovascular mortality compared with Western European populations, suggesting that the epidemiological transition has progressed

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unevenly across the continent [2-4].

Traditional cardiovascular epidemiology has primarily focused on individual behavioral risk factors such as smoking, alcohol consumption, obesity, hypertension, dyslipidemia, and physical inactivity [9]. While these determinants remain critically important, growing evidence indicates that cardiovascular mortality is also strongly influenced by broader structural, socioeconomic, psychosocial, and healthcare-system conditions [5-9]. Socioeconomic deprivation, healthcare-access inequalities, institutional instability, adverse behavioral environments, and chronic psychosocial stress may interact to shape cardiovascular outcomes at the population level [6-8].

Previous European studies have demonstrated strong associations between cardiovascular mortality and indicators of economic development, alcohol-related harm, and social transition, particularly in Eastern Europe following the collapse of socialist political systems during the early 1990s [10-13]. At the same time, persistent infectious-disease inequalities, especially tuberculosis incidence, continue to reflect broader differences in living conditions, public-health capacity, social vulnerability, and institutional organization across Europe [14-16]. These overlapping patterns suggest that cardiovascular mortality may be embedded within wider epidemiological and societal systems rather than resulting solely from isolated disease-specific mechanisms.

This perspective is consistent with structural epidemiology, social-determinants research, and syndemic theory. Structural epidemiology emphasizes the role of macro-level political, economic, and institutional conditions in shaping disease distribution [7], while syndemic approaches propose that multiple diseases and adverse health conditions cluster and interact under conditions of social inequality and population vulnerability [17, 18]. Contemporary social-determinants frameworks similarly emphasize that population-health outcomes emerge through interactions between socioeconomic conditions, psychosocial environments, behavioral patterns, and healthcare-system capacity [5, 6, 9]. Within this broader perspective, cardiovascular mortality may therefore be interpreted not solely as a consequence of individual lifestyle risk factors, but also as an indicator of wider structural and epidemiological organization.

Increasing attention within social epidemiology and cardiovascular medicine has also been directed toward the role of psychosocial distress in generating adverse population-health outcomes [1, 13, 19-23]. Psychological distress, including chronic stress, anxiety, depression, and post-traumatic stress responses, has been increasingly recognized not only as a consequence of cardiovascular disease, but also as an upstream determinant contributing to cardiovascular risk [3, 19, 21]. Recent cardiovascular research has demonstrated that psychological distress following myocardial infarction is associated with poorer adherence to preventive treatment, reduced participation in rehabilitation, impaired quality of life, recurrent cardiovascular events, and increased long-term mortality [21, 24]. More broadly, chronic psychosocial stress appears capable of influencing cardiovascular health through interconnected behavioral, neuroendocrine, autonomic, inflammatory, and immune-mediated pathways [15, 21, 25].

Contemporary social-determinants frameworks conceptualize psychosocial distress not merely as an individual psychological condition, but as a socially patterned response to adverse structural environments [6]. Chronic economic insecurity, unemployment,

institutional instability, social fragmentation, low social cohesion, discrimination, and reduced access to social and healthcare protection may collectively generate prolonged stress exposure at the population level [6, 11, 25, 26]. Importantly, adverse social environments may also shape the emergence of unhealthy behavioral patterns through stress-mediated coping mechanisms [25, 26]. Hazardous alcohol consumption, tobacco use, self-harm, poor adherence to preventive healthcare, and reduced participation in health-promoting behaviors may therefore represent maladaptive responses to chronic social adversity rather than purely individual lifestyle choices [1, 23, 27, 28].

Emerging evidence further suggests that psychosocial stress may directly contribute to cardiovascular pathology through sustained activation of neurocardiac and inflammatory pathways [6, 21, 24]. Chronic stress exposure has been associated with autonomic dysregulation, endothelial dysfunction, increased inflammatory signaling, metabolic disturbances, vascular hyperreactivity, and accelerated atherosclerotic progression [21]. The cumulative physiological burden of repeated stress exposure, conceptualized as allostatic load, may therefore provide a biologically plausible mechanism linking adverse social environments with long-term cardiovascular mortality patterns [24].

These mechanisms may be particularly relevant in post-socialist Europe, where rapid political and economic transformations following the collapse of socialist systems were accompanied by profound social disruption, widening inequality, labor-market instability, weakening of institutional security, and deterioration of public-health systems in several countries [3, 11, 12, 25, 26]. Previous studies have suggested that these transitions contributed to excess mortality through both direct material deprivation and indirect psychosocial mechanisms [3, 12]. Persistently elevated levels of alcohol-related mortality, suicide mortality, infectious disease burden, and cardiovascular mortality observed in several post-socialist countries may therefore reflect broader processes of structural and psychosocial destabilization [3, 11].

Within this context, alcohol poisoning mortality, tuberculosis incidence, and suicide mortality may be interpreted as complementary indicators of psychosocial and structural distress. Alcohol poisoning mortality reflects not only excessive alcohol exposure, but also broader processes of social marginalization, stress-related coping, and societal dislocation [12, 27]. Similarly, suicide mortality may serve as a marker of impaired social integration, economic insecurity, chronic distress, and collective psychosocial strain [29].

Tuberculosis was conceptualized in the present study not simply as an infectious disease, but as a socially determined disease strongly conditioned by structural inequality [7]. Although caused by a biological pathogen, tuberculosis incidence is highly sensitive to social and environmental conditions, including poverty, overcrowding, malnutrition, incarceration, homelessness, weakened healthcare systems, and chronic psychosocial stress [9]. Historically, tuberculosis has long been recognized as a “social disease” because its distribution closely reflects socioeconomic organization and living conditions rather than pathogen exposure alone [9]. In Eastern Europe, the post-socialist period was associated with substantial increases in tuberculosis incidence and multidrug-resistant tuberculosis, partly linked to healthcare-system disruption and deteriorating social conditions [9]. From this perspective, tuberculosis burden may function as a population-level marker of structural vulnerability and institutional fragility.

The coexistence of elevated cardiovascular mortality, alcohol-related mortality, tuberculosis burden, suicide mortality, and psychosocial distress indicators suggests the presence of interconnected syndemic processes operating across post-socialist societies [3]. Rather than representing isolated epidemiological phenomena, circulatory disease mortality, alcohol-related harm, tuberculosis incidence, and suicide mortality may reflect different manifestations of shared upstream determinants rooted in adverse social environments, chronic psychosocial stress, and unequal structural conditions.

Accordingly, the present study aimed to examine long-term East-West differences in circulatory-system mortality and selected structural, behavioral, infectious-disease, and psychosocial indicators across European countries between 1990 and 2019. In addition, the study investigated whether tuberculosis incidence, alcohol poisoning mortality, suicide mortality, smoking prevalence, and economic conditions were independently associated with cardiovascular mortality at the population level within a multivariable ecological framework spanning different phases of the European epidemiological transition.

Methods

Study design and data sources

A repeated cross-sectional ecological study was conducted using country-level population health data from 42 European countries for four observation years: 1990, 2000, 2010, and 2019. The study was designed to examine structural, behavioral, and epidemiological determinants of age-standardized circulatory-system mortality across Europe during different phases of the epidemiological transition. The analyses were conceptually informed by the Epidemiological System Synchronization Framework (ESSI), which conceptualizes circulatory-system mortality as a systems-level outcome emerging from interactions among infectious-disease burden, socioeconomic conditions, behavioral risk environments, psychosocial stress, and healthcare-system capacity.

All indicators were obtained from publicly available international databases, including the World Health Organization (WHO) European Health Information Gateway and World Bank datasets. Country-level observations were harmonized across study years to ensure temporal and cross-national comparability. Countries were additionally grouped into Eastern and Western European regions for comparative analyses of long-term epidemiological divergence, following regional classifications commonly used in comparative European public health research.

Outcome variable

The dependent variable was age-standardized mortality from diseases of the circulatory system (Standardized Death Rate [SDR] per 100,000 population). This indicator represented mortality attributable to cardiovascular and circulatory diseases. Age-standardized rates were used to minimize the influence of differences in population age structure between countries and across time.

Independent variables

Five independent variables representing behavioral, structural, and epidemiological population-health determinants were included in the analyses: prevalence of current tobacco smoking among persons aged ≥ 15 years (%), Standardized Death Rate (SDR) from accidental poisoning by alcohol (per 100,000 population), tuberculosis incidence

(new and relapse cases per 100,000 population), standardized suicide mortality rate (SDR per 100,000 population), Gross National Income (GNI) per capita (current US\$). Smoking prevalence was included as a conventional behavioral cardiovascular risk factor. Alcohol poisoning mortality was treated as an indicator of adverse alcohol-related behavioral and social-health environments. Tuberculosis incidence was incorporated as a marker of infectious-disease burden and broader structural population-health vulnerability. Suicide mortality was included as an indicator of psychosocial stress and social-health instability. Gross national income per capita was used as a proxy measure of socioeconomic development and systemic healthcare capacity. Gross national income per capita was additionally scaled per US\$1,000 in regression analyses to improve coefficient interpretability.

Statistical analysis

Separate multivariable linear regression models were first estimated for each study year (1990, 2000, 2010, and 2019) to evaluate independent associations between structural, behavioral, and epidemiological determinants and age-standardized circulatory-system mortality. In all models, age-standardized circulatory-system mortality served as the dependent variable. The general regression specification was:

$$\text{CVD}_i = \beta_0 + \beta_1 \text{Smoking}_i + \beta_2 \text{Alcohol}_i + \beta_3 \text{TB}_i + \beta_4 \text{Suicide}_i + \beta_5 \text{GNI}_i + \epsilon_i$$

where: (CVD_i) represents circulatory-system mortality in country (i), (Smoking_i) represents smoking prevalence, (Alcohol_i) represents alcohol poisoning mortality, (TB_i) represents tuberculosis incidence, (Suicide_i) represents suicide mortality, (GNI_i) represents gross national income per capita, (ϵ_i) represents the residual error term.

Regression coefficients (β), standard errors (SE), *t*-values, *p*-values, and model fit statistics (*R*² and adjusted *R*²) were calculated for each model. Statistical significance was defined as a two-sided *p*-value < 0.05 .

We subsequently examined temporal and regional patterns of circulatory-system mortality and related structural and epidemiological indicators descriptively and compared outcomes between Eastern and Western Europe, following regional classifications commonly used in comparative European public health research. Differences between regions were assessed using Welch's unequal variances *t*-tests, and East/West ratios were calculated for each study year.

Results

Across all study years, the multivariable regression models demonstrated high explanatory power for between-country variation in age-standardized circulatory-system mortality in Europe, with adjusted *R*² values ranging from 0.713 to 0.820 (all $p < 0.001$) (Table 1). Tuberculosis incidence consistently remained the strongest positive independent predictor of circulatory-system mortality across all models. Gross National Income (GNI) per capita demonstrated a stable inverse association with circulatory-system mortality throughout the study period.

Alcohol poisoning mortality was significantly associated with higher circulatory-system mortality in 1990, 2010, and 2019, whereas smoking prevalence showed only borderline or non-significant associations after multivariable adjustment. Suicide mortality was

Table 1: East/West ratio trends for structural and epidemiological indicators, 1990-2019.

Variable	1990	2000	2010	2019
Cardiovascular disease	1.72	2.30	2.70	3.05
Smoking prevalence	1.21	1.00	1.10	1.30
Alcohol poisoning	7.65	9.04	5.23	6.26
Tuberculosis incidence	2.54	4.79	5.12	3.77
Suicides	1.30	1.61	1.39	1.06
GNI percapita	0.11	0.12	0.20	0.25

not independently associated with circulatory-system mortality in any study year (Table 2).

The highest overall model fit was observed in 2010 (adjusted $R^2=0.820$), suggesting particularly strong alignment between structural, behavioral, infectious-disease, and socioeconomic determinants during the post-transition period in Europe.

Table 3 presents the comparative analysis of structural and epidemiological indicators between Eastern and Western Europe in 1990, 2000, 2010, and 2019. Significant East-West differences were observed for most indicators throughout the study period.

Age-standardized circulatory-system mortality declined substantially in both regions between 1990 and 2019 (Table 3). In Eastern Europe, circulatory disease SDR decreased from 589.5 to 390.8 per 100,000 population, whereas in Western Europe it declined from 341.9 to 127.9 per 100,000 population. Despite these absolute improvements, relative East-West inequality progressively increased over time. As shown in Table 3, the East/West ratio for circulatory-system mortality increased from 1.72 in 1990 to 3.05 in 2019, indicating substantially faster cardiovascular mortality decline in Western Europe.

Smoking prevalence demonstrated partial convergence between regions during the 2000s. Although smoking prevalence remained significantly higher in Eastern Europe in both 1990 and 2019, regional differences were no longer statistically significant in 2000 and 2010 (Table 3). The East/West smoking ratio approached parity in 2000 (1.00) before increasing modestly again by 2019 (1.30) (Table 1).

These findings suggest that conventional behavioral risk factors alone are insufficient to explain the widening cardiovascular mortality gap.

Alcohol poisoning mortality remained consistently elevated in Eastern Europe throughout the study period and demonstrated marked regional inequality (Table 3). The East/West ratio for alcohol poisoning SDR reached 9.04 in 2000 and remained above six-fold higher in 2019 despite partial declines over time (Table 1). Similarly, tuberculosis incidence demonstrated persistent and substantial East-West divergence. Tuberculosis incidence increased markedly in Eastern Europe after 1990 and remained significantly higher than in Western Europe across all study years, with East/West ratios ranging from 2.54 to 5.12 (Tables 2 and 3).

Suicide mortality demonstrated a different temporal pattern characterized by gradual convergence. Although suicide SDR was higher in Eastern Europe during most of the study period, the difference was no longer statistically significant by 2019 (9.3 vs. 8.8 per 100,000 population; $p=0.742$) (Table 2). Correspondingly, the East/West suicide mortality ratio declined from 1.61 in 2000 to 1.06 in 2019, indicating near-complete regional convergence (Table 1).

Substantial economic disparities persisted throughout the study period despite marked economic growth in Eastern Europe. Gross national income per capita increased more than six-fold in Eastern Europe between 1990 and 2019; however, the East/West GNI ratio rose only from 0.11 to 0.25, indicating incomplete economic convergence (Table 1).

Overall, the findings demonstrate that despite broad epidemiological and economic improvements across Europe, major East-West inequalities persisted for circulatory-system mortality, tuberculosis incidence, and alcohol-related mortality. The coexistence of persistent cardiovascular disadvantage with elevated tuberculosis burden and alcohol-related harm supports the interpretation of a durable postsocialist syndemic pattern in Eastern Europe. In contrast, the partial convergence observed for smoking prevalence and suicide mortality suggests that East-West mortality divergence was selective rather than universal across health indicators.

Discussion

The present study demonstrates that cardiovascular mortality

Table 2: Multivariable linear regression models for age-standardized circulatory-system mortality in Europe, 1990-2019.

Predictor	1990 β (p-value)	2000 β (p-value)	2010 β (p-value)	2019 β (p-value)
Smoking prevalence (%)	2.14 (0.389)	1.87 (0.399)	4.92 (0.064)	3.50 (0.210)
Alcohol poisoning	8.72 (0.008)	1.18 (0.752)	11.74 (0.005)	13.36 (0.021)
Tuberculosis incidence	3.41 (<0.001)	2.80 (<0.001)	3.41 (<0.001)	3.96 (<0.001)
Suicide SDR	1.28 (0.466)	0.96 (0.624)	1.88 (0.482)	-1.23 (0.743)
GNI per capita	-0.006 (0.001)	-0.007 (<0.001)	-2.94 (<0.001)	-2.25 (0.011)

Table 3: East-West comparison of structural and epidemiological indicators in Europe, 1990-2019.

Variable	East 1990	West 1990	p-value	East 2000	West 2000	p-value	East 2010	West 2010	p-value	East 2019	West 2019	p-value
Cardiovascular diseases	589.5	341.9	<0.001	584.9	254.0	<0.001	492.9	182.3	<0.001	390.8	127.9	<0.001
Smoking prevalence (%)	39.4	32.6	<0.001	35.4	35.3	0.965	31.8	29.0	0.118	28.9	22.2	<0.001
Alcohol poisoning	3.06	0.40	0.013	4.43	0.49	0.023	3.35	0.64	0.041	2.63	0.42	0.036
Tuberculosis incidence	35.6	14.0	<0.001	50.8	10.6	<0.001	42.0	8.2	<0.001	26.0	6.9	<0.001
Suicides	17.3	13.3	0.130	18.2	11.3	0.024	13.9	10.0	0.047	9.3	8.8	0.742
GNI percapita	2,086	18,942	<0.001	3,006	25,808	<0.001	9,355	46,446	<0.001	12,734	51,497	<0.001

in Europe is strongly associated with broader structural, infectious, behavioral, and psychosocial conditions rather than with isolated lifestyle risk factors alone. Across all study years between 1990 and 2019, tuberculosis incidence remained the strongest positive independent predictor of circulatory-system mortality, while gross national income per capita showed a stable inverse association. Alcohol poisoning mortality also demonstrated consistent positive associations with cardiovascular mortality in most study years. Collectively, these findings suggest that cardiovascular mortality patterns in Europe emerge from interconnected epidemiological and structural processes operating at the population level.

Tuberculosis incidence likely functions not merely as an infectious disease indicator, but also as a marker of structural vulnerability encompassing poverty, healthcare-access limitations, institutional fragility, adverse living conditions, social marginalization, and chronic underinvestment in public-health infrastructure [7, 30]. The persistence of the tuberculosis –cardiovascular mortality association across three decades suggests that cardiovascular inequalities in Europe remained closely connected to broader epidemiological and socioeconomic disparities throughout the continental epidemiological transition. From a structural epidemiology perspective, infectious disease burden may therefore reflect wider deficiencies in social protection systems, healthcare organization, and population resilience.

The findings additionally demonstrate a persistent East-West divergence in European mortality patterns. Countries from Eastern and post-socialist Europe generally combined higher tuberculosis incidence, higher alcohol-related mortality, lower national income, and substantially elevated circulatory-system mortality compared with Western European countries. These observations are consistent with previous evidence demonstrating delayed mortality convergence in post-socialist Europe following the political and economic transitions of the early 1990s [11, 12, 25, 26]. Whereas many Western European countries experienced earlier improvements in infectious-disease control, socioeconomic stability, healthcare-system capacity, and chronic-disease prevention, several Eastern European populations continued to experience overlapping burdens of infectious disease, hazardous behavioral environments, psychosocial distress, and cardiovascular mortality.

The positive association between alcohol poisoning mortality and circulatory-system mortality further supports a syndemic interpretation of cardiovascular mortality patterns in Europe [12, 18, 23, 31, 32]. Alcohol poisoning mortality likely represents more than a direct toxic exposure pathway. At the population level, it may additionally reflect chronic psychosocial stress, social disintegration, deprivation, maladaptive coping behaviors, weakened institutional support, and adverse behavioral environments [1, 12, 23, 27]. Hazardous alcohol use in post-socialist societies has frequently been interpreted within the context of economic insecurity, unemployment, social instability, and deterioration of collective social structures during periods of rapid political transformation [1, 13, 19–22, 27]. Within this framework, alcohol-related mortality may therefore function as an indirect marker of broader psychosocial and structural distress.

The present findings are also compatible with growing evidence linking psychosocial distress with adverse cardiovascular outcomes. Contemporary cardiovascular research increasingly recognizes chronic psychosocial stress, anxiety, depression, and social adversity

not only as consequences of cardiovascular disease, but also as upstream contributors to cardiovascular risk [4, 21]. Chronic stress exposure has been associated with autonomic dysregulation, endothelial dysfunction, vascular hyperreactivity, inflammatory activation, immune dysregulation, and accelerated atherosclerotic progression [21]. The concept of allostatic load provides a biologically plausible mechanism through which prolonged exposure to adverse social environments may become physiologically embedded and contribute to long-term cardiovascular mortality patterns [4].

Importantly, psychosocial distress may also influence cardiovascular mortality indirectly through behavioral pathways. Chronic stress and social instability are associated with increased smoking prevalence, hazardous alcohol consumption, reduced healthcare adherence, physical inactivity, social withdrawal, and lower participation in preventive care [6, 15]. From this perspective, unhealthy behaviors may partly represent downstream manifestations of adverse structural and psychosocial environments rather than purely autonomous individual choices. The coexistence of elevated alcohol-related mortality, suicide mortality, infectious-disease burden, and cardiovascular mortality observed in several Eastern European countries therefore supports the hypothesis that these conditions share common upstream determinants rooted in structural inequality and chronic psychosocial stress.

Gross national income per capita demonstrated a stable inverse association with circulatory-system mortality throughout the study period. This finding aligns with extensive literature linking socioeconomic development with lower cardiovascular mortality through improvements in healthcare access, preventive services, education, living standards, nutritional quality, institutional stability, and public-health infrastructure [11, 26, 24, 33]. National income may therefore be interpreted as an indicator of societal adaptive capacity – the ability of healthcare systems and social institutions to mitigate epidemiological risks while simultaneously improving chronic disease prevention, treatment, and resilience against adverse social conditions.

Smoking prevalence demonstrated positive but statistically unstable associations after multivariable adjustment. Although tobacco smoking is unequivocally established as a major cardiovascular risk factor at the individual level [28], its ecological association appeared partially attenuated after inclusion of broader structural and epidemiological covariates. This observation may suggest that smoking prevalence clusters within wider socioeconomic, behavioral, and psychosocial environments. Similarly, suicide mortality did not remain independently associated with cardiovascular mortality in adjusted models, although suicide may still represent an important downstream manifestation of chronic psychosocial strain, impaired social integration, and structural disadvantage [3].

Interestingly, the strongest overall model fit was observed in 2010, suggesting that interactions between infectious, socioeconomic, behavioral, and psychosocial determinants may have become particularly pronounced during the post-transition period in Europe. This pattern may reflect the delayed long-term health consequences of post-socialist restructuring combined with persistent inequalities in healthcare capacity, economic development, and social protection systems across European populations.

The present findings are additionally compatible with syndemic theory and structural epidemiology. Syndemic frameworks emphasize that diseases do not occur in isolation, but instead cluster and interact

under conditions of social inequality and structural vulnerability [17, 34-36]. In this context, cardiovascular mortality, alcohol-related mortality, tuberculosis burden, and suicide mortality may represent interconnected manifestations of shared upstream determinants rather than independent epidemiological phenomena. Structural epidemiology similarly emphasizes that disease distribution is shaped by macro-level political, economic, and institutional environments operating across the life course and at the population level [17].

Several limitations should be acknowledged. First, the ecological design precludes causal inference at the individual level and introduces the possibility of ecological fallacy. Second, national-level indicators may conceal important within-country heterogeneity and regional inequalities. Third, the analysis relied on international databases that may differ in reporting quality, diagnostic practices, and completeness across countries and time periods. Fourth, several variables included in the analysis likely function as proxy indicators of broader structural and psychosocial processes rather than isolated causal determinants. Finally, unmeasured confounding factors, including dietary patterns, obesity prevalence, healthcare expenditure, and treatment accessibility, may also contribute to observed mortality differences.

Despite these limitations, the study provides evidence that cardiovascular mortality in Europe is closely embedded within broader structural, epidemiological, and psychosocial systems. The persistence of strong associations between tuberculosis incidence, alcohol-related mortality, socioeconomic disadvantage, and circulatory-system mortality suggests that cardiovascular inequalities are shaped not only by conventional behavioral risk factors, but also by wider social, economic, institutional, and psychosocial conditions. These findings indicate that reducing cardiovascular inequalities in Europe may require not only individual-level prevention strategies targeting smoking, alcohol use, hypertension, and dyslipidemia, but also broader structural policies addressing social inequality, public-health infrastructure, psychosocial stress, healthcare-system resilience, and population-level vulnerability.

Conclusion

Cardiovascular mortality in Europe appears to be strongly embedded within broader structural, infectious, socioeconomic, and psychosocial systems rather than being determined solely by individual behavioral risk factors. Across the period 1990-2019, tuberculosis incidence consistently remained the strongest independent positive predictor of circulatory-system mortality, while national income demonstrated a stable protective association. Alcohol poisoning mortality also showed persistent positive associations with cardiovascular mortality, supporting the interpretation that adverse behavioral environments and psychosocial distress contribute to broader population-health inequalities.

The findings reveal a persistent East-West divide in European mortality patterns, characterized by the coexistence of elevated cardiovascular mortality, infectious disease burden, alcohol-related mortality, and socioeconomic disadvantage in several Eastern and post-socialist countries. These patterns support the view that cardiovascular mortality reflects broader interactions between infectious burden, psychosocial stress, socioeconomic conditions, behavioral environments, and healthcare-system capacity rather than isolated disease-specific mechanisms alone.

The study additionally supports syndemic and structural-

epidemiological interpretations of cardiovascular inequalities in Europe. Tuberculosis burden, alcohol-related mortality, and suicide mortality may function not only as isolated health indicators, but also as markers of broader structural vulnerability, institutional instability, and chronic psychosocial distress. The coexistence of these conditions suggests that cardiovascular mortality may reflect the cumulative effects of adverse social environments and incomplete epidemiological transition.

Although the ecological design precludes causal inference at the individual level, the results highlight the importance of integrating social, structural, and psychosocial determinants into cardiovascular epidemiology and public-health policy. Reducing cardiovascular inequalities in Europe may therefore require not only conventional prevention strategies targeting smoking, alcohol use, hypertension, and dyslipidemia, but also broader interventions addressing socioeconomic inequality, psychosocial stress, healthcare-system resilience, public-health infrastructure, and structural vulnerability at the population level.

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