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Spirit of the Hawk: Colonial intensity and alcohol use disorders across 178 countries

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ABSTRACT

Qualitative research links colonial history to alcohol-related harms in specific populations, and econometric studies show that colonial institutions shape long-run health trajectories, yet no study has connected these literatures by testing whether colonial intensity predicts national-level alcohol outcomes in a spatially explicit panel framework. We construct a colonial intensity composite (the duration of colonial rule weighted by colony type—extractive versus settler) from COLDAT 3.0 and estimate panel Spatial Durbin Models on 178 countries over 21 years (2000–2020), using geographic contiguity and shared-colonizer spatial weights across four WHO alcohol outcomes. Colonial intensity shows a significant positive direct association with alcohol use disorder (AUD) prevalence across all eight specifications (0.065–0.238 percentage points per unit), accompanied by negative spillover effects (associations transmitted to geographically neighbouring or co-colonized countries). The AUD association emerges only after controlling for cultural confounders (suppression effect) and shows no temporal decay, while consumption effects weaken over time—a pattern consistent with intergenerational trauma persistence. Effects concentrate in British colonies. Standard alcohol policies reduce consumption but not AUD, suggesting regulatory tools alone cannot close the colonial legacy gap in disorder prevalence. These findings provide the first large-scale quantitative evidence linking colonial intensity to contemporary AUD.

> "And, indeed, if it be the design of Providence to extirpate these savages in order to make room for cultivators of the earth, it seems not improbable that rum may be the appointed means."
>
> — Benjamin Franklin, The Autobiography (recalling the Carlisle treaty, 1753)

> "Nothing can be done to advantage unless the great Council … will prohibit any person from selling spirituous liquors among their Red Brothers; … owing to the introduction of this fatal poison we have become less numerous and happy."
>
> — Little Turtle (Meshekinoquah), Miami chief, address to President Jefferson, 1802

1. Introduction

Alcohol occupied a distinctive place in the colonial encounter: deliberately introduced as a trade commodity, used as a tool of economic exploitation, and subsequently associated with elevated rates of harmful consumption among colonized populations (Saggers and Gray, 1998). Qualitative and case-level research has documented these links across diverse settings, from Aboriginal Australians to Indigenous North Americans to post-conflict sub-Saharan Africa (Brave Heart et al., 2011; Greywolf, 2020; Thomas et al., 2025). In parallel, the econometric literature demonstrates that colonial institutions shape long-run economic and health trajectories (Acemoglu et al., 2001; Nunn, 2008; Miller et al., 2018; Wispelwey et al., 2023).

These two lines of inquiry remain disconnected. The qualitative literature establishes that colonial history and alcohol harms are linked in specific populations, while the econometric literature shows that colonial institutions produce measurable, persistent effects on national outcomes. Yet no study has tested whether colonial intensity—the joint product of duration and institutional type—predicts national-level alcohol outcomes across the full range of formerly colonized and non-colonized states using panel data and spatially explicit methods. Bridging these literatures matters: if colonial history is a distal but measurable correlate of population-level alcohol harms, it has

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implications for how international health agencies allocate resources and frame alcohol policy in post-colonial contexts.

This study bridges these literatures in four ways. First, we construct a composite colonial intensity variable combining duration and colony type (extractive vs. settler) from COLDAT 3.0 (Becker, 2019), which enumerates and summarizes the number of years each country was governed by each colonial power. Second, we employ a balanced panel covering 178 countries over 21 years (2000–2020), enabling estimation of random-effects panel Spatial Durbin Models that account for both spatial dependence and unobserved country heterogeneity. Third, we estimate models under two theoretically motivated spatial weights matrices (geographic contiguity and shared-colonizer networks) and for both the full sample and the colonial-country subsample, producing eight primary specifications per dependent variable. Fourth, we examine four distinct WHO alcohol outcomes (alcohol consumption per capita, consumer prevalence, heavy episodic drinking, and AUD prevalence), covering the full range of colonial–alcohol relationships.

2. Colonial intensity as a distal determinant of health

Building on Acemoglu et al.'s (2001) distinction between extractive and settler colonies, we argue that colonial intensity—the joint product of duration and institutional type—generates differential long-run health trajectories. Extractive colonies, designed to exploit resources and labor with minimal investment in settler welfare, typically produced weaker institutional legacies, lower human capital investment, and more acute disruption of pre-existing social norms (Pérez Ramos et al., 2022). Settler colonies, while also harmful to indigenous populations, created different institutional structures that paradoxically produced higher post-independence development outcomes for the settler-descended majority population, while indigenous populations remained marginalized (Acemoglu et al., 2001).

Colonial intensity is not a binary condition but a continuous quantity shaped by the interaction of duration and institutional type. A country colonized for three centuries under extractive rule experienced qualitatively different institutional, cultural, and demographic disruptions than one colonized for three decades under settler administration. Duration compounds institutional effects: longer colonial periods allowed deeper penetration of extractive labor systems, more thorough displacement of indigenous governance structures, and more sustained disruption of pre-colonial social organization (Osterhammel, 1997/2005; Go, 2011). Most cross-national colonial legacy research relies on binary indicators (colonized versus non-colonized) or simple categorical classifications (extractive versus settler), collapsing substantial variation within each category. Our composite measure captures this variation by weighting duration against institutional type, producing a continuous scale that distinguishes, for example, among the very different colonial experiences of Nigeria (British extractive, 1861–1960), Australia (British settler, 1788–1901), and the Philippines (Spanish then American, 1565–1946).

Whether colonial legacies on health are stable over time matters here. Research on colonial legacies suggests that institutional effects of colonialism in Africa are declining over time as states develop autonomous trajectories. However, such research also notes that imprints on health, inequality, and ownership structures may prove more durable. This asymmetric persistence aligns with intergenerational trauma theory (Brave Heart et al., 2011), which posits that psychosocial wounds transmitted across generations through family systems, cultural practices, and epigenetic mechanisms are less responsive to institutional reform than economic indicators.

The relationship between colonialism and alcohol has a specific historical trajectory distinct from other colonial health legacies. European colonizers introduced distilled spirits as trade commodities, used alcohol as a medium of exchange in labor recruitment, and imposed licensing systems that restructured indigenous drinking practices (Saggers and Gray, 1998). In many settings, pre-colonial alcohol cultures centered on communal consumption of low-alcohol fermented beverages within ritual and social frameworks that regulated intake. Colonial disruption replaced these regulated systems with commercial markets in high-alcohol distilled products, while simultaneously destroying the social structures that had previously governed consumption (Thomas et al., 2025). Post-independence states inherited colonial alcohol regulatory frameworks (licensing laws, excise structures, age restrictions) that were designed to generate colonial revenue rather than to protect public health. The persistence of these regulatory architectures may partly explain why colonial history continues to shape contemporary alcohol outcomes decades after independence.

2.1. Three transmission pathways

We theorize three distinct but partially overlapping pathways through which colonial intensity translates into contemporary alcohol outcomes.

These pathways are not mutually exclusive; in practice, they likely operate simultaneously and interact. Economic deprivation heightens vulnerability to trauma-related coping through substance use, while weak institutions reduce the availability of treatment and support services for populations experiencing intergenerational trauma. Our empirical strategy does not attempt to decompose the relative contribution of each pathway definitively. Instead, we use sequential model entry (adding pathway indicators progressively) to observe how the colonial intensity coefficient changes as potential mediators are introduced. Coefficient stability across nested models is suggestive of which pathways mediate the association, though we acknowledge this approach cannot establish mediation conclusively without individual-level data or formal causal mediation analysis.

Pathway 1—Economic Deprivation: Colonial extraction systematically depleted economic resources and suppressed human capital formation. Post-independence poverty and economic inequality are established risk factors for alcohol misuse through material stress and coping mechanisms (Degenhardt et al., 2022). GDP per capita is the pathway indicator.

Pathway 2—Institutional Weakness: Extractive colonial governance created state institutions oriented toward resource extraction rather than social welfare. Weak regulatory capacity, poorly developed alcohol policy infrastructure, and inadequate addiction treatment services represent institutional legacies that facilitate higher-harm drinking. Miller et al. (2018) demonstrate that colonial institutional legacies predict contemporary universal health coverage, with extractive colonies exhibiting systematically weaker health systems. The Worldwide Governance Indicators (WGI; World Bank, 2025) composite and the Alcohol Policy Stringency Index (APSI; World Health Organization, 2023) are the pathway indicators.

Pathway 3—Intergenerational Trauma: Colonial violence, cultural destruction, forced displacement, and ongoing structural marginalization generate collective and intergenerational trauma (Brave Heart et al., 2011; Thomas et al., 2025; Walters et al., 2011). Brave Heart et al. (2011) identify four mechanisms: massive group trauma creating collective emotional wounds, intergenerational transmission through family socialization and cultural narratives, unresolved grief and loss, and ongoing structural oppression reinforcing trauma. These linkages appear across diverse settings: Greywolf (2020) documents shared histories of colonization and alcohol use among Native Hawaiians, Teyra and Wan-Jung (2022) trace historical trauma–alcohol pathways in Indigenous Taiwanese communities, and Toombs et al. (2022) demonstrate that intergenerational residential school attendance increased substance use among First Nations adults in Canada. Consistent with the self-medication hypothesis, populations experiencing historical trauma exhibit elevated rates of alcohol use disorder as a coping mechanism (Ertl et al., 2016; Smith and Cottler, 2018). We note a methodological limitation: because our data do not directly measure collective trauma, this pathway is identified only residually, if the colonial intensity

coefficient remains stable after controlling for economic and institutional mediators. Coefficient stability across nested models is suggestive but neither necessary nor sufficient for establishing a mechanism. The residual association is consistent with trauma/psychosocial mechanisms, but also with other unmeasured country-level factors (e.g., post-colonial conflict, diagnostic infrastructure legacies). We therefore treat all three pathways as candidate explanations that the current data cannot definitively distinguish.

3. Method

3.1. Sample and procedure

We used a balanced panel of 178 countries over 21 years (2000–2020) by compiling country-level data from multiple international databases, matching annual observations across all sources by ISO 3166 country code. Countries with missing alcohol indicators data were excluded (Montenegro, Serbia, South Sudan, Saudi Arabia), as were countries with missing public health expenditure data (North Korea, Venezuela, Zimbabwe, Iraq, Somalia, Timor-Leste, Afghanistan). The geographic contiguity W sample comprises 178 countries (3738 observations); the shared-colonizer W sample comprises 177 countries (3717 observations) due to one additional unmatched boundary. The colonial-only subsample comprises 135 countries (2835 observations).

All models use the same balanced panel to ensure comparability across specifications. Time-varying covariates (GDP per capita, governance quality, public health expenditure, alcohol policy stringency, and temperature) were measured annually; time-invariant variables (colonial intensity, Islamic population share, pre-colonial alcohol culture, and absolute latitude) were matched at the country level across all panel years.

3.2. Measures

Dependent Variables. We examine four WHO alcohol indicators drawn from the Global Health Observatory and Our World in Data (Ritchie and Roser, 2024), each capturing a distinct dimension of population alcohol burden. These dimensions are partially independent: a country may have moderate consumption per capita but high AUD prevalence if drinking is concentrated in harmful patterns among a subpopulation.

Alcohol consumption per capita measures aggregate alcohol availability and intake, defined as litres of pure alcohol consumed per person per year among adults aged 15 and older. This indicator combines recorded sales data with modelled estimates of unrecorded consumption.

Consumer prevalence captures participation in drinking, defined as the percentage of the adult population who consumed any alcohol in the past 12 months. This measure distinguishes abstaining populations from consuming populations.

Heavy episodic drinking prevalence captures harmful consumption patterns, defined as the percentage of the population aged 15 and older engaging in heavy episodic drinking (60+ grams of pure alcohol on at least one occasion) in the past 30 days, age-standardized.

Alcohol use disorder prevalence captures clinical pathological consequences, defined as the percentage of adults meeting ICD-10 or DSM-IV diagnostic criteria for alcohol dependence or harmful use. Fig. 2 maps the four WHO alcohol outcomes as country means over 2000–2020; both maps, presented at the end of the manuscript, illustrate the spatial distribution of the study's key variables.

Colonial Intensity. The main predictor is a composite measure of colonial intensity constructed from COLDAT 3.0 (Becker, 2019), which aggregates four major datasets (Correlates of War Project, 2017, Lange et al., 2006; Olsson, 2009; Wimmer and Min, 2006) covering eight European colonial powers. Supplementary entries for non-European colonialism (Japanese, Russian/Soviet, American) and European-on-European cases (e.g., Ireland) are drawn from Myers and Peattie (1984), Wimmer and Min (2006), Go (2011), and Hechter (1975). Colony type (settler vs. extractive) follows Acemoglu et al. (2001), supplemented by Osterhammel, 1997/2005. For countries colonized by multiple powers, the colonizer with the longest duration is used. The composite is constructed as ColInt_i = YearsColonized_i × TypeWeight_i, where TypeWeight = 1.5 for extractive colonies, 1.2 for mixed colonies, 0.75 for settler colonies, and 0 for non-colonized countries, then log-transformed (ln[ColInt + 1]) to address right-skew. Fig. 1 maps colonial intensity across the 178-country panel,

Control Variables. We controlled for several economic, institutional, geographic, and cultural factors known to influence national-level alcohol outcomes. Annual GDP per capita (World Bank WDI, PPP, constant dollars, log-transformed) captures economic development and alcohol affordability. We rely on GDP per capita as the sole indicator for the economic deprivation pathway because it is the only deprivation-related measure with complete annual coverage across all 178 panel countries for 2000–2020; alternative indicators such as the Gini

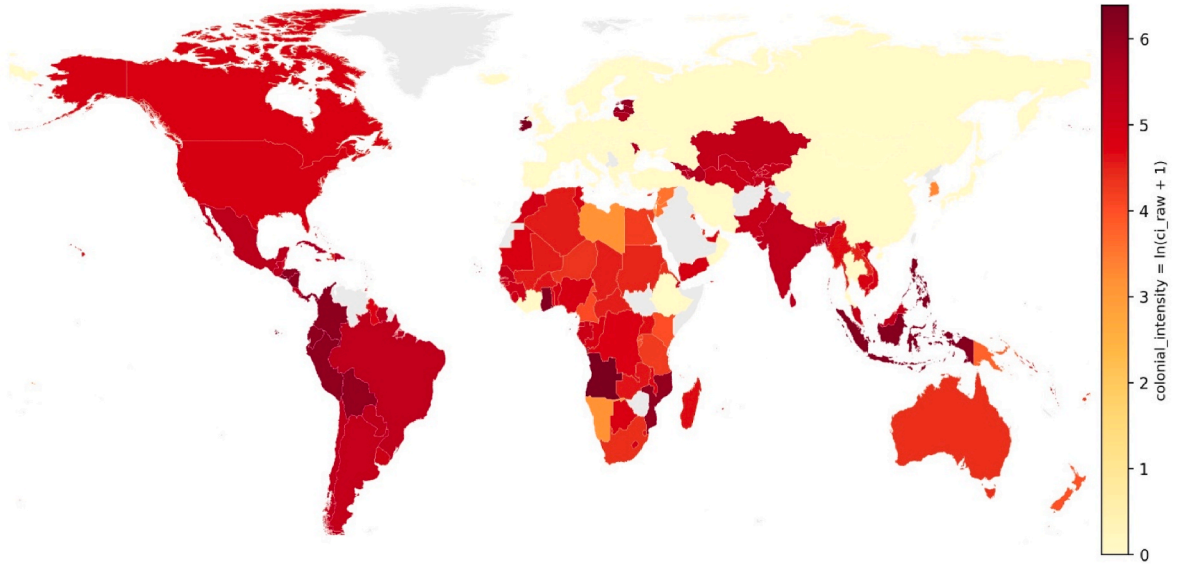

Fig. 1. Colonial intensity (the natural log of raw colonial intensity plus one) across the 178-country panel. Never-colonized countries take the value 0; countries outside the panel are shown in grey.

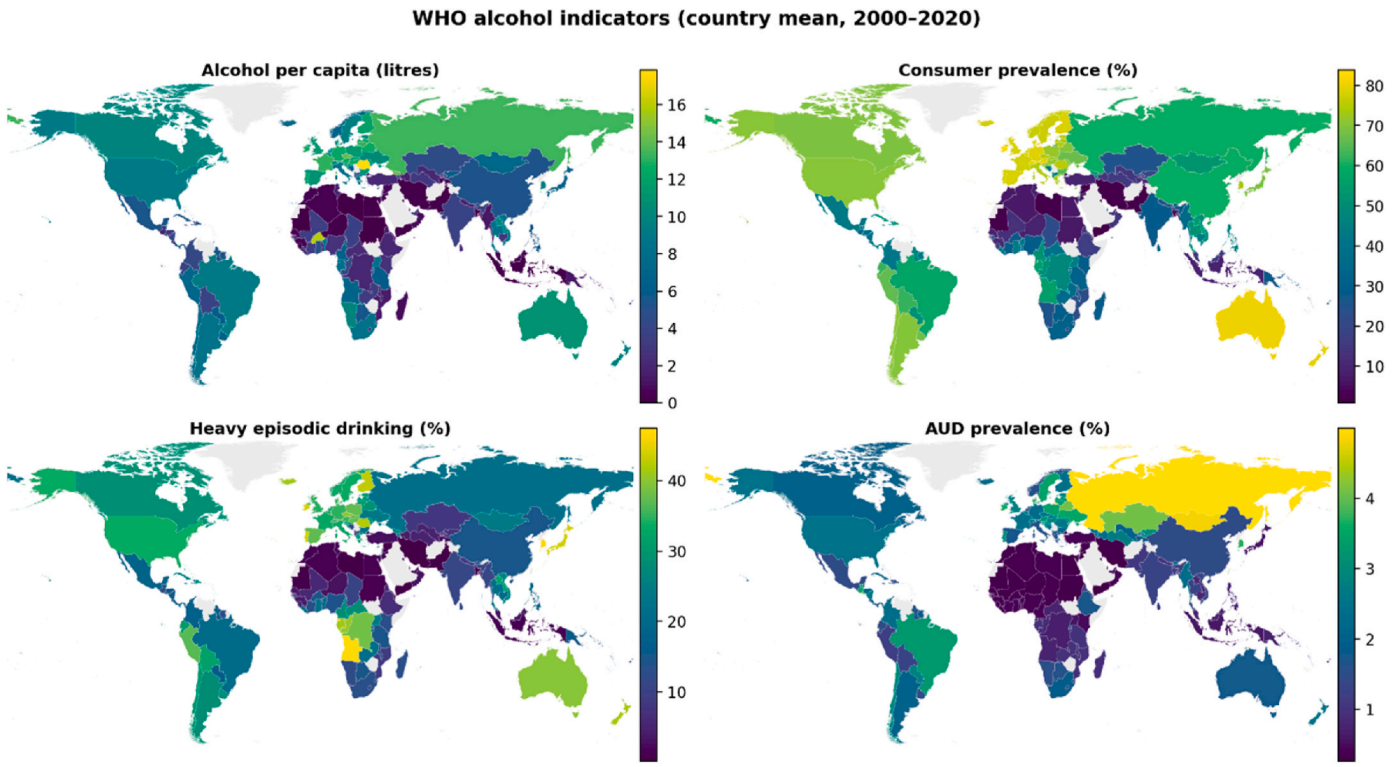

Fig. 2. The four WHO alcohol indicators, country means over 2000–2020: alcohol consumption per capita, consumer prevalence, heavy episodic drinking, and alcohol use disorder prevalence.

coefficient or poverty headcount ratios are unavailable for a substantial share of country-years, with missingness concentrated precisely in the formerly colonized countries central to our research question, and their inclusion as primary covariates would significantly break the balanced-panel design and induce selection on the variable of interest (over 50 percent missing observations). The Worldwide Governance Indicators composite (WGI; World Bank, 2025) captures institutional quality across six dimensions of governance, including rule of law and voice and accountability, thereby partially controlling for judicial fairness and rights protections. Public health expenditure as a percentage of total health spending (OECD, 2025) proxies for health system investment. The Alcohol Policy Stringency Index (APSI) is constructed from WHO GISAH indicators across four policy domains: physical availability (D1: national policy, sales restrictions, minimum legal age), pricing and taxation (D2: excise tax, VAT, government monopoly), marketing restrictions (D3: traditional advertising, digital/sponsorship, product placement/promotions), and drink-driving countermeasures (D4: BAC limits, enforcement). Each domain is scored 0–1, and the weighted composite follows Casswell et al. (2023), rescaled to 0–100. WHO policy data are available at 2 time points (2008 and 2016); values are linearly interpolated between survey waves and carried backward/forward for years outside the survey window, defensible because fewer than 15% of countries change major alcohol policies within any 5-year window (World Health Organization, 2023). Islamic population share (Pew Research Center) captures religious prohibition norms, log-transformed. Pre-colonial alcohol culture (Murdock Ethnographic Atlas/D-PLACE; Murdock, 1967) is coded as 0 = no indigenous alcohol tradition, 1 = fermented beverages (beer/mead), 2 = distilled spirits. Mean annual surface air temperature (Climate Change Knowledge Portal) and absolute latitude (Rest Countries API) are included as geographic controls.

3.3. Data analysis

We employed panel Spatial Durbin Models (SDMs) estimated via maximum likelihood using Stata 19.5, as country-level observations violate independence assumptions due to cultural diffusion, migration, and trade linkages (Anselin, 1988). We chose SDMs over standard panel regression for three reasons. First, cross-national alcohol data exhibit spatial dependence: neighbouring countries share cultural, economic, and regulatory environments that create correlated residuals. Standard panel models that ignore this structure produce biased standard errors and potentially misleading inference (Anselin, 1988; Gruda et al., 2024, 2026; Sharafi Zadegan et al., 2026). Second, the SDM framework allows simultaneous estimation of direct (own-country) and indirect (spillover) effects via the LeSage and Pace (2009) spatial multiplier decomposition. Third, the SDM nests the spatial autoregressive (SAR) and spatial error (SEM) models as special cases, providing a general specification that avoids pre-testing bias (Koley and Bera, 2024). Put differently, the Spatial Durbin Model decomposes the association between colonial intensity and each alcohol outcome into a direct effect (the association between a country's own colonial history and its own alcohol outcomes) and an indirect or 'spillover' effect (the association between a country's alcohol outcomes and the colonial history of the countries it is connected to, whether through shared borders or a shared former colonizer).

Colonial empires created dense networks of trade, migration, and cultural exchange that persisted after independence as shared legal systems, linguistic ties, diaspora communities, and economic relationships (Go, 2011). Since alcohol outcomes may diffuse both geographically (through cross-border trade, migration of drinking norms, and regional policy spillovers) and through these colonial network structures (through shared alcohol licensing systems, cultural transmission of drinking practices via diaspora communities, and policy learning among countries with shared colonial experiences) we employed two spatial weights matrices (geographic contiguity matrix (W) versus shared-colonizer matrix (W_sp)).

Random effects (RE) are employed because colonial intensity is time-invariant and would be absorbed by fixed effects. To validate this choice, Hausman specification tests were conducted on the time-varying covariates for all four dependent variables. All four tests rejected the null hypothesis: alcohol consumption per capita ($\chi^2(6) = 86.57$, $p < 0.001$),

consumer prevalence ($\chi^2(6) = 88.52$, $p < 0.001$), heavy episodic drinking ($\chi^2(6) = 21.33$, $p = 0.002$), and AUD ($\chi^2(6) = 84.19$, $p < 0.001$). These rejections indicate that FE is preferred for the time-varying covariates, creating a genuine tension: FE would eliminate the colonial intensity coefficient entirely (because it is time-invariant), yet RE may produce inconsistent estimates of time-varying covariate effects. We retain RE to identify the colonial intensity association, accepting this trade-off. The CRE models reported below (Section 4.3) partially address this problem by relaxing the RE orthogonality assumption; the colonial intensity–AUD association survives CRE estimation, suggesting the RE/FE discrepancy does not drive our main finding. We further test the sensitivity of the colonial intensity composite to alternative weighting schemes and report models with and without APSI to address interpolation concerns (see Supplementary Materials).

4. Results

4.1. Descriptive statistics and diagnostics

Table 1 presents panel descriptive statistics for the full sample (N = 3738 country-year observations; 178 countries over 21 years). Formerly colonized countries (N = 135) exhibit lower mean alcohol consumption per capita (4.66 vs. 9.24 L), lower consumer prevalence (37.4% vs. 63.8%), and lower AUD prevalence (1.41% vs. 2.31%) than non-colonized countries (N = 43), despite higher colonial intensity scores (mean = 4.93 vs. 0.00). This bivariate pattern reflects confounding by governance, GDP, and Islamic population share, which the multivariate models address.

Table 2 presents pairwise correlations among all variables. Colonial intensity is negatively correlated with all four alcohol outcomes: alcohol consumption per capita ($r = -0.40$), consumer prevalence ($r = -0.38$), heavy episodic drinking ($r = -0.29$), and AUD ($r = -0.27$), all $p < 0.001$. The strongest inter-predictor correlations are between WGI governance and GDP ($r = 0.79$), temperature and absolute latitude ($r = -0.86$), and between the three consumption-level DVs ($r = 0.77$–0.91). AUD correlates more modestly with the consumption indicators ($r = 0.45$–0.62), consistent with its status as a partially distinct clinical outcome.

Variance inflation factors (VIF) from pooled OLS diagnostics yield a mean VIF of 2.49 (range: 1.04 for year to 5.41 for absolute latitude). The temperature–latitude pair has the highest VIFs (4.35 and 5.41 respectively), consistent with their strong bivariate correlation; however, no VIF exceeds the conventional threshold of 10, and both variables serve distinct theoretical roles (climatic influence on alcohol metabolism vs. geographic positioning). We retain both predictors.

Non-spatial random-effects panel baselines (without spatial lags) provide a useful comparison. In these simpler models, colonial intensity is significant for consumer prevalence ($b = -1.808$, $p < 0.001$) and heavy episodic drinking ($b = -0.842$, $p = 0.009$), marginally significant

Table 1
Panel descriptive statistics.

Variable	Mean	SD
Alcohol consumption per capita (litres)	5.696	4.232
Consumer prevalence (%)	43.653	24.029
Heavy episodic drinking (%)	20.215	13.137
AUD prevalence (%)	1.633	1.069
Colonial intensity (ln)	3.741	2.230
WGI governance	−0.209	0.881
Log GDP per capita	8.400	1.473
Muslim % (ln)	1.957	1.697
Public health exp. (% GDP)	50.388	21.953
APSI (0–100)	44.459	19.402
Temperature (°C)	19.436	8.037
Absolute latitude	25.410	17.036

Note. N = 3738 (178 countries × 21 years). Colonial intensity is time-invariant; all other variables vary annually.

Table 2
Pairwise correlations among study variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Colonial intensity													
2. Alc. per capita	−0.40***												
3. Consumer prev.	−0.38***	0.91***											
4. Heavy episodic	−0.29***	0.77***	0.78***										
5. AUD prevalence	−0.27***	0.62***	0.58***	0.45***									
6. GDP per capita	−0.47***	0.55***	0.56***	0.32***	0.38***								
7. WGI governance	−0.42***	0.52***	0.54***	0.28***	0.35***	0.79***							
8. Public health exp.	0.05**	−0.03	−0.01	0.02	−0.06***	0.12***	0.14***						
9. APSI	0.08***	0.11***	0.14***	0.09***	−0.01	0.18***	0.21***	0.10***					
10. Islamic pop.	0.38***	−0.60***	−0.62***	−0.42***	−0.32***	−0.19***	−0.18***	−0.02	−0.08***				
11. Pre-colonial alc.	−0.20***	0.38***	0.36***	0.30***	0.22***	0.15***	0.12***	−0.01	0.06***	−0.44***			
12. Temperature	0.33***	−0.29***	−0.25***	−0.19***	−0.18***	−0.45***	−0.42***	−0.08***	−0.12***	0.25***	−0.05**		
13. Abs. latitude	−0.30***	0.31***	0.27***	0.17***	0.20***	0.48***	0.43***	0.06***	0.11***	−0.22***	0.08***	−0.86***	

Note. N = 3738 country-year observations. Pearson correlations. CI = colonial intensity (log); APC = alcohol per capita; CP = consumer prevalence; HED = heavy episodic drinking; AUD = alcohol use disorder prevalence; GDP = GDP per capita (log); WGI = World Governance Indicators; PHX = public health expenditure; APSI = Alcohol Policy Stringency Index; Islam = Islamic population share (log); PreAlc = pre-colonial alcohol culture; Temp = temperature; Lat = absolute latitude.
**p < 0.01. ***p < 0.001.

for alcohol consumption per capita (b = −0.223, p = 0.053), and non-significant for AUD (b = 0.030, p = 0.304). Notably, the sign of the AUD coefficient reverses from positive (non-significant) in the non-spatial model to significantly positive in the spatial Durbin specifications reported below, indicating that spatial dependence changes inference for this outcome.

Global Moran's I statistics (KNN = 5, year = 2010) confirm substantial spatial autocorrelation in all four dependent variables: alcohol consumption per capita (I = 0.553, z = 12.94, p < 0.001), consumer prevalence (I = 0.621, z = 14.49, p < 0.001), heavy episodic drinking (I = 0.548, z = 12.80, p < 0.001), and AUD (I = 0.588, z = 13.78, p < 0.001). Significant spatial clustering also persists in regression residuals after conditioning on all covariates: alcohol consumption per capita (I = 0.191, z = 4.64, p < 0.001), consumer prevalence (I = 0.333, z = 7.84, p < 0.001), heavy episodic drinking (I = 0.386, z = 9.14, p < 0.001), and AUD (I = 0.182, z = 4.37, p < 0.001). This residual spatial dependence confirms that a non-spatial model is misspecified and justifies the Spatial Durbin specification.

All 16 primary spatial Durbin models converge successfully. Table 3 summarizes model fit across specifications. The shared-colonizer W fails the Wald test of joint spatial significance for alcohol consumption per capita ($\chi^2 = 2.36$, p = 0.307 in the full sample; $\chi^2 = 3.54$, p = 0.170 in the colonial subsample). Colonial network linkages do not generate meaningful spatial dependence in aggregate consumption. In contrast, the spatial terms are highly significant for AUD ($\chi^2 = 158.8$ and 220.5, both p < 0.001) and heavy episodic drinking ($\chi^2 = 22.7$ and 19.2, both p < 0.001). Colonial networks transmit disorder risk and harmful drinking patterns, but not aggregate consumption levels.

4.2. Colonial intensity: direct, indirect, and total effects

Table 4 presents the central results: the direct, indirect, and total effects of colonial intensity on each dependent variable, decomposed via the LeSage-Pace spatial multiplier, across all eight specifications.

Colonial intensity has a significant positive direct effect in all eight specifications, with coefficients ranging from 0.065 (p = 0.032) to 0.238 (p = 0.012). This means that, holding all controls constant, a one-unit increase in log colonial intensity is associated with a 0.065–0.238 percentage point increase in AUD prevalence within the country itself. The effect is strongest in the colonial-only subsample under the shared-colonizer W (0.238), where comparison is restricted to countries with colonial histories and spatial connections reflect shared colonial heritage.

The AUD direct effect is accompanied by a significant negative indirect effect in three of four specifications (−0.104, p < 0.001 under geographic W; −0.291, p < 0.001 under shared-colonizer W full sample). This means countries neighbouring or sharing colonial heritage with intensely colonized nations tend to have lower AUD rates, conditional on their own characteristics. The resulting total effects are near zero or weakly negative, as the positive direct and negative indirect channels partially cancel.

For alcohol consumption per capita, colonial intensity shows no significant direct, indirect, or total effects in any specification. The coefficient is small and varies in sign, which fits the Islam suppression pattern: the concentration of Muslim-majority countries among extractive colonies masks any underlying positive association. For consumer prevalence and heavy episodic drinking, the full-sample results show significant negative total and indirect effects, driven by strong negative spillover. However, in the colonial-only subsample, direct effects flip positive (though imprecise), suggesting the full-sample negative effects are driven by the inclusion of non-colonial countries.

4.3. Robustness and additional analyses

To evaluate whether the colonial intensity–AUD association is robust to control variable specification and to identify potential suppression or mediation, we estimate a sequence of six nested SDM models (M1–M6) under the geographic W for the full sample. For AUD, the CI coefficient is non-significant in M1–M4 (Colonial Intensity only, +GDP, +WGI, +Islam), turns significant in M5 when cultural and geographic controls are added (b = 0.080, p = 0.005), and remains significant in M6, the full specification (b = 0.065, p = 0.032). This pattern indicates a suppression effect: confounders with opposing signs (particularly Islamic population share and pre-colonial alcohol norms) mask the positive colonial intensity–AUD association until they are controlled. As noted in Section 2.1, coefficient stability across nested models is suggestive rather than definitive for mechanism identification; nevertheless, the suppression pattern is informative about the confounding structure and confirms that the AUD association is not an artifact of omitted cultural variables.

For alcohol consumption per capita, colonial intensity is negative and significant in the first four models (M1: b = −0.283, p = 0.014; M4: b = −0.308, p = 0.005) but is fully attenuated once cultural and geographic controls (pre-colonial alcohol culture, temperature, and absolute latitude) enter at M5; in the full model the direct effect is small and non-significant (M6: b = −0.015, p = 0.898; total effect = −0.121, p = 0.506). This attenuation indicates that the apparent negative association between colonial intensity and per-capita consumption is largely accounted for by these cultural and geographic confounders, consistent with the null consumption effects reported in Table 4.

To test whether colonial effects are strengthening or weakening over

Table 3
Model fit summary across all specifications.

Specification	DV	Group	Pseudo R^2	Log-lik.	ρ	Wald spatial
Geo W, All countries	Alcohol per capita	178	0.506	−4385.0	0.409***	$\chi^2 = 261.6$***
	Consumer prevalence	178	0.680	−7570.3	0.299***	$\chi^2 = 117.5$***
	Heavy episodic drinking	178	0.635	−6966.6	0.248***	$\chi^2 = 67.1$***
	AUD prevalence	178	0.520	1417.3	0.445***	$\chi^2 = 261.5$***
Geo W, Colonial countries	Alcohol per capita	135	0.366	−3146.2	0.361***	$\chi^2 = 107.5$***
	Consumer prevalence	135	0.583	−5832.3	0.213***	$\chi^2 = 30.2$***
	Heavy episodic drinking	135	0.561	−5335.2	0.158***	$\chi^2 = 17.8$***
	AUD prevalence	135	0.387	1467.3	0.528***	$\chi^2 = 210.0$***
Col. W, All countries	Alcohol per capita	177	0.455	−4468.5	−0.029	$\chi^2 = 2.4$
	Consumer prevalence	177	0.684	−7585.3	0.091*	$\chi^2 = 7.5$*
	Heavy episodic drinking	177	0.632	−6948.8	0.150***	$\chi^2 = 22.7$***
	AUD prevalence	177	0.531	1352.3	0.394***	$\chi^2 = 158.8$***
Col. W, Colonial countries	Alcohol per capita	135	0.382	−3194.1	−0.069†	$\chi^2 = 3.5$
	Consumer prevalence	135	0.582	−5844.1	0.089*	$\chi^2 = 5.7$†
	Heavy episodic drinking	135	0.552	−5334.5	0.148***	$\chi^2 = 19.2$***
	AUD prevalence	135	0.409	1471.8	0.430***	$\chi^2 = 220.5$***

Note. ρ = spatial autoregressive parameter on DV lag. Wald spatial = joint test of spatial lag terms. All models were estimated with RE and ML via spxtregress. ***p < 0.001, **p < 0.01, *p < 0.05, †p < 0.10.

Table 4
Colonial intensity direct, indirect, and total effects (all 8 specifications).

DV	W matrix	Sample	Direct (SE)	Indirect (SE)	Total (SE)
Alcohol per capita	Geographic	All countries	−0.021 (0.120)	−0.100 (0.105)	−0.121 (0.181)
	Geographic	Colonial countries	0.138 (0.306)	−0.006 (0.102)	0.132 (0.381)
	Shared-col.	All countries	0.302 (0.395)	−0.397 (0.309)	−0.096 (0.149)
	Shared-col.	Colonial countries	0.312 (0.379)	−0.282 (0.420)	0.030 (0.352)
Consumer prevalence	Geographic	All countries	−0.745 (0.540)	−1.731*** (0.430)	−2.476** (0.732)
	Geographic	Colonial countries	2.314 (1.453)	−0.439 (0.370)	1.875 (1.653)
	Shared-col.	All countries	0.034 (1.670)	−2.241† (1.343)	−2.207** (0.733)
	Shared-col.	Colonial countries	2.071 (1.823)	0.413 (2.177)	2.483 (2.011)
Heavy episodic drinking	Geographic	All countries	−0.402 (0.312)	−0.454* (0.229)	−0.856* (0.405)
	Geographic	Colonial countries	0.822 (0.834)	0.032 (0.193)	0.854 (0.918)
	Shared-col.	All countries	1.213 (0.965)	−2.242** (0.776)	−1.029* (0.428)
	Shared-col.	Colonial countries	1.721 (1.059)	−2.400† (1.319)	−0.679 (1.257)
AUD prevalence	Geographic	All countries	0.065* (0.030)	−0.104*** (0.029)	−0.040 (0.048)
	Geographic	Colonial countries	0.161* (0.082)	−0.074† (0.041)	0.087 (0.117)
	Shared-col.	All countries	0.183* (0.087)	−0.291*** (0.075)	−0.108* (0.051)
	Shared-col.	Colonial countries	0.238* (0.095)	−0.143 (0.156)	0.095 (0.167)

Note. Standard errors in parentheses (Delta-method from estat impact). Direct = own-country effect; Indirect = spillover through W neighbours; Total = Direct + Indirect.
****p < 0.001, **p < 0.01, *p < 0.05, †p < 0.10.*

time, we add a Colonial Intensity × Year interaction term to the full SDM. For the three consumption outcomes, the interaction is highly significant and positive (alcohol consumption per capita: b = 0.007, p < 0.001; consumer prevalence: b = 0.007, p < 0.001; heavy episodic drinking: b = 0.010, p < 0.001), indicating that the negative colonial effects on consumption are attenuating over time at roughly 0.007–0.010 units per year. Over the 21-year panel, this implies approximately 15–20% erosion of the consumption penalty, in line with evidence that colonial institutional effects decline over time.

For AUD, however, the interaction is non-significant (b = 0.0002, p = 0.234), while the direct colonial intensity effect remains significant (b = 0.064, p = 0.032). The colonial–AUD association shows no evidence of temporal decay over two decades. This asymmetry fits several interpretations. Under the economic deprivation pathway, post-independence development should erode the consumption penalty over time (which it does), but clinical disorder may respond more slowly if it is driven by entrenched social conditions rather than current income. Under the trauma pathway, psychosocial wounds transmitted intergenerationally would be resistant to secular economic change, producing exactly the temporal stability observed for AUD. A less substantive explanation is also possible: WHO AUD estimates may be temporally smoother than consumption data because they rely partly on statistical modelling rather than annual surveys, making it harder to detect decay even if it exists. These interpretations are not mutually exclusive; partial contributions from all three are plausible.

Subsample analyses by colonizing power reveal heterogeneity. Among British colonies (N = 1,239, 59 countries), colonial intensity has a significant positive direct effect on AUD (b = 0.387, p = 0.040), matching the full-sample finding. Among French colonies (N = 525, 25 countries), the effect is null (b = 0.060, p = 0.258). For alcohol consumption per capita, the pattern reverses: British colonies show a marginally positive colonial intensity–alcohol consumption per capita association (b = 1.112, p = 0.099), while French colonies show a null effect (b = −0.182, p = 0.808).

This heterogeneity has a plausible explanation. British colonial administration was associated with widespread introduction of commercial alcohol markets and less restrictive alcohol regulation, while French colonies, particularly in Muslim-majority regions, operated under different cultural and regulatory contexts. The concentration of the AUD effect in British colonies suggests that the association varies with particular configurations of colonial rule—legal systems, alcohol licensing regimes, and psychiatric recording institutions—rather than operating uniformly across all colonial experiences.

Additional robustness checks are reported in the Supplementary Materials, including gender-disaggregated heavy episodic drinking (S3.1), alternative colonial intensity measures (S3.2), inverse distance spatial weights (S3.3), Mundlak-Chamberlain correlated random effects (S3.4; Mundlak, 1978), colonial intensity composite sensitivity (S3.5), and APSI interpolation robustness (S3.6). The colonial intensity–AUD association remains significant across nearly all alternative specifications. Control variable patterns and spatially lagged colonial intensity estimates are also reported in the Supplementary Materials (S1–S2).

5. Discussion

Colonial intensity has a consistently significant positive direct effect on AUD prevalence across all eight model specifications. To our knowledge, this is the strongest cross-national quantitative evidence to date that colonial history is independently associated with contemporary alcohol use disorders. The effect sizes (0.065–0.238 percentage points per unit of log colonial intensity) translate to approximately 0.3–1.2 percentage point differences in AUD prevalence between countries at the extremes of the colonial intensity distribution, holding all else constant. Given global AUD prevalence of approximately 5%, this amounts to a 6–24% relative difference, which is not trivial at population scale.

What else could explain the residual colonial intensity–AUD association? First, diagnostic infrastructure bias: British colonies inherited Western psychiatric recording systems that may inflate AUD detection rather than reflect higher true prevalence (Gone, 2013). The concentration of effects in British colonies is consistent with this alternative, though it does not explain why APSI reduces consumption but not AUD within the same diagnostic systems. Second, post-colonial conflict: intensely colonized countries experienced elevated post-independence conflict, which independently predicts substance use disorders. Colonial intensity may partly proxy for conflict exposure rather than (or in addition to) intergenerational trauma. Third, disruption of indigenous food systems: colonial extraction created nutritional deficiencies (thiamine, folate) that increase vulnerability to alcohol-related harm, a material pathway distinct from psychosocial trauma. Fourth, genetic population structure: allele distributions affecting alcohol metabolism (ADH1B, ALDH2) vary across populations and correlate with colonial history, though this is unlikely to explain the full pattern given the cross-national scale of our analysis. Future research should incorporate direct measures of collective trauma or psychosocial distress (e.g., conflict deaths from UCDP, mental health infrastructure indicators from WHO Mental Health Atlas) to adjudicate among these competing explanations.

The robustness checks confirm this finding survives sequential model entry, alternative spatial weights, CRE estimation, colonial intensity composite sensitivity analysis, and colonizer-type subsamples. The sequential entry analysis reveals a suppression effect, and the temporal interaction shows no evidence of decay over two decades for the AUD association.

Why do the positive direct effects on AUD pair with negative indirect (spillover) effects? In spatial Durbin models, the indirect effect captures the net influence of neighbours' colonial intensity on a country's own AUD, conditional on its own characteristics. A negative indirect effect means that countries whose neighbours are intensely colonized tend to have lower AUD, all else equal. Several mechanisms could produce this: compositional heterogeneity (intensely colonized countries in sub-Saharan Africa and South Asia are often adjacent to Muslim-majority countries where prohibition suppresses AUD, and the Islamic population control may not fully absorb this spatial pattern), reactive policy diffusion (countries neighbouring high-burden nations may adopt stricter regulations in response), or measurement artifact (countries with weaker health surveillance may underreport AUD while neighbouring countries with stronger systems report higher rates). We also note that in the SDM framework, negative indirect effects can arise mechanically when the spatial lag coefficient on colonial intensity is strongly negative, even without a substantive spillover mechanism; the indirect effect combines the spatial autoregressive parameter and the lag coefficient in a nonlinear fashion (LeSage and Pace, 2009, Ch. 2). We cannot adjudicate among these mechanisms with the current data. Under the shared-colonizer W, the negative indirect effect attenuates in the colonial-only subsample (-0.143, $p = 0.358$), suggesting the full-sample spillover is partly driven by the colonial and non-colonial contrast.

The failure of the shared-colonizer W to capture spatial dependence in alcohol consumption per capita (Wald $p = 0.307$), despite performing well for AUD (Wald $p < 0.001$) and heavy episodic drinking (Wald $p < 0.001$), is telling. Aggregate consumption appears driven by economic development, affordability, and religious norms, which vary independently of colonial heritage. Harmful drinking patterns and clinical disorder, by contrast, are more closely tied to colonial-network-specific mechanisms such as shared legal frameworks for alcohol regulation and common patterns of cultural disruption.

The consistent protective association of APSI with consumption-related outcomes (alcohol consumption per capita, consumer prevalence, heavy episodic drinking) but not with AUD prevalence matters for global health policy. Standard WHO-recommended alcohol policies (taxation, availability restrictions, advertising bans) reduce population-level consumption (where APSI is significant) but show no detectable association with AUD prevalence in any specification. This dissociation suggests that supply-side and pricing interventions are necessary but not sufficient: they lower aggregate intake without reaching the clinical disorder burden associated with colonial legacies. If the colonial–AUD association partly reflects intergenerational trauma or other unmeasured psychosocial factors, closing this gap may require a different class of intervention: trauma-informed, culturally specific approaches that operate through social cohesion, community agency, and mental health infrastructure rather than alcohol availability alone. Evidence-based models include New Zealand's Whānau Ora (Boulton et al., 2013), Canada's National Native Alcohol and Drug Abuse Program (NNADAP; Rowan et al., 2014), and Australia's Aboriginal Community Controlled Health Services (ACCHS; Campbell et al., 2018). These findings also connect to the WHO's Global Alcohol Action Plan 2022–2030 (World Health Organization, 2022), which increasingly recognizes equity as a core dimension of alcohol harm reduction. The question of whether former colonial powers bear responsibility for funding targeted AUD treatment in former colonies, a form of health reparation as argued for example by the CARICOM Reparations Commission's Ten-Point Plan, whose "Public Health Crisis" demand calls on European powers to help fund Caribbean health care (CARICOM Reparations Commission, 2014), is beyond our scope but follows from the results.

6. Limitations

Several limitations apply, organized here by type. Regarding identification and estimation, colonial intensity is time-invariant; despite the panel structure, causality cannot be established. Instrumental variable approaches using settler mortality or geographic instruments would strengthen identification but face data constraints at this scale. Relatedly, the random-effects assumption may be violated: although we report Mundlak-Chamberlain correlated random effects models as a robustness check, the Hausman test rejections for all four DVs indicate that unobserved country-level heterogeneity may correlate with both colonial intensity and time-varying controls, potentially biasing RE estimates. With 32 primary models and multiple effect decompositions, some individually significant p-values (particularly those near 0.05) should be interpreted with caution under multiple comparison frameworks; however, the consistency of the AUD finding across all eight specifications provides stronger evidence than any individual test. Regarding measurement, WHO alcohol indicators may suffer from measurement error, particularly in countries with large informal markets. More critically, AUD prevalence data derive from WHO estimates that combine survey data, treatment records, and statistical modelling. Countries with colonial histories, particularly British colonies, may have inherited Western psychiatric diagnostic infrastructure that detects and reports AUD at higher rates, not because of higher true prevalence, but because of diagnostic system calibration. This 'diagnostic infrastructure bias' alternative cannot be fully excluded without controlling for mental health system capacity (e.g., psychiatrists per capita from the WHO Mental Health Atlas), which we do not include in our primary models. Directly testing this alternative by including psychiatrists per capita as a control variable would help; without it, diagnostic infrastructure bias cannot be ruled out. An epistemological caveat also bears on measurement: this study applies Western diagnostic categories (AUD as defined by ICD/DSM) to populations across diverse cultural contexts. Some scholars argue that imposing Western nosological frameworks on colonized populations is itself a colonial practice (Gone, 2013), and differential cultural meanings of 'disordered' drinking may affect measurement validity. We acknowledge this tension while noting that the WHO data represent the best available cross-national comparable measures. Regarding scope and specification, the shared-colonizer W assumes shared colonial heritage creates meaningful connectivity; the non-significance for consumption confirms this holds selectively. Nation-level estimation cannot capture the heterogeneity of colonial experience within societies. Individuals and groups were exposed to colonial intensity in qualitatively different ways, and the buffering effects of privilege mean that nationally aggregated associations do not describe how harms are distributed within countries. Relatedly, country-level data cannot show how colonial intensity relates to the drinking patterns of subgroups within societies, multilevel and individual-level designs are needed to address these questions. Moreover, the Murdock Ethnographic Atlas assigns a single pre-colonial alcohol classification per country, which may misrepresent multi-ethnic states where diverse drinking traditions coexisted. Our models do not directly measure several cultural and institutional correlates of alcohol misuse such as human rights practices, judicial fairness, and histories of racism and eugenics. The WGI composite partially captures contemporary judicial quality and rights through its rule-of-law and voice-and-accountability dimensions, but no comparable cross-national panel measures historical racism or eugenic policy, and we recommend that future research account for them. Finally, whether colonial intensity has also left health legacies among colonizing populations and their descendants is an open question; future work could construct analogous measures of colonialism perpetrated for former imperial powers and test whether the disruptions of empire echo in their contemporary health outcomes as well.

7. Conclusion

This study provides the first cross-national panel spatial econometric test of colonial intensity as a predictor of national-level alcohol outcomes. Across 178 countries, 21 years, four dependent variables, two spatial weights matrices, and two sample definitions, colonial intensity is positively and consistently associated with alcohol use disorder prevalence through a direct (own-country) effect that survives all measured confounders. The finding that alcohol policy stringency reduces consumption but not disorder, combined with the stability of the AUD coefficient across institutional and economic controls, is consistent with intergenerational trauma as one plausible mechanism among several. Post-colonial health equity frameworks that ignore this history are incomplete; addressing the colonial legacy in AUD likely requires culturally specific, community-controlled approaches. A growing evidence base supports this direction. Gone (2013) theorizes how Indigenous cultural practices themselves function as mental health treatment, reconnecting people affected by historical trauma to their traditions and to each other, and Rowan et al. (2014) document promising outcomes for culture-based addiction interventions across diverse Indigenous settings. Such community-based approaches may be the necessary complement to the regulatory tools whose limits our results expose: policies that reduce how much populations drink, without addressing why the burden of disordered drinking falls where colonial history left it.

Ethics approval

This study used only publicly available, aggregated secondary data and did not involve human participants, primary data collection, or any identifiable personal information. Ethical approval was therefore not required.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used Claude (Anthropic) in order to improve the language and readability of the manuscript and to assist with revisions made in response to reviewer comments. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

CRediT authorship contribution statement

Milad Sharafi Zadegan: Conceptualization, Data curation, Formal analysis, Methodology, Writing – review & editing. **Dritjon Gruda:** Conceptualization, Methodology, Supervision, Writing – original draft.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2026.119600.

Data availability

We solely used publicly available secondary data. We describe which data were used in the methods section.

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