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NICOTINE AND ALCOHOL EXPOSURE AS MODIFIABLE CARDIOVASCULAR RISK FACTORS: EFFECTS ON BLOOD PRESSURE, VASCULAR FUNCTION, AND CLINICAL CARDIOVASCULAR OUTCOMES: A CONTEMPORARY REVIEW

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ABSTRACT

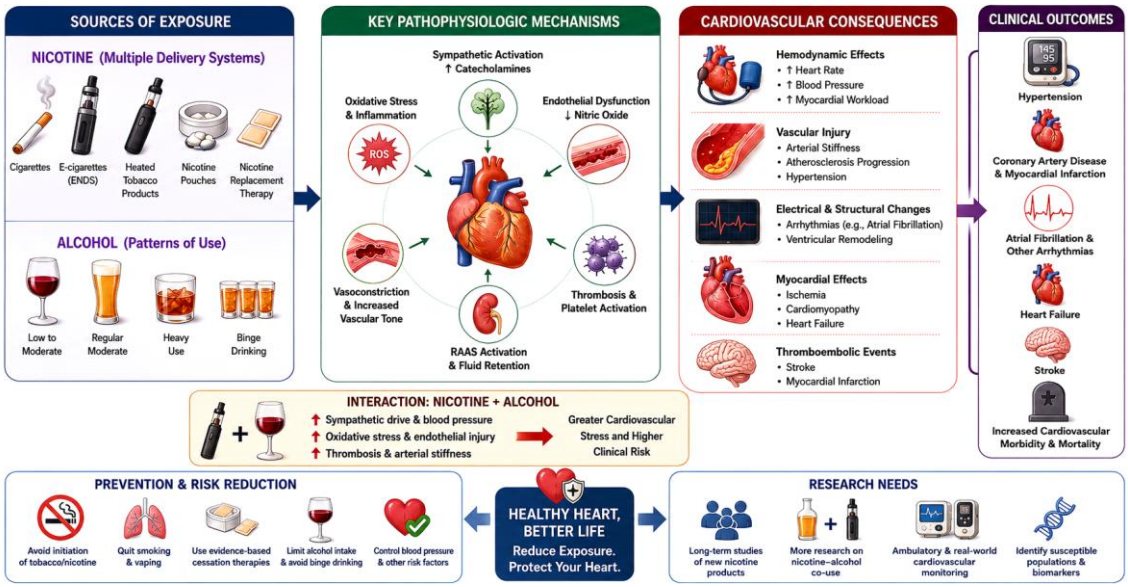
Nicotine and alcohol are widely consumed psychoactive substances with important and potentially interacting effects on cardiovascular health. This narrative review synthesizes recent evidence on nicotine delivered through combustible cigarettes, electronic cigarettes, heated tobacco products, smokeless tobacco, nicotine pouches, and nicotine-replacement therapy, together with low, moderate, heavy, and binge patterns of alcohol consumption. Nicotine acutely activates sympathetic and catecholaminergic pathways, increasing heart rate, myocardial workload, vasoconstriction, and blood pressure; experimental studies also implicate endothelial dysfunction, oxidative stress, arterial stiffness, and thrombogenicity. Combustible tobacco produces substantially greater toxicant exposure because nicotine is accompanied by carbon monoxide, oxidants, particulates, aldehydes, and other combustion products. Alcohol shows a dose-related association with blood pressure and incident hypertension, while heavy and binge drinking are consistently linked with atrial fibrillation, cardiomyopathy, stroke, and heart failure. Observational reports of cardiovascular benefit from low-to-moderate alcohol intake remain uncertain because of residual confounding, exposure misclassification, and inconsistent causal evidence. Human epidemiological studies suggest statistical interaction between smoking and alcohol use for blood pressure, arterial stiffness, and selected cardiovascular outcomes, although direct evidence for modern combinations such as nicotine vaping plus alcohol remains limited. Therapeutic nicotine replacement should be distinguished from recreational nicotine use because randomized evidence has not demonstrated the major cardiovascular-event profile established for

combustible smoking. Overall, reducing combustible tobacco exposure, preventing recreational nicotine initiation, supporting evidence-based cessation, and reducing heavy or binge alcohol consumption remain important cardiovascular prevention strategies. Long-term product-specific and co-exposure studies are still required.

KEYWORDS: Nicotine; Alcohol; Cardiovascular disease; Hypertension; Blood pressure; Endothelial dysfunction.

Abbreviations: AF (atrial fibrillation); AHA (American Heart Association); BP (blood pressure); CV (cardiovascular); CVD (cardiovascular disease); DBP (diastolic blood pressure); EC (electronic cigarette); EC+ (nicotine-containing electronic cigarette); EC− (nicotine-free electronic cigarette); ENDS (electronic nicotine-delivery systems); HR (heart rate); MAP (mean arterial pressure); MR (Mendelian randomization); NRT (nicotine-replacement therapy); PWV (pulse-wave velocity); RAAS (renin–angiotensin–aldosterone system); ROS (reactive oxygen species); SBP (systolic blood pressure).

Graphical abstract: Nicotine and alcohol-induced cardiovascular dysfunction from acute physiological effects to clinical cardiovascular disease.



1. INTRODUCTION: Cardiovascular disease (CVD) remains a leading cause of premature morbidity and mortality, and elevated blood pressure is one of its most important modifiable determinants. Tobacco exposure and harmful alcohol consumption frequently coexist with hypertension, dyslipidemia, obesity, diabetes, and other risk factors. These exposures are clinically important because they can affect autonomic tone, vascular resistance, endothelial

function, oxidative balance, thrombogenicity, and cardiac electrophysiology before overt cardiovascular disease becomes apparent.

The nicotine landscape has changed substantially. Nicotine is now delivered not only by conventional cigarettes, cigars, bidis, and waterpipes, but also by electronic cigarettes, heated tobacco products, smokeless tobacco, nicotine pouches, and medicinal nicotine-replacement products. These products should not be treated as biologically equivalent. Cigarette smoking combines nicotine with carbon monoxide, particulate matter, reactive aldehydes, oxidants, and other combustion products, whereas non-combustible products remove some toxicants but may still produce clinically relevant nicotine exposure and vascular effects [1-3].

The cardiovascular interpretation of alcohol has also evolved. Heavy and binge drinking are consistently associated with hypertension, atrial fibrillation (AF), cardiomyopathy, stroke, and heart failure, whereas the long-standing concept that low-to-moderate drinking is cardioprotective has become increasingly controversial. Recent dose-response studies, Mendelian-randomization analyses, and bias-aware evidence syntheses suggest that apparent benefit in observational cohorts may partly reflect residual confounding, former-drinker misclassification, and other lifestyle differences [4-6].

A further gap concerns co-exposure. Smoking and alcohol use commonly cluster, yet most studies analyze them separately. Available population studies indicate statistical interaction for blood pressure, arterial stiffness, and cardiovascular outcomes, while emerging preclinical work suggests that nicotine vaping combined with alcohol can alter myocardial molecular pathways [7-11]. This review therefore evaluates recent evidence on the independent and combined cardiovascular effects of nicotine and alcohol, with particular emphasis on blood pressure, vascular injury, and clinically relevant cardiac outcomes.

2. Literature approach: This article was prepared as a structured narrative review. Recent peer-reviewed human studies, randomized or controlled exposure studies, prospective and cross-sectional cohort studies, systematic reviews, meta-analyses, scientific statements, and selected mechanistic animal studies were included where required to clarify biological mechanisms. Literature published from 2019 through August 2026 was emphasized, while earlier landmark studies were retained when essential for interpretation. PubMed/MEDLINE and citation tracking were used to identify relevant evidence using combinations of terms related to nicotine, smoking, electronic cigarettes, heated tobacco, nicotine pouches, alcohol, ethanol, binge drinking, blood pressure, hypertension, endothelial function, arterial stiffness, myocardial infarction, atrial fibrillation, cardiomyopathy, heart failure, and stroke.

Because the evidence base is heterogeneous in exposure definition and study design, findings were synthesized narratively rather than pooled de novo. Greater weight was given to randomized human experiments for acute physiological effects, prospective cohorts for long-term outcomes, and systematic reviews or meta-analyses when several studies addressed the same endpoint. Observational associations were interpreted cautiously when reverse causation, former-user classification, smoking history, alcohol pattern, or other confounding could materially affect the result. The review is therefore not presented as a new systematic review or meta-analysis and does not claim PRISMA-level study enumeration.

3. Nicotine exposure and cardiovascular effects: Nicotine activates nicotinic acetylcholine receptors in autonomic ganglia and the adrenal medulla, increasing catecholamine release and sympathetic drive. The immediate cardiovascular consequences include tachycardia, increased myocardial contractility, vasoconstriction, and blood-pressure elevation. The magnitude of response depends on dose, route, speed of delivery, prior exposure, and tolerance. Importantly, total cardiovascular toxicity also depends on non-nicotine constituents of the product [3].

3.1 Product-specific context: Combustible cigarettes have the strongest established relationship with coronary disease, myocardial infarction, stroke, peripheral artery disease, heart failure, and cardiovascular mortality because they combine nicotine with combustion toxicants. Electronic cigarettes and heated tobacco products avoid conventional combustion but can still produce nicotine-related hemodynamic stress, arterial stiffening, endothelial dysfunction, and platelet activation. Nicotine pouches deliver nicotine through the oral mucosa and eliminate inhalational toxicants, but long-term cardiovascular event data remain limited. Medicinal nicotine replacement is different again: it provides controlled nicotine without smoke and is used to facilitate smoking cessation; randomized evidence has not demonstrated the major event profile associated with continued smoking [12].

Table 1. Cardiovascular relevance of major nicotine-delivery systems.

Product	Exposure profile	Main cardiovascular concern	Evidence interpretation	Reference/s
Combustible cigarettes	Rapid nicotine + combustion toxicants	↑ HR/BP, endothelial injury, thrombosis, atherosclerosis	Strongest long-term CVD evidence	[1,3,24]
E-cigarettes/vapes	Nicotine ± aerosol constituents	Acute ↑ HR/BP; arterial stiffness; endothelial dysfunction	Acute evidence consistent; long-term events less certain	[13-18,22,23]
Heated tobacco	Nicotine + heated-tobacco aerosol	Arterial stiffness and platelet activation	Human acute evidence; long-term data limited	[21]
Smokeless tobacco	tobacco-specific constituents + nicotine;	Sympathetic/hemodynamic effects	Product-specific long-term evidence incomplete	[2,17]
Tobacco-free nicotine pouches	nicotine without tobacco leaf.	Sympathetic/hemodynamic effects	Product-specific long-term evidence incomplete	[2,17]
Nicotine replacement therapy	Controlled gum/lozenge/patch exposure	Usually modest transient hemodynamic effects	Risk must be weighed against continued smoking	[12]

3.2 Hemodynamic and autonomic effects: Controlled human experiments consistently show acute cardiovascular activation after nicotine-containing vaping. Gonzalez and Cooke (2021) observed increased arterial pressure and heart rate with altered sympathetic activity after acute JUUL exposure in young nonsmokers [13]. Dimitriadis et al. (2022) similarly demonstrated acute increases in mean arterial pressure and heart rate with changes in skin and muscle sympathetic nerve activity [14]. In a meta-analysis of 15 studies involving 752 participants, nicotine-containing e-cigarettes increased systolic blood pressure by 3.41 mmHg, diastolic pressure by 3.42 mmHg, and heart rate by 5.36 beats/min compared with nicotine-free e-cigarettes [15]. These changes are transient in many experiments, but repeated episodes may increase cumulative hemodynamic load, particularly in people with hypertension or coronary disease.

Product comparisons demonstrate that nicotine is important but not the only determinant. Gernun et al. (2022) found acute increases in blood pressure and arterial-stiffness measures

after both JUUL and cigarette exposure [16]. Hauck et al. (2023) reported hemodynamic and arterial-stiffness changes after nicotine-containing e-cigarettes, cigarettes, and oral nicotine-containing products; some parameters also changed after nicotine-free vaping, implicating aerosol-related factors in addition to nicotine [17].

3.3 Endothelial dysfunction, arterial stiffness, oxidative stress, and thrombosis: The endothelium is an important early target. Haptonstall et al. (2020) reported impaired vascular endothelial function among young cigarette smokers and e-cigarette users [18]. Rezk-Hanna et al. (2024), using nicotine-containing and nicotine-free electronic hookah exposure, demonstrated that nicotine reduced flow-mediated dilation and nitric-oxide bioavailability while increasing reactive oxygen species, providing direct evidence for a nicotine-dependent component of endothelial dysfunction [19].

Arterial stiffness and thrombogenicity offer additional mechanistic links to clinical disease. Lyytinen et al. (2023) showed in a randomized double-blind crossover trial that nicotine-containing e-cigarette exposure increased platelet- and fibrin-dependent thrombus formation and impaired microvascular vasodilation [20]. In a separate randomized crossover study, brief IQOS use increased pulse-wave velocity by 0.365 m/s, heart-rate-corrected augmentation index by 6.22%, and platelet thrombus formation relative to no exposure [21]. These acute findings do not themselves quantify future myocardial-infarction risk, but they demonstrate biologically relevant vascular effects.

Recent syntheses reinforce this interpretation. Cheraghi et al. (2024) found that nicotine-containing e-cigarettes acutely increased arterial-stiffness indices and heart rate compared with nicotine-free devices [22]. Kundu et al. (2025) likewise found increased heart rate and blood pressure after acute vaping compared with non-use, while emphasizing that long-term clinical outcome data remain insufficient to equate exclusive vaping with combustible smoking [23].

Table 2. Selected recent evidence on nicotine-containing products and cardiovascular function.

Study	Design/exposure	Key finding	Interpretive value
[13]	Acute JUUL exposure	↑ arterial pressure and HR; altered sympathetic activity	Direct neurovascular response
[14]	Randomized sham-controlled vaping/smoking	↑ MAP and HR; autonomic changes	Supports sympathetic mechanism
[17]	Four-arm crossover	Hemodynamic/stiffness changes across nicotine products	Separates nicotine and product effects
[20]	Randomized crossover, EC ± nicotine	Nicotine increased thrombogenicity and impaired microvascular dilation	Direct human thrombosis evidence
[21]	Randomized IQOS crossover	↑ PWV, augmentation index, platelet thrombus formation	Heated tobacco not vascularly inert
[15]	Systematic review/meta-analysis	EC+ vs EC−: SBP +3.41, DBP +3.42 mmHg, HR +5.36 bpm	Quantifies acute nicotine effect
[23]	Systematic review/meta-analysis	Acute vaping ↑ HR and BP; long-term event evidence limited	Separates acute from chronic certainty

3.4 Clinical outcomes and the special case of NRT: The strongest long-term clinical evidence concerns cigarette smoking rather than isolated nicotine. Large prospective data show increased risk across a broad spectrum of cardiovascular outcomes among current smokers [24]. For newer products, recent network meta-analysis suggests an association between e-cigarette use and composite CVD, but outcome-specific evidence for myocardial infarction and stroke remains more uncertain and is complicated by dual use and previous smoking [25]. This distinction is essential when counseling smokers: complete cessation of combustible smoking is the primary objective. Nicotine-replacement therapy should not be equated with smoking; a systematic review of randomized trials found no statistically significant increase in clinically diagnosed arrhythmia, nonfatal myocardial infarction, nonfatal stroke, or cardiovascular death during studied treatment periods, although event numbers and follow-up were limited [12].

4. Alcohol consumption and cardiovascular effects: Alcohol has dose-, pattern-, and time-dependent cardiovascular effects. Acute exposure can alter vascular tone and autonomic balance, whereas repeated heavy exposure can promote hypertension, atrial arrhythmogenesis, myocardial remodeling, and cardiomyopathy. Consequently, average weekly intake should not be interpreted without considering binge episodes, duration of use, sex, comorbidity, and former-drinker status [6].

4.1 Blood pressure and hypertension: Blood pressure is one of the clearest cardiovascular endpoints associated with alcohol. Di Federico et al. (2023) analyzed longitudinal cohort data

and found a largely direct dose-response relationship between alcohol intake and blood pressure. Compared with no alcohol intake, approximately 12 g/day was associated with a 1.25-mmHg higher systolic pressure and 48 g/day with a 4.90-mmHg higher systolic pressure. The analysis did not identify a clear threshold below which systolic blood pressure was unaffected [26].

Cecchini et al. (2024) subsequently found a positive dose-response relationship between habitual alcohol consumption and incident hypertension across cohort studies, with sex-related differences in the shape and magnitude of risk. Mechanisms include sympathetic activation, altered renin-angiotensin-aldosterone signaling, impaired baroreflex control, oxidative stress, endothelial dysfunction, reduced nitric-oxide bioavailability, sleep disturbance, weight gain, and changes in renal sodium handling [27].

4.2 Atrial fibrillation, cardiomyopathy, heart failure, and stroke: Alcohol is also a well-established modifiable contributor to AF. Jiang et al. (2022), in a meta-analysis exceeding 10 million participants, reported an approximately 6% increase in AF risk for each additional drink per day overall. Acute binge-associated AF is often described as “holiday heart,” while chronic exposure may promote atrial enlargement, fibrosis, hypertension, oxidative stress, and autonomic instability [28]. Importantly, the randomized trial by Voskoboinik et al. (2020) showed that substantial alcohol reduction/abstinence in regular drinkers with AF reduced recurrence and overall AF burden [29].

Prolonged heavy drinking can directly injure the myocardium through acetaldehyde exposure, oxidative and mitochondrial injury, abnormal calcium handling, and altered protein synthesis, producing dilated cardiomyopathy and heart failure in susceptible individuals. Alcohol may also increase heart-failure risk indirectly through hypertension and arrhythmia. Higher alcohol intake is associated with greater stroke risk, mediated in part by hypertension and AF; these adverse effects are particularly important with heavy or binge patterns [6,30].

4.3 Is low-to-moderate alcohol cardioprotective?: The historical J- or U-shaped association between alcohol and coronary disease remains controversial. Some observational cohorts report lower ischemic risk among low-to-moderate drinkers, but such comparisons may be distorted by socioeconomic differences, healthier lifestyle patterns, former-drinker classification, and reverse causation. Biddinger et al. (2022) showed attenuation of apparent benefit after accounting for healthier lifestyle characteristics [4], and Carr et al. (2024) found discordance between conventional observational estimates and Mendelian-randomization evidence for ischemic heart disease [5]. A systematic review of Mendelian-randomization studies also supported likely causal associations between alcohol exposure and several

adverse cardiovascular outcomes, including hypertension and AF [31]. Accordingly, current evidence does not justify recommending alcohol initiation for cardiovascular prevention [6].

Table 3. Selected evidence linking alcohol exposure with cardiovascular outcomes.

Study	Evidence type	Outcome	Main conclusion
[29]	Randomized trial	AF recurrence	Alcohol abstinence/reduction lowered AF recurrence and burden
[4]	Large cohort/genetic analysis	CVD	Apparent low-dose benefit attenuated after lifestyle adjustment
[28]	Dose-response meta-analysis	AF	≈6% higher AF risk per additional drink/day overall
[26]	Dose-response meta-analysis	Blood pressure	Progressive SBP/DBP increase with increasing intake
[5]	Burden-of-proof analysis	Ischemic heart disease	Observational and MR evidence were discordant
[27]	Systematic review/meta-analysis	Incident hypertension	Increasing intake associated with higher hypertension risk
[6]	AHA scientific statement	CVD spectrum	Heavy/binge use harmful; low-level effects remain outcome-dependent

5. Combined nicotine/tobacco and alcohol exposure: Nicotine/tobacco and alcohol use frequently cluster, and their cardiovascular pathways overlap. Nicotine produces rapid catecholaminergic stimulation, tachycardia, vasoconstriction, and blood-pressure elevation; repeated or heavy alcohol exposure can increase sympathetic activity, blood pressure, oxidative stress, and endothelial dysfunction. Co-use may therefore increase hemodynamic load through convergent autonomic and vascular pathways. However, most human studies evaluate smoking plus alcohol rather than isolated nicotine plus alcohol, so observed interactions cannot be attributed to nicotine alone.

In the Guizhou Population Health Cohort, Gao et al. (2023) identified interaction between smoking and alcohol in incident hypertension; heavy smoking combined with heavy drinking was associated with markedly higher risk than non-smoking/non-drinking [7]. Vallée (2023a) reported synergistic statistical effects of smoking indices and alcohol intake on systolic and diastolic blood pressure in UK Biobank participants [9], while Vallée (2023b) identified interactions for arterial stiffness [10]. Palatini et al. (2023), following young-to-middle-aged patients with stage 1 hypertension for approximately 17 years, found that alcohol use amplified smoking-associated cardiovascular and renal risk [8].

These data support the clinical relevance of joint exposure but do not prove universal biological synergy. Terms such as “combined,” “interactive,” or “potentially more-than-additive” are more defensible than asserting synergy for all outcomes. Direct human evidence

on simultaneous vaping plus alcohol or nicotine-pouch plus alcohol exposure remains sparse. Harris et al. (2026) showed that chronic-plus-binge alcohol combined with nicotine vaping altered the ventricular proteome in mice, providing mechanistic plausibility but not a quantitative estimate of human cardiovascular risk [11].

Table 4. Key evidence on combined tobacco/nicotine and alcohol exposure.

Study	Population/design	Endpoint	Interpretation
[7]	Prospective cohort, n=5,625	Incident hypertension	Heavy smoking + heavy drinking showed higher risk; interaction reported
[8]	Stage 1 hypertension; long follow-up	CV/renal events	Alcohol amplified adverse smoking-associated outcomes
[9]	UK Biobank	SBP, DBP, hypertension	Smoking–alcohol statistical interaction for BP
[10]	UK Biobank	Arterial stiffness	Joint exposure associated with higher stiffness
[11]	Preclinical mouse model	Ventricular proteome	Modern vaping + alcohol co-exposure altered cardiac molecular pathways

6. Clinical Synthesis: Hypertension is the most consistent shared intermediate phenotype in the present evidence. Nicotine acutely increases blood pressure through sympathetic and vascular mechanisms, whereas habitual alcohol consumption shows a more clearly established dose-related relationship with sustained blood-pressure elevation and incident hypertension. Co-exposure may further increase blood-pressure burden, especially in individuals who already have hypertension. Dietary sodium is another modifiable determinant of blood pressure; salt reduction has also been emphasized as a practical measure for maintaining normal blood pressure [34].

For coronary artery disease and myocardial infarction, cigarette smoking remains the exposure with the strongest causal and long-term epidemiological evidence. Newer nicotine products produce acute changes relevant to atherothrombosis, but long-term product-specific event risks remain less certain because of limited follow-up, dual use, and prior smoking. Alcohol has a more complex relationship with ischemic disease at low intake, whereas heavy consumption increases overall cardiovascular burden through hypertension, arrhythmia, and myocardial injury.

For arrhythmias, alcohol has the clearest clinical association with AF and is supported by intervention evidence showing benefit from reduction. Nicotine can increase sympathetic tone and electrical excitability, providing biological plausibility for arrhythmogenesis, but direct clinical risk differs greatly across smoking, recreational nicotine, and medicinal NRT. For cardiomyopathy and heart failure, chronic heavy alcohol exposure is an established toxic

myocardial exposure, while smoking contributes strongly through coronary disease, vascular injury, and increased afterload.

The practical implication is that cardiovascular risk assessment should record product type and pattern rather than simply asking whether a person “uses nicotine” or “drinks alcohol.” Clinicians should distinguish combustible smoking from non-combustible nicotine and therapeutic NRT, quantify alcohol in grams or standard drinks while identifying binge patterns, and specifically ask about co-use. Patients with hypertension, AF, coronary disease, heart failure, chronic kidney disease, diabetes, or established vascular disease are likely to be particularly vulnerable to repeated hemodynamic and vascular stress. In the Indian context, alcohol-related cardiovascular prevention also sits within a broader public-health landscape shaped by heterogeneous policies and alcohol-related health and social hazards [32].

7. CONCLUSIONS AND FUTURE DIRECTIONS: Current evidence supports nicotine/tobacco and alcohol as important modifiable cardiovascular exposures, but their risk profiles are not interchangeable. Nicotine produces reproducible acute sympathetic and hemodynamic effects and can contribute to endothelial dysfunction, arterial stiffness, oxidative stress, and thrombogenicity. Combustible cigarettes remain substantially more hazardous because nicotine is delivered with a complex mixture of cardiovascular toxicants. Electronic cigarettes, heated tobacco, and nicotine pouches should not be considered cardiovascularly inert, yet the magnitude of their long-term clinical-event risk remains less certain. Therapeutic nicotine replacement should be evaluated separately because its purpose, dose pattern, and toxicant profile differ markedly from recreational smoking.

Alcohol demonstrates a convincing dose-related association with blood pressure and hypertension. Heavy and binge drinking additionally increase the risk of AF, cardiomyopathy, stroke, and heart failure. The hypothesis that low-to-moderate intake is universally cardioprotective is not supported strongly enough to justify initiating alcohol consumption for cardiovascular benefit.

The most important knowledge gaps concern long-term cardiovascular outcomes in exclusive users of modern nicotine products and the effects of concurrent nicotine and alcohol exposure. Future studies should use objective exposure biomarkers, 24-hour ambulatory blood-pressure monitoring, continuous ECG or wearable rhythm monitoring, vascular-function testing, and prospective assessment of myocardial infarction, stroke, heart failure, and cardiovascular mortality. Studies should also distinguish never-smokers, former smokers who switch completely, dual users, and users of high-dose oral nicotine. For alcohol, repeated

exposure measurements and intervention studies of reduction or abstinence are needed to clarify reversibility and the low-dose portion of the dose-response curve.

More broadly, effective addiction management may require pharmacotherapy when indicated, together with education, counselling, and psychosocial support [33]. Reducing heavy or binge alcohol exposure remains an important component of cardiovascular risk reduction. When tobacco/nicotine and alcohol are used together, clinicians and researchers should treat co-exposure as a potentially important cardiovascular risk context rather than assuming that each behavior acts independently.

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