RESEARCH PAPER

Critical Appraisal of the Obesogenic and Diabetogenic Potential of Ayurvedic Classified Alcoholic Beverages in the Context of Caloric Load, Carbohydrate Content, and Hepatotoxic Mechanisms: A Critical Review

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ABSTRACT

Background: Alcohol consumption is a significant and modifiable risk factor for obesity (*Sthaulya*), Type 2 Diabetes Mellitus (T2DM) (*Madhumeha*), and alcoholic liver disease (ALD). The classical Ayurvedic compendium *Sushruta Samhitā* enumerates and classifies alcoholic preparations — the *Madya Varga* — assigning each specific pharmacological properties and dietary indications or contraindications. Despite the growing global burden of alcohol-related metabolic disorders, the *Sushrutokta Madya Varga* has received limited evaluation in peer-reviewed biomedical literature.

Objectives: To systematically evaluate the *Madya Varga* as described in the *Sushruta Samhitā* and allied classical texts from the dual perspectives of Ayurvedic pharmacology and contemporary nutritional and hepatological science; and to assess their pro-obesity and diabetogenic potential using caloric content, carbohydrate load, and glycaemic index as objective parameters.

Methods: A comprehensive review of primary Sanskrit texts — principally *Sushruta Samhitā Sūtrasthāna* 46, *Ashtānga Hridayam Sūtrasthāna* 6, and *Dhanvantari Nighaṇṭu* — was undertaken, supplemented by a systematic electronic search of PubMed/MEDLINE, Scopus, Embase, IndMED, and the AYUSH/CCRAS database for peer-reviewed publications from 2000 to 2024 on alcohol consumption, metabolic disease, hepatotoxicity, caloric content, and glycaemic index.

Results: The *Sushruta Samhitā* identifies 12 distinct types of alcoholic preparations, categorised by fermentation substrate, age of preparation, and physiological properties. The text broadly classifies alcoholic beverages as *Kaphakāraka* (obesogenic) and explicitly contraindicates them in *Madhumeha*. Nutritional analysis reveals that distilled spirits (whisky, vodka, rum, gin, brandy) carry the highest caloric burden (≈207 kcal/100 mL), while red wine — the closest modern analogue to *Mārdvīka Madya* — carries the lowest caloric and carbohydrate load among common alcoholic beverages. The patho-mechanisms of ethanol-induced hepatic injury — including ADH/CYP2E1-mediated acetaldehyde production, oxidant stress, NADH redox imbalance, and mitochondrial dysfunction — are examined in the context of classical Ayurvedic hepatoprotective principles.

Conclusion: The dietary prescriptions of the *Sushrutokta Madya Varga* demonstrate clinically coherent alignment with contemporary nutritional epidemiology and hepatology. The Ayurvedic categorisation of alcoholic beverages by substrate, fermentation state, and physiological property constitutes an empirically valid classificatory framework that warrants formal integration into evidence-based integrative medicine practice.

Keywords: *Madya Varga*; *Sushruta Samhitā*; Ayurvedic dietetics; alcoholic beverages; glycaemic index; obesity; diabetes mellitus; alcoholic liver disease; hepatotoxicity; *Madhumeha*; *Sthaulya*; caloric content; integrative medicine

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1. INTRODUCTION

Alcohol consumption is one of the most prevalent and consequential dietary exposures in the modern world. The Global Burden of Disease (GBD) 2019 study estimated that alcohol was the seventh leading risk factor for premature death and disability-adjusted life years (DALYs) globally, responsible for 2.8 million deaths annually.[1] The World Health Organization (WHO) classifies alcohol as a Group 1 carcinogen and causally links it to more than 200 disease and injury conditions.[2]

The metabolic consequences of alcohol consumption are extensive and bidirectional. Ethanol is a calorie-dense macronutrient (7 kcal/g) that displaces essential nutrients without contributing micronutrient value, contributing to both energy excess and malnutrition.[3]

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Epidemiological evidence consistently associates heavy alcohol consumption with insulin resistance, visceral adiposity, dyslipidaemia, and progression to Type 2 Diabetes Mellitus (T2DM).[4] [5] In India, the National Family Health Survey-5 (NFHS-5, 2019–21) reported that approximately 18.7% of men aged 15 years and above consume alcohol, with increasing prevalence in urban settings.[6] The Indian Council of Medical Research (ICMR) has identified alcohol misuse as a rising contributor to the national burden of non-communicable diseases (NCDs).[7]

Remarkably, the ancient Indian surgical compendium *Sushruta Samhitā* (circa 600 BCE) devotes a dedicated chapter to the classification of alcoholic beverages — the *Madya Varga* — within its *Sūtrasthāna* (Chapter 46) and discusses their pharmacological properties, indications, contraindications, and hepatotoxic consequences in considerable detail.[8] The text's explicit condemnation of most alcoholic preparations in individuals with *Madhumeha* (diabetes mellitus) and *Sthaulya* (obesity), and its nuanced distinction between old (*Purāṇa*) and new (*Nava*) preparations, anticipates the modern nutritional evidence on fermentation kinetics and glycaemic response.

The establishment of the Ministry of AYUSH by the Government of India in 2014 [9] and the formal recognition of Ayurvedic medicine in the National Health Policy 2017[10] have renewed scholarly interest in the biomedical validation of classical Ayurvedic dietary principles. The *Sushrutokta Madya Varga* offers a particularly compelling case study: a pre-modern classification system that, as this review demonstrates, encodes a scientifically verifiable understanding of alcohol metabolism, caloric burden, and metabolic risk.

2. AIMS AND OBJECTIVES

1. To examine the classical Ayurvedic classification and pharmacological properties of alcoholic beverages (*Madya Varga*) as described in the Sushruta Samhitā, Ashtānga Hridayam, and allied Nighaṇṭu texts.
2. To identify alcoholic beverages indicated and contraindicated in *Madhumeha* (T2DM) and *Sthaulya* (obesity) as per classical Ayurvedic texts.
3. To compile and critically analyse the nutritional profile — including caloric content, carbohydrate value, alcohol content, and glycaemic index — of modern alcoholic beverages and their classical Ayurvedic analogues.
4. To evaluate the patho-mechanisms of ethanol-induced hepatic injury described in modern hepatology in the context of Ayurvedic principles of liver (*Yakṛit*) protection.
5. To draw evidence-based correlations between Ayurvedic pharmacological properties and contemporary nutritional epidemiology of alcohol consumption in metabolic disease.

3. MATERIALS AND METHODS

3.1 Classical Textual Sources

Primary classical Sanskrit sources reviewed include: (i) *Sushruta Samhitā — Sūtrasthāna* Chapter 46 (*Dravyasaṅgrahaṇīya Adhyāya*), which enumerates the *Madya Varga*; (ii) *Ashtānga Hridayam* of Vāgbhaṭa — *Sūtrasthāna* Chapter 6 (*Dravyādi Vijñānīya*); (iii) *Dhanvantari Nighaṇṭu*; (iv) *Rāja Nighaṇṭu*; (v) *Kaideva Nighaṇṭu*; and (vi) *Arkaprakāśa*, which specifies therapeutic alcoholic preparations including *Rājavarūṇī*. Sanskrit ślokas were drawn from Kaviraj Kunja Lal Bhishagratna's critical edition and verified against the Chaukhamba Sanskrit Pratishthan edition.[11]

3.2 Electronic Literature Search

A systematic literature search was conducted from January to May 2024 across PubMed/MEDLINE, Scopus, Embase, Google Scholar, IndMED (NIC India), the CCRAS digital repository, and the WHO Global Status Report on Alcohol and Health database. Boolean search strings combined the following Medical Subject Headings (MeSH) and free-text terms: 'alcohol consumption', 'ethanol metabolism', 'glycaemic index alcoholic beverages', 'alcohol diabetes mellitus', 'alcohol obesity', 'alcohol liver disease', 'Ayurveda alcohol', 'Sushruta Madya', 'fermented beverages India', 'wine caloric content', and 'alcoholic hepatitis pathogenesis'. Systematic reviews, meta-analyses, randomised controlled trials (RCTs), and large prospective cohort studies published between 2000 and 2024 were prioritised. Only full-text articles indexed in PubMed or Scopus were included.

3.3 Nutritional Data Sources

Caloric, carbohydrate, and alcohol content data for each beverage category were sourced from:[11] (i) the National Institute of Nutrition (NIN), Hyderabad — *Nutritive Value of Indian Foods*;[12] (ii) the USDA FoodData Central database;[13] (iii) the International Tables of Glycaemic Index and Glycaemic Load by Atkinson et al.;[14] and (iv) the ICMR–NIN Dietary Guidelines for Indians (2024).[15] Alcohol content is expressed as percentage volume per volume (% v/v).

4. REVIEW OF LITERATURE

4.1 Historical and Textual Context of Madya Varga

The *Sushruta Samhitā*, attributed to the great surgeon Suśruta and compiled approximately in the 6th century BCE, contains one of the most elaborate pre-modern pharmacological classifications of fermented and distilled beverages. Chapter 46 of the *Sūtrasthāna* — the *Dravyasaṅgrahaṇīya Adhyāya* — devotes detailed attention to the *Madya Varga*, cataloguing preparations by their fermentation substrate, age, physiological properties, and therapeutic or contraindicated status. The classification reflects a sophisticated empirical understanding of fermentation chemistry, caloric content, and metabolic effects that is without parallel in ancient medical literature. The text's general characterisation of alcoholic beverages is succinctly summarised in the *Ashtānga Hridayam Sutrasthana* chapter six. This verse encodes several clinically important properties of alcohol: it is *Dīpana* (digestive

fire stimulant), *Tīkṣṇa* (sharp/penetrating), *Uṣṇa* (hot in potency), *Srotas-viśodhana* (channel-clearing), and when used judiciously (*yuktyā*), it reduces *Kapha* and *Vāta*. Crucially, the qualifier *yuktyā pītam* (consumed with wisdom/in appropriate measure) signals that indiscriminate use — particularly in metabolically compromised individuals — is contraindicated. The reference to both *kṛśa* (thin/emaciated) and *sthūla* (obese) persons receiving benefit reflects the context-dependent nature of Ayurvedic dietary recommendations.[16]

4.2 Classification of Madya Varga: Individual Beverages and Their Properties

4.2.1 Mārdvīka Madya (Wine from Grapes)

Wine prepared from grapes (*Drākṣā*) by fermentation with the skin intact. It is described as *Madhura* (sweet in post-digestive effect), *Laghu* (light), *Kaṣāya-anurasa* (with an astringent after-taste), *Laghupāki* (easily digestible), and *Viṣamajvara-nāśana* (therapeutic in intermittent fever). Uniquely, it is stated to be permissible even in *Raktapitta* (haemorrhagic disorders) — the only alcoholic beverage accorded this distinction. Its modern analogue is red wine, which carries the lowest caloric and carbohydrate burden among common alcoholic beverages (68 kcal/100 mL; 2.5 g carbohydrate/100 mL), lending scientific support to its comparatively favourable Ayurvedic classification.[17]

4.2.2 Kharjūra Madya (Date Palm Wine)

Kharjūra Madya is fermented from date palm (*Kharjūra*, *Phoenix dactylifera*). It is described as *Vātakopana* (aggravates Vāta), *Viṣada* (clarifying), *Rucya* (appetising), *Kaphagna* (reduces Kapha), *Karśana* (reducing body mass), and *Laghu* (light). Its modern analogue is palm wine or date brandy, known to have moderate caloric content. The *Karśana* property is clinically relevant in the context of weight management, though its *Vātakopana* nature limits its use in individuals with Vāta predominance.[18]

4.2.3 Surā Madya (Grain-based Spirit/Beer)

Surā is the primary grain-fermented beverage, prepared from fermented cereals or rice. It is described as *Hṛdya* (cardiotonic), *Sugandhī* (aromatic), *Indriya-bodhana* (stimulates sensory clarity), *Bṛṃhaṇa* (nourishing), and *Dīpanī* (kindles digestive fire). It is indicated in cough (*Kāsa*), haemorrhoids (*Arśas*), malabsorption (*Grahaṇī*), and respiratory disorders (*Śvāsa*). Its modern analogue is beer or grain-based spirits. The *Bṛṃhaṇa* and *Dīpana* properties correspond with the known caloric density and appetite-stimulating effect of ethanol.[19]

4.2.4 Prasannā Madya (Clarified Rice Wine)

Prasannā is a clarified, white-coloured rice wine described as *Mūtrakṛit* (diuretic), *Stanya-kara* (galactagogue), and *Māṃsa-kara* (tissue-building). It relieves nausea, anorexia, abdominal colic, and distension. Its galactagogue property parallels the modest epidemiological evidence suggesting that low-alcohol fermented beverages may marginally stimulate prolactin — an effect now approached with caution given alcohol's established reproductive toxicity.[20] [21]

4.2.5 Śārkarā Madya (Sugar-cane/Molasses Spirit)

Śārkarā Madya is prepared from fermented sugar-rich plants, including sugarcane (*Ikṣu*). It is described as *Madhura* (sweet), *Dīpana* (digestive stimulant), *Basti-śodhana* (cleanses urinary bladder), and *Hṛdya* (cardiotonic). Its modern equivalents include rum and cane-based spirits. Notably, these carry a high caloric burden (≈207 kcal/100 mL) with negligible carbohydrate (due to complete fermentation of sugars), making them particularly obesogenic through empty-calorie mechanisms.[22]

4.2.6 Surāsava (Fermented Herbal Wine)

Surāsava represents medicated fermented preparations. It is described as *Tīkṣṇa* (sharp), *Hṛdya* (cardioprotective), *Mūtrala* (diuretic), *Kapha-Vāta-nut* (pacifies Kapha and Vāta), and *Sthiramada* (produces stable intoxication). This category corresponds to classical Ayurvedic medicated wines (*Āsava*) used therapeutically and has a modern analogue in herbal liqueurs with established pharmacological profiles.[23]

4.2.7 Madhvāsava (Honey-based Wine/Mead)

Madhvāsava (mead) is prepared by fermentation of honey. It is *Laghu* (light), *Chedī* (expectorant), *Meha-apaha* (urinary disease-relieving — directly relevant to *Madhumeha*), *Tīkṣṇa* (sharp), and *Śoṣaghna* (relieves wasting). The explicit designation *Meha-apaha* marks this as the single Ayurvedic alcoholic preparation conditionally indicated in urinary and metabolic disorders, reflecting honey's established hypoglycaemic properties when unfermented — an effect that may be partially preserved at low fermentation levels.[24]

4.2.8 Ikṣurasa-āsava (Sugarcane Juice Wine)

Ikṣurasa-āsava is fermented sugarcane juice. It is described as *Balya* (strength-promoting), *Pitta-hara* (reduces Pitta), *Varṇya* (complexion-improving), and *Hṛdya* (cardioprotective). Its modern analogue is fermented sugarcane juice or low-alcohol cane wine. Among sweetened fermented beverages, this preparation has relatively higher natural polyphenol content, which may partially account for its cardioprotective designation.[25]

4.2.9 Maireyaka (Multi-ingredient Fermented Beverage)

Maireyaka is a complex fermented preparation from multiple substrates. It is *Tīkṣṇa*, *Kaṣāya* (astringent), *Madakṛt* (intoxicating), *Kṛmighna* (anti-parasitic), *Guru* (heavy in quality), and *Kaphagulmaghna* (disperses abdominal masses). The *Guru* property and high caloric density make it unsuitable in obesity and metabolic disorders.[26]

4.2.10 Madhūkapu-ṣpa Madya (Mahua Flower Wine)

Madhūkapuṣpa Madya is wine prepared from flowers of *Madhūka* (*Madhuca longifolia*, Mahua). It is *Rūkṣa* (dry), *Kaṣāya* (astringent), *Kaphagna* (anti-Kapha), but simultaneously *Vāta-Pitta-prakopana* (aggravates Vāta and Pitta). Mahua wine is an important traditional beverage among tribal communities of central India (Madhya Pradesh, Chhattisgarh, Odisha, and Jharkhand) and has been documented in CCRAS ethnopharmacological surveys.[27] The dual aggravation of Vāta and Pitta underscores the classical Ayurvedic caution against indiscriminate use.[28]

4.2.11 Nava Madya (New/Freshly Prepared Alcoholic Beverage)

Nava Madya (newly prepared beverage) is described as *Abhiṣyandi* (producing channel congestion), *Guru* (heavy), *Vāta-ādi-kopana* (aggravates all three Doṣas), and *Vidāhī* (acidic/burning in the gut). This characterisation corresponds strikingly with the modern nutritional observation that freshly fermented (unaged) beverages contain higher concentrations of fusel alcohols, congeners, and residual sugars — all of which increase caloric density, glycaemic response, and gastrointestinal irritation.[29] [30]

4.2.12 Purāṇa Madya (Old/Aged Alcoholic Beverage)

Sugandhī dīpanaṃ hṛdyaṃ kṛmanāśanaṃ | sphūṭa srotaskaraṃ jīrṇaṃ laghu vātakaphāpaham || (Su. Sū. 46/193)

Purāṇa Madya (aged beverages) is described as *Sugandhī* (aromatic), *Dīpana* (digestive stimulant), *Hṛdya* (cardioprotective), *Laghu* (lighter than fresh preparations), and *Vāta-Kapha-apaha* (reduces Vāta and Kapha). Aged alcoholic preparations are thus preferred over freshly fermented ones — a clinical nuance validated by modern oenology and spirits science, which demonstrates that aging reduces fusel oil and congener content, lowers residual sugar levels, and increases beneficial polyphenol concentration, particularly in wine.[31][32]19

4.3 Ayurvedic Dietary Position on Madya in Madhumeha and Sthaulya

The *Sushruta Samhitā* is explicit and categorical in its prohibition of alcoholic beverages in *Madhumeha*. The rationale embedded in the classical texts is multidimensional: (i) Most alcoholic preparations are *Kaphakāraka* (phlegm-augmenting and obesogenic); (ii) they are *Abhiṣyandi* (produce systemic channel congestion — correlating with the modern understanding of alcohol-induced endothelial dysfunction and portal hypertension); (iii) they are *Bṛṃhaṇa* (anabolic) and thus increase adipose tissue; and (iv) fermented preparations derived from sugar-rich substrates directly augment *Meda Dhātu* (adipose tissue) and *Kleda* (pathological moisture/humour excess) — the two primary substrate conditions for *Madhumeha* and *Sthaulya*.[33] The one conditional exception is red wine (*Mārdvīka Madya*), which is described as non-*Vidāhī* (non-acidic), *Laghu* (light), and permissible in individuals who are already habituated to alcohol and cannot abstain (*Arkaprakāśa, Rājavarūṇī* formulation). The *Dhanvantari Nighaṇṭu* echoes this by categorising grape-based preparations among the least metabolically harmful of alcoholic beverages.[34]

5. PATHOGENESIS OF ETHANOL-INDUCED HEPATIC INJURY: MODERN AND AYURVEDIC PERSPECTIVES

5.1 Overview of Alcoholic Liver Disease (ALD)

Alcoholic liver disease (ALD) represents a spectrum of hepatic pathology ranging from reversible steatosis (fatty liver) to alcoholic hepatitis, fibrosis, and irreversible cirrhosis. The threshold cumulative ethanol dose for serious liver disease has been estimated at 600 kg for men and 150–300 kg for women. This corresponds to consuming approximately 8–12 oz of beer (or 1 litre of wine, or 0.5 pint of distilled spirits) daily for 20 years in men, or approximately 10 years in women — a significantly lower threshold that reflects sex-specific differences in hepatic alcohol dehydrogenase (ADH) activity. Notably, serious liver disease develops in fewer than 50% of individuals consuming the estimated threshold dose, implicating genetic, nutritional, and environmental cofactors. [35] [36] [37]

5.2 Enzymatic Pathways of Hepatic Ethanol Metabolism

Within the liver, ethanol is metabolised via three enzymatic systems: (i) alcohol dehydrogenase (ADH), the primary pathway at physiological blood ethanol concentrations; (ii) the microsomal ethanol-oxidising system (MEOS), predominantly the cytochrome P-450 isoenzyme CYP2E1, which is induced by chronic ethanol exposure and predominates when blood ethanol exceeds 10 mmol/L (approximately 50 mg/dL); and (iii) catalase, which contributes minimally.Both ADH and CYP2E1 convert ethanol to acetaldehyde, which is then oxidised to acetate in hepatocyte mitochondria by aldehyde dehydrogenase (ALDH).[21] CYP2E1-mediated oxidation generates reactive oxygen intermediates (ROIs) including the hydroxyethyl radical, superoxide anion (O2−), and hydroxyl radical (OH•), which inflict oxidative damage on intracellular lipids, proteins, and DNA. [38] An important ethnogenetic dimension is the polymorphism in ADH and ALDH isoenzymes across populations. Approximately 50% of East Asian individuals (Japanese and Chinese) carry the *ALDH2*2* allele encoding a catalytically inactive ALDH2 enzyme; this leads to acetaldehyde accumulation, manifesting as facial flushing, tachycardia, and circulatory instability.[39] In the Indian subcontinent, specific *ADH1B* and *CYP2E1* polymorphisms have been documented in patients with ALD, with the *CYP2E1 c2* allele showing higher inducibility and greater susceptibility to oxidant stress-mediated liver injury.[40]

5.3 NADH Redox Imbalance and Hepatic Steatosis

ADH-mediated ethanol oxidation generates an excess of reduced nicotinamide adenine dinucleotide (NADH), shifting the hepatic redox state (NADH:NAD+ ratio).

Excess NADH provokes hepatic steatosis through two complementary mechanisms: (i) stimulation of de novo fatty acid synthesis (lipogenesis) and (ii) inhibition of fatty acid β-oxidation in mitochondria. The resulting triglycerides accumulate as cytoplasmic lipid droplets, progressing from microvesicular to macrovesicular steatosis with continued ethanol exposure. NADH excess also impairs gluconeogenesis (contributing to fasting hypoglycaemia) and alters the metabolism of lactate, pyruvate, and the citric acid cycle intermediates.[34]

5.4 Oxidant Stress and Mitochondrial Dysfunction

Chronic ethanol consumption depletes hepatic antioxidant reserves — specifically vitamin A (retinol), vitamin E (α-tocopherol), and glutathione — through multiple mechanisms. Ethanol-induced vitamin E deficiency accelerates hepatic lipid peroxidation; vitamin A depletion causes lysosomal membrane damage; and selective depletion of mitochondrial glutathione impairs mitochondrial respiratory function.[41] Mitochondrial dysfunction is a central feature of ALD. CYP2E1-derived free radicals induce mitochondrial DNA deletions and point mutations that impair oxidative phosphorylation and the tricarboxylic acid (TCA) cycle by up to 40%. Morphologically, this manifests as megamitochondria (giant mitochondria) detectable in approximately 25% of ALD patients and microvesicular steatosis — both early markers of ethanol-induced liver injury.

5.5 Acetaldehyde-Mediated Hepatocellular Injury

Acetaldehyde, the primary toxic metabolite of ethanol, exerts hepatocellular injury through several mechanisms: (i) formation of acetaldehyde-protein adducts that serve as neoantigens, triggering autoimmune responses — antibodies against such neoantigens are detectable in the circulation of patients with ALD; (ii) impairment of mitochondrial β-oxidation of fatty acids, exacerbating steatosis; (iii) stimulation of hepatic collagen synthesis by hepatic stellate cells (HSCs), driving fibrogenesis; and (iv) inhibition of protein secretion, the primary mechanism underlying hepatocellular ballooning (swelling).The pathogenic role of autoantibodies in ALD has been confirmed in animal models: guinea pigs previously immunised with acetaldehyde-protein adducts and subsequently fed ethanol developed signs of hepatic injury within 40 days, with fibrosis evident at 90 days — demonstrating a clear causal link between neoantigen formation, immune activation, and fibrotic progression.[42]

5.6 Histopathological Spectrum of Alcoholic Liver Disease

5.6.1 Hepatic Steatosis (Fatty Liver)

Fatty liver is the earliest and most reversible stage of ALD. Even moderate acute ethanol intake produces microvesicular lipid droplets in hepatocytes. With chronic excess intake, macrovesicular steatosis develops — large lipid globules that displace the nucleus to the hepatocellular periphery. Macroscopically, the steatotic liver is enlarged (up to 6 kg), soft, and yellow. At this stage, the condition is entirely reversible with abstinence. Pericentral fibrosis around the central vein may develop with continued intake, marking progression to early fibrosis.[34][35]

5.6.2 Alcoholic Hepatitis

Alcoholic hepatitis is characterised by hepatocyte swelling (ballooning), necrosis, and the pathognomonic *Mallory bodies* — eosinophilic cytoplasmic inclusions composed of misfolded keratin intermediate filaments. A neutrophilic inflammatory infiltrate accumulates around ballooned hepatocytes. Sinusoidal and perivenular fibrosis is invariably present. Macroscopically, the liver is mottled red with bile-stained areas and visible nodules indicating early cirrhotic transformation. In India, a study by Amarapurkar et al. found that alcoholic hepatitis constituted approximately 20% of chronic liver disease admissions in a tertiary Mumbai hospital, with a 30-day mortality exceeding 35% in severe cases.[43] [44]

5.6.3 Alcoholic Cirrhosis (Laennec's Cirrhosis)

Alcoholic cirrhosis represents the final, irreversible stage of ALD. The cirrhotic liver undergoes progressive fibrotic transformation from a yellow-tan, fatty, enlarged organ (>2 kg) to a brown, shrunken, non-fatty organ (<1 kg) over years. Regenerative nodules (initially micronodular, later mixed micro/macronodular) become encircled by broad fibrous septa ('hobnail' surface on gross examination). End-stage cirrhosis is histologically indistinguishable from viral hepatitis-related cirrhosis. Portal hypertension, oesophageal varices, ascites, hepatic encephalopathy, and hepatorenal syndrome are major sequelae. [43] The Global Burden of Disease 2019 study attributed 25.2% of all cirrhosis deaths globally to alcohol.

5.7 Cofactors in Alcoholic Liver Disease: Gender, Nutrition, and Genetics

Women develop ALD at substantially lower cumulative ethanol doses than men, a disparity attributed to lower gastric ADH activity in women (resulting in greater first-pass ethanol delivery to the liver), higher body fat percentage (altering ethanol distribution volume), and hormonal modulation of hepatic CYP2E1 expression. A landmark study by Tuyns and Péquignot established that women require approximately 40% less cumulative ethanol to develop cirrhosis compared to men — data subsequently confirmed in Indian cohorts by Duseja et al. [45] Nutritional cofactors play a critical modulatory role. Polyunsaturated fat (PUFA) intake amplifies ethanol-induced oxidant stress by upregulating CYP2E1 and providing additional substrates for lipid peroxidation. Conversely, antioxidant-rich diets (high in vitamins C and E, selenium, and polyphenols) attenuate hepatocellular oxidant damage. The ICMR-NIN dietary guidelines for India identify alcohol as an independent risk factor for liver disease and recommend complete abstinence in all individuals with established metabolic disorders.

6. OBSERVATIONS

6.1 Nutritional Profile and Ayurvedic Analogues of Common Alcoholic Beverages

Table 1 presents the caloric, carbohydrate, and alcohol content (per 100 mL) of common alcoholic beverages, alongside their Ayurvedic analogues and classical pharmacological properties.

Beverage	Calories (kcal/100 mL)	Carbohydrate (g/100 mL)	Alcohol % v/v	Ayurvedic Analogue	Classical Properties
Red Wine	68	2.5	9.6	*Mārdvīka Madya*	*Laghu, Madhura, non-Vidāhī, Hṛdya, conditionally permitted*
Whisky	207	0.0	29.6	*Tushodaka / Surā*	*Kaphakāraka, Tīkṣṇa, Uṣṇa, Balya*
Vodka	207	0.0	29.6	*Śārkarā Madya*	*Kaphakāraka, Tīkṣṇa, Uṣṇa*
Gin	207	0.0	29.6	*Surā variant*	*Kaphakāraka, Tīkṣṇa*
Brandy	207	0.0	29.6	*Kharjūra / Phalāsava*	*Vātakopana, Kaphagna, Karśana*
Port Wine	157	12.0	15.9	*Mārdvīka Madya*	*Madhura, Guru, Abhiṣyandi*
Rum	207	0.0	29.6	*Ikṣurasa-āsava / Kohal*	*Kaphakāraka, Guru, Bṛṃhaṇa*
Beer	43	3.5	4.5	*Surā (Nava)*	*Abhiṣyandi, Guru, Kapha-kara*
Mahua Wine	90	5.0	11.0	*Madhūkapuṣpa Madya*	*Rūkṣa, Kaṣāya, Kaphagna, Vāta-Pitta-kara*
Mead (Honey Wine)	75	7.5	8.0	*Madhvāsava*	*Laghu, Meha-apaha, Chedī*

Table 1: Nutritional composition, Ayurvedic analogues, and classical properties of common alcoholic beverages (per 100 mL). Sources: NIN Hyderabad[12]; USDA FoodData Central[13]; Atkinson et al.[14]; Sushruta Samhitā[8]; Ashtānga Hridayam[11]; Dhanvantari Nighaṇṭu.[20]

6.2 Key Analytical Observations

(i) Caloric density of distilled spirits: Distilled spirits (whisky, vodka, gin, rum, brandy) uniformly carry the highest caloric burden (≈207 kcal/100 mL) despite zero carbohydrate content, owing entirely to their alcohol content (≈29.6% v/v; alcohol = 7 kcal/g). These correspond to the Ayurvedic *Surā*, *Śārkarā*, and *Kharjūra* preparations characterised as *Guru, Bṛṃhaṇa*, and *Kaphakāraka* — directly validating their contraindication in obesity and diabetes.

(ii) Red wine — the closest permissible analogue: Red wine carries the lowest caloric and carbohydrate load (68 kcal/100 mL; 2.5 g carbohydrate/100 mL) among common alcoholic beverages, consistent with its designation as *Mārdvīka Madya* — the only preparation conditionally permitted in Ayurvedic texts. The earlier mentioned PREDIMED trial and the Nurses' Health Study both identified moderate red wine consumption as carrying lower cardiovascular risk than equivalent alcohol intake from spirits or beer, attributable to the polyphenol content (particularly resveratrol and quercetin) of the grape skin fermentation — the precise preparation method described for *Mārdvīka Madya* (fermentation with skin intact, *sakala-drākṣā*).

(iii) *Nava* (New) vs. *Purāṇa* (Old) Madya — the fermentation age principle: The classical distinction between *Nava Madya* (new, *Abhiṣyandi, Guru*) and *Purāṇa Madya* (aged, *Laghu, Hṛdya*) is validated by modern oenological and spirits science data: freshly fermented beverages contain higher concentrations of residual sugars, fusel alcohols, and acetaldehydes (glycaemic and hepatotoxic respectively), while aging reduces these compounds and concentrates beneficial polyphenols.

(iv) Beer — high glycaemic impact despite low alcohol content: Beer carries a relatively modest caloric load (43 kcal/100 mL) but contributes 3.5 g/100 mL of bioavailable carbohydrate with a moderately high glycaemic index (GI ≈ 89–110), explaining its designation as *Abhiṣyandi* and *Guru* in the classical text — properties that directly correlate with its tendency to impair glycaemic control in T2DM.

(v) Mahua wine — a culturally specific Indian beverage: Mahua wine, the traditional beverage of tribal communities across central India, carries a moderate caloric burden (90 kcal/100 mL) with 5 g/100 mL carbohydrate and approximately 11% alcohol content. Its classical characterisation as *Rūkṣa* (dry) and *Kaphagna* but *Vāta-Pitta-kara* (simultaneously aggravating Vāta and Pitta) suggests significant metabolic perturbation — consistent with published reports of higher rates of gastric ulceration, liver disease, and neurological complications in mahua-consuming tribal populations.

7. DISCUSSION

7.1 Convergence of Ayurvedic Classification and Modern Nutritional Epidemiology

The *Sushrutokta Madya Varga*'s classification of alcoholic beverages exhibits substantial convergence with contemporary nutritional epidemiology. The GBD 2019 Collaborators[1] documented that no level of alcohol consumption is without associated health risk — echoing the Ayurvedic categorical prohibition of alcoholic beverages in *Madhumeha* and *Sthaulya*. A landmark 2022 JAMA Network Open meta-analysis[4] pooling data from 30 prospective cohort studies (n = 1,107,806) confirmed that alcohol consumption — even at moderate levels — significantly increased risk of T2DM compared to lifetime abstinence, with a dose-response relationship that mirrors the *Kaphakāraka* dosage principle in Ayurveda.

7.2 Alcohol, Insulin Resistance, and the Ayurvedic Kaphakāraka Principle

The Ayurvedic concept of *Kaphakāraka* alcoholic preparations corresponds mechanistically with the modern understanding of alcohol-induced insulin resistance. Ethanol increases hepatic de novo lipogenesis (DNL) via the NADH redox shift and SREBP-1c activation, producing intrahepatic triglyceride accumulation (steatosis). Hepatic steatosis impairs insulin signalling through diacylglycerol-mediated protein kinase C-ε activation, inhibiting the insulin receptor substrate-1 (IRS-1) pathway and reducing hepatic glucose uptake. In the Ayurvedic framework, this corresponds precisely to the *Medodushti* (adipose tissue derangement) and *Dhatvagnimāndya* (reduced tissue metabolic fire) mechanisms underlying *Madhumeha*.

7.3 Red Wine and the Resveratrol Paradox: Reconciling Ayurvedic Permission with Modern Evidence

The conditional Ayurvedic permission of *Mārdvīka Madya* (grape wine) in habituated individuals corresponds with the much-debated 'Mediterranean paradox' and the specific cardio-metabolic properties of resveratrol and other grape polyphenols. Resveratrol activates SIRT1 (sirtuin-1), a NAD+-dependent deacetylase that improves mitochondrial biogenesis, reduces hepatic lipid accumulation, and enhances insulin sensitivity. The PREDIMED trial[32] demonstrated that moderate red wine consumption (150–200 mL/day) within the Mediterranean diet context significantly reduced cardiovascular events and T2DM incidence. However, it is critical to note that the Ayurvedic permission is explicitly conditional (*yuktyā*), applies only to individuals already habituated to alcohol (not as a recommendation for new consumption), and specifies a lean, fresh grape wine preparation — corresponding to wines with the highest polyphenol content.

7.4 Madhvāsava and the Meha-apaha Designation: Scientific Plausibility

The designation of *Madhvāsava* (honey mead) as *Meha-apaha* (therapeutic in urinary/metabolic disorders including diabetes) warrants careful examination. While raw honey contains biologically active compounds including methylglyoxal, polyphenols, and chromium — all of which have demonstrated hypoglycaemic effects in experimental and clinical studies[38] — the fermentation of honey to produce mead converts these fructose-containing substrates to ethanol, substantially altering the pharmacological profile. A systematic review by Bogdanov et al.[38] concluded that the antidiabetic properties of honey are primarily attributable to its unfermented form; the therapeutic value of mead in diabetes therefore requires independent clinical validation before it can be recommended. The Ayurvedic designation likely reflects the residual bioactivity of incompletely fermented honey preparations with low alcohol content.

7.5 Mahua Wine in the Indian Tribal Health Context

Mahua wine (*Madhūkapuṣpa Madya*) occupies a unique position in the Indian cultural and public health landscape. Prepared from the flowers of *Madhuca longifolia* by tribal communities across Madhya Pradesh, Chhattisgarh, Jharkhand, and Odisha, it constitutes an ethnopharmacologically documented beverage with deep socioeconomic significance.[17] A CCRAS field survey (2019) documented the widespread consumption of mahua wine in Bastar district, Chhattisgarh, and noted associations with hepatic morbidity, peripheral neuropathy, and nutritional deficiency syndromes. The classical Ayurvedic dual warning of *Vāta-Pitta-kara* (aggravating both Vāta and Pitta) for *Madhūkapuṣpa Madya* — which in clinical Ayurvedic terms implies simultaneous neurotoxic and hepatotoxic potential — thus demonstrates remarkable prescience given the modern morbidity data from mahua-consuming populations.

7.6 Gender Differences in Alcohol Metabolism: Classical and Modern Perspectives

The modern evidence that women develop serious ALD at substantially lower cumulative alcohol doses than men[23] has a classical Ayurvedic counterpart in the explicit qualification *Strī-viśeṣa* (gender-specific prescriptions) embedded in dietary guidance. The *Ashtānga Hridayam* cautions against alcohol consumption during pregnancy and lactation, consistent with the teratogenic and galactoprotective evidence

reviewed by ICMR-NIN.[15] The lower gastric ADH activity in women — resulting in higher hepatic first-pass ethanol delivery — provides the mechanistic explanation for sex-specific ALD susceptibility documented in Indian cohorts by Duseja et al.

7.7 Alcohol Consumption in India: Epidemiological Burden and Policy Context

India's alcohol consumption profile has undergone significant transformation. The NFHS-5 (2019–21)[6] documented alcohol consumption in 18.7% of men and 1.3% of women aged 15 years and above, with a secular increase from NFHS-3 figures (23.9% and 1.3% respectively) masked by state-level heterogeneity — notably, some northeastern states and Telangana report consumption exceeding 40% among men. A nationally representative survey by the ICMR[7] estimated that alcohol-attributable liver disease, T2DM complications, and cardiovascular disease collectively contributed to 4.1% of all disease burden in India. The WHO Global Status Report on Alcohol and Health (2022) identified India as a priority nation for alcohol policy intervention given its large and growing population of at-risk drinkers. The AYUSH Ministry's National Action Plan on Lifestyle Disorders (2022–23)[9] has begun incorporating Ayurvedic dietary principles — including the *Madya Varga* prohibitions — into public health messaging. The present review provides the evidence base necessary to formally validate these recommendations within an integrative medicine framework.

8. CONCLUSION

The *Madya Varga*, as codified in the *Sushruta Samhitā* and elaborated in the *Ashtānga Hridayam* and allied Nighaṇṭu texts, constitutes a pharmacologically sophisticated, clinically coherent classification of alcoholic beverages whose core dietary prohibitions in *Madhumeha* and *Sthaulya* are strongly supported by contemporary nutritional epidemiology, metabolic biochemistry, and hepatology.

The *Kaphakāraka* designation applied to most alcoholic preparations corresponds mechanistically with the modern understanding of alcohol-induced hepatic lipogenesis, insulin resistance, and visceral adiposity. The preferential classification of *Mārdvīka Madya* (grape wine) as the least metabolically harmful beverage favours red wine's polyphenolic profile over other alcoholic beverages. The distinction between *Nava* (freshly fermented, hepatotoxic, obesogenic) and *Purāṇa* (aged, lighter, hepatoprotective) preparations embeds a remarkable empirical understanding of fermentation kinetics, congener chemistry, and polyphenol development.

The pathomechanisms of ethanol-induced hepatic injury — ADH/CYP2E1 metabolism, acetaldehyde toxicity, NADH redox imbalance, oxidant stress, mitochondrial dysfunction, and neoantigen-mediated autoimmunity — are mirrored in the Ayurvedic concepts of *Abhiṣyandi* (channel congestion), *Ama* production (toxic metabolite accumulation), *Agni-dushti* (metabolic fire impairment), and *Yakṛt-dosha* (hepatic derangement).

Future research should prioritise: (i) prospective clinical trials in Indian cohorts evaluating Ayurvedic alcohol-restriction protocols on glycaemic and hepatic endpoints; (ii) comparative metabolomic studies of classical *Madya* preparations (*Mārdvīka, Madhvāsava, Surāsava*) versus modern analogues; and (iii) pharmacogenomic studies assessing the interaction between Indian population-specific *ADH1B* and *ALDH2* polymorphisms and Ayurvedic body constitution (*Prakriti*) in determining ALD susceptibility. Such research would constitute a meaningful translational bridge between classical Ayurvedic wisdom and twenty-first-century precision medicine.

9. DECLARATIONS

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