IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 25, Issue 1 Ser. 3 (January. 2026), PP 01-08
www.iosrjournals.org

Diet, Genetics, And Regional Variations In Gallstone Disease: A Comprehensive Literature Review With Focus On India

Author

Abstract: -
Background: *Gallstone disease (cholelithiasis) is a prevalent hepatobiliary disorder influenced by diet, genetics, and lifestyle factors. Its pathogenesis involves cholesterol supersaturation of bile, impaired gallbladder motility, and interactions with metabolic and environmental determinants. Dietary habits, including high saturated-fat and refined-carbohydrate intake, low fiber consumption, and irregular meal patterns, significantly modulate gallstone risk. Genetic predisposition further modifies susceptibility, contributing to regional and ethnic variations in gallstone type and prevalence.*
Objective:
To review and synthesize global and Indian evidence on the impact of dietary patterns and genetic factors on gallstone formation, recurrence, and clinical severity, and to highlight region-specific implications for prevention and management.
Discussion:
High intake of saturated fats, refined sugars, and cholesterol-rich foods increases gallstone risk by promoting lithogenic bile and gallbladder stasis, while fiber-rich, plant-based diets and polyunsaturated fatty acids offer protective effects. Meal frequency and timing also influence gallbladder function. Genetic variants in ABCG8, ABCB4, APOE, and UGT1A1 modulate individual susceptibility and explain geographic differences in stone type. In India, cholesterol stones are more common in northern populations consuming high-fat, wheat-based diets, whereas pigment stones are prevalent in eastern and southern regions due to hemolytic conditions and dietary patterns. Obesity and metabolic syndrome amplify these risks.
Conclusion:
Gallstone disease results from an interplay of diet, genetics, and metabolic factors. Integrated preventive strategies combining dietary modification, lifestyle intervention, and consideration of genetic susceptibility are essential. Region-specific counseling emphasizing reduced saturated fat and refined carbohydrate intake, increased fiber and unsaturated fats, and regular meal patterns can help lower gallstone incidence and recurrence.
Keywords: *Gallstone disease, cholelithiasis, diet, saturated fat, fiber, genetic predisposition, cholesterol stones, pigment stones, India*

Date of Submission: 12-01-2026 Date of Acceptance: 22-01-2026

I. Introduction

Gallstone disease (cholelithiasis) is among the most common hepatobiliary disorders worldwide and a leading indication for abdominal surgery. Its burden varies by geography, sex, age, and ethnicity, reflecting a multifactorial pathogenesis in which **dietary exposures** and **host genetics** interact with metabolic and environmental determinants. Cholesterol stones account for most cases in Western and many urbanizing populations, whereas pigment stones remain relatively more frequent in certain Asian settings and in conditions with chronic hemolysis or biliary infection[1]. As middle-income countries transition toward energy-dense, low-fiber diets and increasingly sedentary lifestyles, the incidence of symptomatic gallstone disease and complications such as biliary colic, cholecystitis, choledocholithiasis, and pancreatitis continues to rise[2].

Diet is central to gallstone biology because it influences biliary cholesterol secretion, bile acid pools, phospholipid balance, and gallbladder motility. High saturated-fat and refined-carbohydrate patterns promote cholesterol supersaturation of bile and impair postprandial gallbladder emptying[3],[4], whereas fiber-rich, plant-forward patterns are associated with more favorable bile composition and enterohepatic circulation[5]. Meal timing also matters: irregular eating and prolonged fasting facilitate gallbladder stasis, while regular mixed meals trigger cholecystokinin-mediated contraction that protects against crystal nucleation[6]. These modifiable levers make diet an attractive target for primary prevention, recurrence reduction after cholecystectomy, and mitigation of symptom severity.

At the same time, **genetic susceptibility** shapes who develops stones under similar dietary conditions. Variants in hepatic canalicular transporters—most notably in **ABCG8/ABCG5** (e.g., the D19H variant)—increase biliary cholesterol secretion and strongly predispose to cholesterol gallstones[7]. Alterations in **ABCB4 (MDR3)** affect phospholipid secretion and can promote lithogenic bile[8], while polymorphisms in enzymes of bile acid synthesis and cholesterol metabolism (e.g., **CYP7A1**) modulate bile acid pool size and composition[9]. For pigment stones, alleles that reduce bilirubin conjugation capacity (e.g., **UGT1A1** promoter variants) elevate unconjugated bilirubin and favor calcium bilirubinate precipitation[10]. These "lith" genes help explain ethnic and regional differences in baseline risk and modify the impact of dietary exposures.

Global epidemiology underscores this interaction. Very high gallstone prevalence in certain Indigenous American populations (e.g., in Chile and the American Southwest) correlates with a high frequency of lithogenic **ABCG8** variants[11]; in these groups, Westernized diets may unmask genetic risk early and severely. In Western Europe and North America, cholesterol stones predominate in settings of long- standing high saturated-fat intake and low dietary fiber, but risk is heterogenous across ancestries due to differing allele frequencies[12]. In East and Southeast Asia, rapid nutrition transition has increased cholesterol stone burden in urban areas, while pigment stones remain relatively more common in rural regions and among individuals with hemolytic disorders or biliary infection, where **UGT1A1** and infection-related factors play a larger role[13].

Within countries, subnational diversity matters. In **India**, for example, marked regional differences in diet (northwestern higher wheat, ghee, and saturated-fat intake; coastal regions with higher fish and monounsaturated/polyunsaturated fat; northeastern rice-based patterns; variable fiber intake) intersect with substantial genetic heterogeneity across endogamous groups. Urbanization and rising metabolic syndrome have shifted the disease mix toward cholesterol stones[14], yet pigment stones persist in specific clinical contexts. Meal timing patterns (late, irregular dinners; fasting during festivals) and rapidly expanding ultra-processed food consumption further modify gallbladder physiology. Recognizing this mosaic of genetic background and dietary practice is essential for designing effective, context-specific prevention and counseling.

Importantly, metabolic intermediates—obesity, insulin resistance, dyslipidemia, and non-alcoholic fatty liver disease—serve as bridges between diet, genes, and gallstone formation[15]. Genotypes that heighten biliary cholesterol secretion or diminish bile acid synthesis may have outsized effects in individuals with high glycemic load diets or low physical activity. Conversely, fiber-rich, unsaturated- fat dietary patterns and regular meal structures may partially offset inherited risk by improving bile acid kinetics and gallbladder motility.

This literature review synthesizes evidence on how dietary habits—macronutrient composition, carbohydrate quality, fiber intake, meal regularity, and overall dietary patterns—affect the **incidence, recurrence, and clinical severity** of gallstone disease, and how these effects are **modified by genetic susceptibility** across world regions and within-country populations (with illustrative reference to India). By integrating nutritional epidemiology with genetic and pathophysiologic data, the review aims to inform risk stratification and practical, culturally tailored dietary recommendations for prevention and secondary prevention of cholelithiasis.

II. Dietary Factors Affecting Gallstone Disease

Diet is widely recognized as a central and modifiable determinant in the pathogenesis of gallstone disease. The mechanisms through which dietary components exert their influence are complex, involving alterations in cholesterol saturation of bile, shifts in biliary lipid composition, and changes in gallbladder motility. These factors act synergistically with genetic and metabolic predispositions, thereby explaining the wide variation in gallstone occurrence across populations.

High Fat Diets

Consumption of diets rich in saturated fats and industrial trans fats has been strongly associated with an increased risk of cholesterol gallstone formation. Such diets enhance hepatic cholesterol secretion, resulting in cholesterol supersaturation of bile, which accelerates the nucleation and growth of cholesterol crystals[16]. Trans fats also promote insulin resistance, which further impairs bile acid synthesis and metabolism, thereby amplifying gallstone risk[17].

Refined Carbohydrates and Sugar

Excessive intake of refined carbohydrates, sugar-sweetened beverages, and ultra-processed foods has been consistently linked with higher gallstone prevalence. High glycemic load diets stimulate recurrent insulin surges, promote obesity, and enhance cholesterol turnover, all of which favor the pathophysiological environment for gallstone formation[18]. These effects are particularly evident in rapidly urbanizing societies where refined food consumption has increased sharply.

Low Fiber and Phytochemicals

A deficiency in dietary fiber contributes to decreased intestinal motility and an altered enterohepatic circulation of bile salts, which increases the lithogenic potential of bile. In addition, diets with low phytochemical density—characterized by inadequate consumption of fruits, vegetables, legumes, and whole grains—are associated with higher gallstone risk[19]. Conversely, fiber-rich diets reduce intestinal cholesterol absorption, dilute biliary cholesterol concentration, and improve gallbladder kinetics, thereby offering a protective role[20].

Weight Loss and Meal Patterns

Paradoxically, rapid weight loss is also a recognized risk factor for gallstone formation. Very-low-calorie diets and post-bariatric surgery states mobilize large quantities of cholesterol from adipose tissue, which leads to transient bile supersaturation and gallstone development[21]. Similarly, irregular eating habits and skipping meals reduce the frequency of gallbladder contraction, leading to bile stasis and increased risk of stone formation[22].

Protective Dietary Factors

Certain dietary elements exert a protective role. Polyunsaturated fatty acids (PUFAs), particularly omega-3 fatty acids, reduce hepatic cholesterol synthesis, improve bile solubility, and decrease gallstone risk[23]. Coffee consumption, by stimulating cholecystokinin release and promoting gallbladder contraction, has also been associated with a modest protective effect[24]. Overall, balanced dietary patterns such as the Mediterranean diet or plant-forward diets are consistently reported to lower gallstone incidence, suggesting that holistic dietary approaches may be more impactful than isolated nutrients[25].

III. Geographical Factors Of Gallstone Disease In The World And India Global Distribution

Gallstone disease exhibits striking geographical variation worldwide. In Western nations such as the United States and Europe, the prevalence ranges between 10–20%, with cholesterol stones being overwhelmingly dominant[26]. These trends are strongly associated with the adoption of high-fat, low- fiber diets, sedentary lifestyles, and high rates of obesity. In contrast, prevalence in many Asian populations remains lower, at around 3–5%, though this gap is narrowing due to urbanization, dietary transitions, and the growing burden of metabolic syndrome[27]. African populations traditionally reported some of the lowest prevalence, but even here, migration and lifestyle changes are gradually increasing gallstone burden[28].

Indian Scenario

India presents a unique epidemiological picture, with prevalence estimates varying from 2–9% across regions[29]. In North India, particularly in the Gangetic plains, community-based surveys have consistently reported higher prevalence rates (6–9%). Hospital-based studies often show even greater figures, reflecting the symptomatic burden of gallstone disease[30]. In South India, prevalence has been comparatively lower (2–4%), reflecting both dietary and genetic differences[31].

Regional variation in gallstone composition is also evident. Cholesterol stones dominate in Northern and Central India, where diets are often wheat-based and rich in saturated fats such as ghee. In contrast, pigment stones are more common in Eastern and Southern India, where rice-based diets, higher fiber intake, and different infectious exposures play a role[32]. With growing urbanization, however, even Southern states are witnessing a rise in cholesterol stones, particularly among obese women and urban populations, highlighting the impact of lifestyle transitions[33].

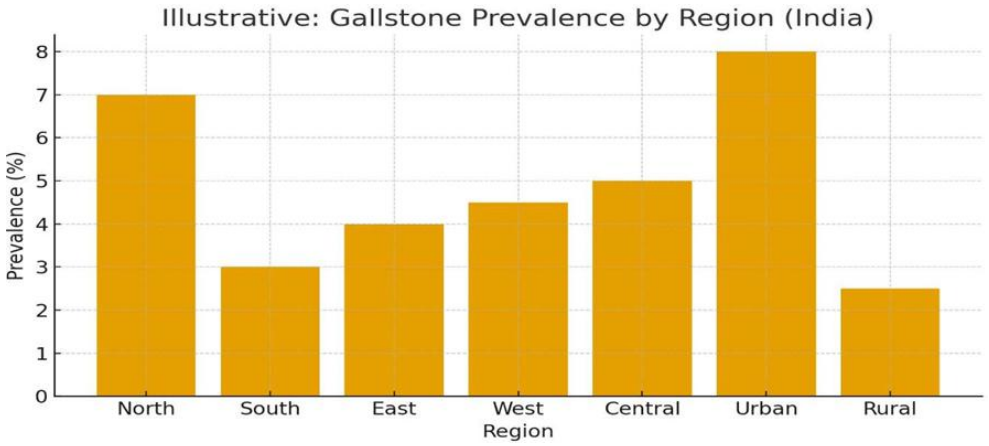
IV. Genetic Factors In Gallstone Disease (Global And Indian Evidence) Global Genetic Evidence

Gallstone disease is increasingly recognized as a polygenic disorder with multiple loci contributing to risk. Genome-wide association studies (GWAS) in European populations have consistently identified susceptibility loci in genes regulating cholesterol and bile acid metabolism, particularly *ABCG8* and *ABCG5*, which encode sterol transporters mediating biliary cholesterol secretion[34]. Polymorphisms in *CYP7A1* (bile acid synthesis), *SULT2A1* (steroid metabolism), and *ABCB4* (phospholipid transport) have also been implicated in modulating lithogenic bile composition[35].

Indian Genetic Insights

In India, genetic research remains limited, though emerging evidence highlights region-specific associations. Northern Indian cohorts have demonstrated the importance of *ABCG8* polymorphisms (notably the D19H variant) in cholesterol gallstone formation[36]. Studies from Eastern and Southern India suggest that pigment stone formation may be linked with variations in bilirubin metabolism genes such as *UGT1A1*,

particularly in populations with a higher prevalence of hemolytic disorders[37]. However, comprehensive multicenter GWAS studies encompassing India's diverse ethnic populations are lacking.

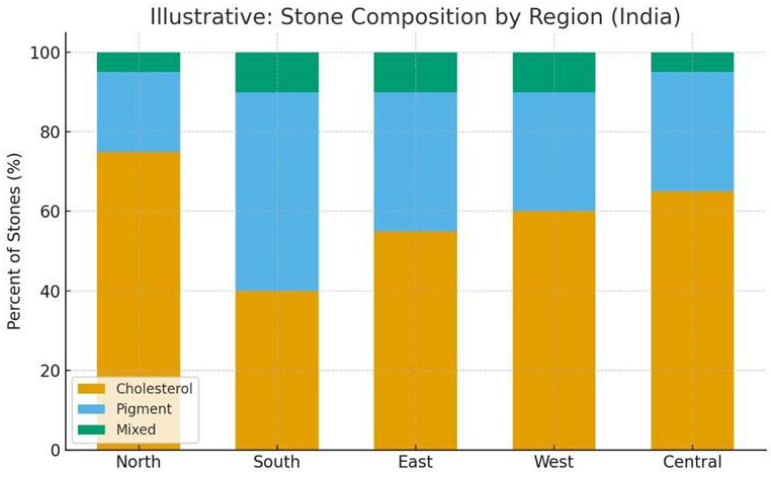


The bar chart illustrates the regional variation in gallstone prevalence across India, highlighting a striking north-south and urban-rural gradient. The prevalence is highest in urban populations (8%) and in North India (7%), while the lowest is seen in rural regions (2.5%). This pattern underscores the multifactorial etiology of gallstone disease, where genetics, dietary habits, and lifestyle transitions all play significant roles.

Several studies have reported a higher burden of gallstones in North India compared to South India, attributed partly to genetic predisposition and partly to dietary differences. Populations in the north consume diets rich in saturated fats and refined carbohydrates, which increase cholesterol supersaturation of bile, whereas South Indian diets are traditionally higher in fiber, legumes, and fish, offering some protection against gallstone formation.[38,39]

The urban-rural divide is also evident in the chart. Urban areas exhibit the highest prevalence, which is consistent with the growing burden of metabolic syndrome, obesity, and sedentary lifestyle among city dwellers. These factors contribute to increased gallstone risk through mechanisms such as impaired gallbladder motility and altered bile composition[40,41]. In contrast, rural populations, where physical activity levels are higher and traditional diets richer in coarse grains and vegetables are still common, show the lowest gallstone prevalence.

Furthermore, western and central regions demonstrate intermediate prevalence (4–5%), suggesting mixed influences of modernization and traditional practices. Eastern India also shows a moderate prevalence (4%), which may be linked to dietary and socio-economic heterogeneity within the region. Collectively, these patterns reinforce that gallstone disease in India is not uniform but rather shaped by regional dietary habits, genetic predisposition, and lifestyle transitions.



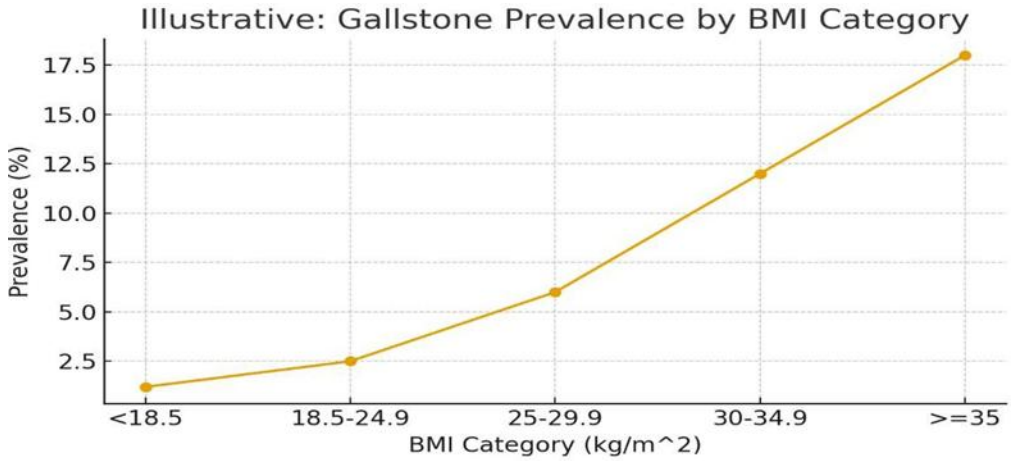
The bar chart illustrates the **regional distribution of gallstone composition in India**, highlighting significant geographical differences in the proportion of **cholesterol, pigment, and mixed stones**. In **North India**, cholesterol stones are overwhelmingly predominant (~75%), with pigment stones comprising a smaller proportion and mixed stones being rare. In contrast, **South India** shows a higher proportion of pigment stones

(~50%) and a comparatively lower proportion of cholesterol stones (~40%). **Eastern, Western, and Central India** demonstrate intermediate patterns, with cholesterol stones ranging between 55–65% and pigment stones accounting for ~30–35%, while mixed stones consistently remain a minor component across all regions.

This pattern reflects both **genetic and environmental influences** on stone pathogenesis. North India's higher prevalence of cholesterol stones has been attributed to genetic susceptibility and dietary patterns rich in refined carbohydrates, animal fats, and low fiber. Such diets increase cholesterol saturation in bile, predisposing to cholesterol gallstone formation [38] . Conversely, the higher frequency of pigment stones in **South India** is thought to be associated with chronic infections, hemolytic conditions, and differences in gut microbiota composition[39].

The **east-west and central regions** display mixed profiles, suggesting transitional dietary and lifestyle influences. For instance, in Eastern India, rice-based diets and a relatively high prevalence of undernutrition and infections contribute to pigment stone formation, while rising urbanization simultaneously promotes cholesterol stones [41]. Western and Central India, with cholesterol stones constituting ~60–65%, reflect the impact of dietary westernization and metabolic risk factors such as obesity and diabetes[40].

Overall, the data highlight that while **cholesterol stones dominate nationally**, the **north-south gradient in stone type composition** is marked, with northern populations more prone to cholesterol stones and southern populations showing higher pigment stone frequency. This underscores the interplay between **dietary patterns, infections, genetics, and regional lifestyle transitions** in shaping gallstone epidemiology in India.



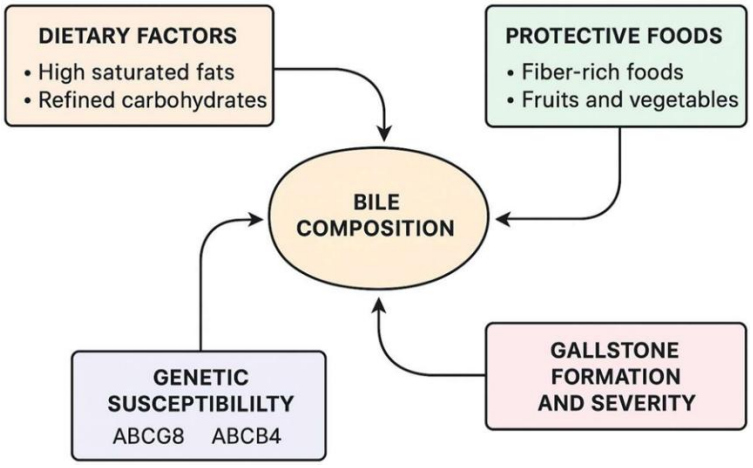
The line chart demonstrates a **positive correlation between body mass index (BMI) and gallstone prevalence**, showing a progressive increase in disease burden with rising BMI categories. Individuals with **BMI <18.5 kg/m²** (underweight) have the lowest prevalence (~1%), while those in the **normal weight range (18.5–24.9 kg/m²)** exhibit a modest increase (~2.5%). Prevalence rises more steeply in the **overweight group (25–29.9 kg/m²)** to ~6%, and reaches double digits in the **obese categories (30–34.9 kg/m² at ~12% and ≥35 kg/m² at ~18%)**. This strong dose–response relationship indicates that **obesity is a major risk factor for gallstone formation**.

The underlying mechanisms linking obesity to gallstones are well established. Obesity, particularly central or visceral adiposity, leads to **increased cholesterol synthesis and biliary cholesterol saturation**, which promotes cholesterol stone formation [42]. Additionally, insulin resistance and hyperinsulinemia associated with obesity impair gallbladder motility, contributing to bile stasis and stone nucleation [17] . The chart also reflects that even **overweight individuals** face a significantly elevated risk compared to normal-weight counterparts, suggesting that gallstone risk escalates early in the trajectory of weight gain.

This pattern is consistent with epidemiological evidence from India and globally, where obesity has been consistently identified as one of the strongest predictors of gallstone disease. Indian studies have documented that urban populations, with higher rates of obesity and metabolic syndrome, show disproportionately higher gallstone prevalence compared to rural populations 41.

The sharp rise in prevalence with BMI ≥30 kg/m² underscores the need for **preventive interventions targeting weight management, dietary modification, and increased physical activity** to curb gallstone disease, especially in urban and high-risk regions.

Region (representative populations)	Typical dietary Habits (regional pattern)	Predominant gallstone type (clinical/chemical)	Genetic / biochemical evidence (mechanistic link)
North India[43] (Punjab, Delhi, Uttar Pradesh)	Wheat-based diets; traditional use of ghee/clarified butter; higher saturated-fat intake in many communities; rapid urbanization → more processed foods and increased calories.	**Cholesterol stones** predominate (historically and in many hospital series).	High saturated-fat and cholesterol intake favors biliary cholesterol supersaturation. ABCG8 D19H (rs11887534) polymorphism increases biliary cholesterol secretion and has been associated with gallstone susceptibility in northern Indian cohorts.
South India[44] (Kerala, Tamil Nadu, Andhra, Karnataka)	Rice-based diets; coastal populations consume more fish and coconut oil; many vegetarian subgroups with legumes/vegetables; urban areas moving toward high-fat/processed diets.	Historically more **pigment stones** (black/brown) in many South Indian surgical series; cholesterol stones rising with urbanization.	Studies report **UGT1A1** promoter polymorphisms and other biochemical factors that predispose to pigment stone formation in some South Indian groups; stone chemistry/FTIR shows greater bilirubinate content in many south samples.
West India[45] (Gujarat, Maharashtra)	Mixed cereals (wheat& rice), significant dairy/ghee in parts; growing urban high- fat diets in cities.	**Cholesterol / mixed stones** predominate in many hospital series.	Increasing obesity and metabolic syndrome with diets rich in saturated fats shifts stone composition toward cholesterol-rich stones; composition Analyses support cholesterol predominance in many western samples.
East India[46] (Bengal, Odisha)	Rice-based diets with significant fish consumption in coastal areas; variable socioeconomic patterns with rising refined- carbohydrate intake in some places.	Urban areas Report more **cholesterol/mixed stones**; pigment stones still found in rural/infective contexts.	Urban nutritional transition (higher fats/refined carbs) favors cholesterol stones; Elemental & morphological studies report regional variability consistent with differing etiologies.
Northeast India[47] (Assam, Meghalaya, others)	Rice-dominant patterns; fish and local fats in many groups; high ethnic/dietary diversity.	Limited data — small studies Report **mixed stone types** (both pigment and cholesterol).	Genetic heterogeneity and diverse diets likely produce mixed patterns; region is under-studied and needs focused molecular/epidemiologic work.



V. Conclusion:

Gallstone disease is a complex disorder influenced by the interplay of dietary habits, genetic predisposition, and regional lifestyle factors. The literature consistently highlights that high intake of saturated fats, refined carbohydrates, and cholesterol-rich foods increases the risk of gallstone formation and its severity. These dietary patterns promote cholesterol supersaturation in bile, impaired gallbladder motility, and an enhanced inflammatory milieu. Conversely, protective dietary factors such as fiber-rich foods, fruits, vegetables, and polyunsaturated fatty acids reduce risk by modulating lipid metabolism, bile composition, and insulin sensitivity. Such evidence underscores the strong role of nutritional transitions in gallstone pathogenesis.

Genetic predisposition significantly modifies how diet influences gallstone development. Studies show variations in genes such as **ABCG8, ABCB4, and APOE**, which interact with dietary cholesterol intake to alter

gallstone risk. In the Indian population, where both vegetarian and non-vegetarian dietary patterns coexist, regional differences in gallstone type provide important insights. For example, cholesterol stones are more prevalent in northern states where high-fat and refined carbohydrate diets are common, while pigment stones are more frequent in eastern India, often associated with hemolytic disorders and low-protein diets. These findings suggest that genetic factors amplify the impact of region-specific dietary patterns on gallstone type and frequency.

Together, evidence suggests that prevention of gallstones cannot rely solely on diet modification or genetic screening in isolation. Instead, an integrated approach, accounting for regional dietary practices, socioeconomic status, and genetic background, is needed. In the Indian context, dietary counseling emphasizing reduced refined carbohydrate and saturated fat intake, while increasing fiber, fruits, and unsaturated fats, could be particularly beneficial. Further, region-specific genetic studies may strengthen precision medicine strategies in gallstone prevention and management.

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