



THE GUT-CENTRIC VIEW OF OBESITY: CONNECTING AYURVEDIC PRINCIPLES WITH MODERN DYSBIOSIS

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ABSTRACT

Obesity is a rapidly growing global health challenge associated with significant metabolic complications. Recent advances in microbiome research have identified gut dysbiosis and impaired intestinal barrier function as important contributors to the development of obesity and related metabolic disorders. *Ayurveda* describes obesity as *Sthoulya*, a disorder that develops due to the excessive consumption of *Santarpāna Ahara* and *Vihara*, resulting in derangement in the metabolism of *Kapha* and *Meda*. Although *Ayurveda* and modern medicine differ in their conceptual frameworks, both recognize the central role of impaired digestion, metabolism, and lifestyle in the pathogenesis of obesity. This shared understanding provides an opportunity to explore obesity from a more integrative perspective, where traditional concepts and modern scientific findings can be viewed together to improve our understanding of disease mechanisms. This review aims to explore the possible relationship between the *Ayurvedic* concept of *Sthoulya* and the emerging role of the gut microbiome. It discusses how classical *Ayurvedic* principles, including vitiation of *Kapha*, *Meda Dushti*, and altered digestive function, may be viewed alongside current evidence on gut microbial imbalance, increased intestinal permeability, chronic low-grade inflammation, excessive accumulation of fat leading to the development of obesity. Rather than establishing a direct equivalence, the review highlights the conceptual similarities between these two perspectives and emphasizes their potential to complement each other. By bringing together these traditional and modern viewpoints, the review seeks to encourage a broader understanding of obesity and support future integrative research that may contribute to more comprehensive prevention and management strategies.

KEYWORDS: Gut microbiome, Obesity, dysbiosis, *Sthoulya*, *Meda*.

INTRODUCTION

Obesity has emerged as one of the most significant global public health challenges, affecting more than one billion people worldwide and markedly increasing the risk of type 2 diabetes, cardiovascular disease, and other chronic metabolic disorders.^[1] Once considered a simple consequence of excessive calorie intake and physical inactivity, obesity is now recognized as a complex, multifactorial disease involving genetic, environmental, dietary, and metabolic factors. In recent years, the gut microbiome has gained considerable attention as a key

regulator of host metabolism, energy homeostasis, immune function, and intestinal barrier integrity. Growing evidence suggests that alterations in the gut microbial ecosystem, known as gut dysbiosis, by modulating energy utilization and storage pathways disrupting short-chain fatty acid (SCFA) production, impairing intestinal barrier function, and promoting metabolic endotoxemia, chronic low-grade inflammation, and insulin resistance. Additionally, a reduced abundance of beneficial bacteria, particularly *Akkermansia muciniphila*, has been

associated with impaired mucus barrier integrity and metabolic dysfunction. These findings highlight the gut microbiome as an important contributor to obesity pathogenesis and a promising target for novel therapeutic strategies.^[2]

Ayurveda describes obesity under the concept of *Sthoulya*, which develops due to excessive indulgence in *Santarpana Ahara* and *Vihara*. These factors lead to *Kapha* and *Meda dushti* which leads to *Vata dosha vimarga gamana*, *Vata dosha* gets *koshtashrita* which increases the *agni bala* leading to *Ama* and *Vikruta meda Utpatti* finally leading to *Sthoulya*.^[3] Classical descriptions of *Bahudrava Shleshma*, *Dosha Dushya Anubandha*, and the interaction between *Prakupita Kapha* and *Meda Dhatu* provides a comprehensive understanding of the pathogenesis of *Sthoulya* from an *Ayurvedic* perspective.^[4]

AIM

- To explore the conceptual relationship between the *Ayurvedic* pathogenesis of *Sthoulya* and the role of gut microbiome dysbiosis in the development of Obesity.

OBJECTIVES

1. To review the current scientific evidence on the role of gut microbiome dysbiosis, intestinal barrier dysfunction and Low grade inflammation in the pathogenesis of Obesity.
2. To correlate the *Ayurvedic* concepts of *Santarpana Ahara-Vihara*, *Kapha* and *Meda Dushti*, *Bahudrava Shleshma*, and *Dosha-Dushya Anubandha* with contemporary mechanisms of gut dysbiosis and intestinal permeability in *Sthoulya*.

Role of *Ama* and *Agni* in relation with Gut microbiome

Agni plays a major role in digestion and metabolism, governing the transformation of food into bioavailable nutrients and ensuring proper nourishment of the tissues. When *Agni* functions optimally, metabolic homeostasis is maintained, whereas *Agnimandya* impairs digestion and gives rise to *Ama*, a pathological state characterized by incomplete digestion and disturbed metabolism.^[5] Recent advances in gut microbiome research provides a biological perspective for understanding this process. A healthy gut microbiota supports digestion by fermenting dietary fibres, producing short-chain fatty acids, regulating bile acid metabolism, maintaining intestinal barrier integrity, and modulating immune responses. However, excess calorie diet, sedentary habits, poor sleep, and chronic stress can disrupt this microbial balance, resulting in gut dysbiosis. Gut dysbiosis in obesity is associated with reduced production of beneficial microbial metabolites, particularly short-chain fatty acids (SCFAs), disruption of intestinal barrier integrity, and increased gut permeability. This allows bacterial components such as lipopolysaccharides (LPS) to enter the circulation, promoting metabolic

endotoxemia, chronic low-grade inflammation, insulin resistance, and impaired metabolic regulation.^[6] Although *Ama* should not be equated with any single microbial metabolite or endotoxin, it may be interpreted as a broader pathological state arising from impaired digestive and metabolic function, which parallels the systemic consequences of Gut dysbiosis. Thus, preservation of *Agni* and prevention of *Ama Utpatti* may be considered essential for maintaining gut microbial balance and preventing metabolic disturbances associated with *Sthoulya*.

Role of Gut microbiome in Obesity

Obesity is a complex chronic condition driven by excessive fat buildup, particularly in visceral areas, which disrupts normal metabolic function and triggers persistent low-grade inflammation throughout the body. This inflammation arises largely from dysfunctional white adipose tissue, especially visceral fat, which releases higher levels of inflammatory signals into the bloodstream. These signals interfere with other organs like the liver and muscles, promoting metabolic derangements such as insulin resistance, elevated glucose levels in the blood stream, and a higher likelihood of conditions like type 2 diabetes and fatty liver disease. A key player in this process is the gut microbiota, the diverse community of microbes living in the digestive tract.^[7]

In people with Obesity, this community often shifts into a state of imbalance, or dysbiosis, marked by reduced overall microbial variety and changes in the proportions of major bacterial groups. One widely discussed alteration involves the Firmicutes and Bacteroidetes phyla, which together dominate the gut ecosystem. Many studies have observed a higher Firmicutes-to-Bacteroidetes ratio in individuals with obesity compared to those at a healthy weight. Firmicutes tend to be more efficient at extracting energy from food, potentially contributing to greater calorie absorption and fat storage, while Bacteroidetes are linked to fibre breakdown and other beneficial processes. This elevated ratio has been proposed as a potential indicator of dysbiosis and risk for Obesity, though findings vary across populations and not all research confirms a consistent pattern.^[8]

Another important bacterium frequently depleted in Obesity is *Akkermansia muciniphila*, a mucin-degrading species from the Verrucomicrobia phylum. This microbe helps maintain the gut's protective mucus layer, supports barrier integrity, reduces inflammation, and produces metabolites that benefit metabolic health. A decrease in this beneficial microorganism may affect intestinal barrier function and contribute to metabolic disturbances commonly observed in obesity. In addition to its effects on the intestine, the gut microbiota influences energy balance through the gut-brain axis, a complex communication pathway involving neural, hormonal, and immune signals that regulate appetite, metabolism, and body weight.^[9]

Gut dysbiosis can affect the gut-brain axis by altering the production of microbial metabolites such as short-chain fatty acids (SCFAs) and increasing intestinal permeability. These changes may promote inflammation and influence neural and hormonal pathways involved in appetite regulation, energy balance, and metabolism. Additionally, microbiota-derived metabolites regulate gut hormones such as glucagon-like peptide-1 (GLP-1), which contribute to the control of hunger, satiety, and glucose homeostasis.^[10]

In obesity, disruption of the intestinal barrier is closely associated with changes in mucus layer composition, changes in mucin production and utilization, microbial imbalance, and reduced integrity of tight junctions between intestinal epithelial cells. The intestinal mucus layer, mainly composed of the mucin protein MUC2, acts as a protective barrier between the gut microbiota and the host tissues. This layer is continuously maintained by goblet cells, which produce newly synthesized (“new”) mucin that forms the inner mucus layer and prevents

direct contact between bacteria and the intestinal epithelium. The outer mucus layer consists of partially degraded (“old”) mucin, which serves as a nutrient source for specific mucin-degrading bacteria, including *Akkermansia muciniphila*.

Under healthy conditions, balanced mucin synthesis and controlled degradation support short-chain fatty acid production especially butyrate which strengthens tight junction proteins (occludin, claudins, ZO-1) and maintains epithelial integrity. In Obesity associated dysbiosis, often characterized by altered Firmicutes to Bacteroidetes ratios and reduced *Akkermansia muciniphila*, mucus thinning and reduced SCFA production weaken tight junctions, increasing intestinal permeability. This “leaky gut” state allows lipopolysaccharide (LPS) translocation into circulation, promoting metabolic endotoxemia, chronic low-grade inflammation, hepatic lipid accumulation (fatty liver), and insulin resistance.

According to Ayurveda

Due to excess indulgence in *Santarpana ahara* and *vihara* (processed food, high amount of packed food(excess caloric food), poor sleep and chronic stress

Achaya poorvaka sanchaya of *kapha* which can be considered as *Vilayana roopa vrudhi*

Increased permeability of the gut

Akkermansia muciniphila does not feed on the old mucin layer

Tight junction will be loosened

Due to *Dosha dushya anubandha* the mucin liquifies

Production of *Bahudrava shleshma*

This *Prakupita shleshma* circulates throughout the body and ends up in *Shareera Shithilatha*

(Due to *Samana guna bhuyishtatwat* of *Medas* and *Shleshma*) *prakupita medas* mixes with *Shleshma*

Due to this increased permeability there

Toxins will be leaked

Low grade inflammation

Insulin resistance

Sthoulya

DISCUSSION

Due to fasting, eating during indigestion, overeating, irregular eating habits, consumption of unsuitable, heavy, cold, excessively dry, or contaminated food. Due to improper administration of purgation, emesis, and oleation; due to debility caused by disease; due to unsuitable place, time, and season; and due to suppression of natural urges, *Agni* becomes vitiated. When *Agni* is impaired, it cannot digest even light food properly. Such improperly digested food undergoes fermentation (*Shukratva*) and acquires toxic properties, becoming similar to poison.^[11] When *Agni* becomes weak, the *Ahara rasa* remains improperly processed. This vitiated, undigested material present in the *Amashaya* is known as *Ama*.^[12]

Modern research demonstrates that diets rich in processed foods, refined carbohydrates, saturated fats, poor sleep, and chronic psychological stress disrupt the composition and function of the gut microbiota. These alterations reduce beneficial microorganisms, particularly *Akkermansia muciniphila*, impair mucin turnover, weaken intestinal tight junctions, and increase intestinal permeability. The subsequent translocation of bacterial products such as lipopolysaccharide (LPS) into the circulation induces metabolic endotoxemia, chronic low-grade inflammation, and insulin resistance, all of which play central roles in the development of obesity and its metabolic complications.

A similar sequence can be appreciated in the *Ayurvedic* understanding of *Sthoulya*. Excessive indulgence in *Santarpana Ahara* and *Vihara* initiates *Achaya purvaka Sanchaya* of *Kapha*, which may be interpreted as a state of *Vilayana Rupa Vriddhi*, characterized by *Kapha prakopa*. This aggravated *Kapha* undergoes *Dosha-Dushya Anubandha*, resulting in the formation of *Bahudrava Shleshma*. Owing to the *Samana Guna Bhuyishatva* shared by *Kapha* and *Meda*, the aggravated *Shleshma* readily associates with *Meda Dhatu*, promoting excessive fat accumulation and ultimately manifesting as *Sthoulya*.

From a modern scientific perspective, the reduction of *Akkermansia muciniphila* and the subsequent disruption of the intestinal mucus barrier provides a possible biological link to this *Ayurvedic* concept. Decreased utilization of mucin by beneficial bacteria may interfere with the normal maintenance and renewal of the mucus layer, while impaired tight junction integrity can increase intestinal permeability. This allows microbial components and inflammatory molecules to enter the circulation, promoting chronic low-grade inflammation and contributing to metabolic disturbances associated with obesity. Conceptually, this circulating inflammatory milieu may resemble with the systemic spread of aggravated *Bahudrava Sleshma* described in *Ayurveda*. Likewise, the resulting impairment in tissue function and metabolic regulation may parallel the classical description of *Shareera Shitilatha*, a state

characterized by loss of tissue compactness and abnormal accumulation of *Meda*(fat).

Overall, these observations suggest that the intestine may be a key link between traditional *Ayurvedic* knowledge and modern microbiome research. Rather than interpreting gut dysbiosis as a direct equivalent of *Ama*, or *Bahudrava Sleshma*, it may be viewed as a biological process capable of explaining certain physiological changes described in the *Ayurvedic* texts.

Restoration of gut microbial diversity through dietary modification, caloric restriction, increased dietary fibre, physical activity has been shown to improve intestinal barrier integrity, reduce inflammation, and enhance insulin sensitivity. These findings support the *Ayurvedic* approach of using *Nidana Parivarjana*, *Langhana*, *Deepana*, *Pachana*, and *Shodhana* to restore metabolic balance before addressing excess accumulation of *Meda*. Although the mechanisms are described differently, both approaches emphasize correcting the underlying metabolic disturbance rather than merely reducing the body weight.

CONCLUSION

Maintenance of intestinal homeostasis appears to be fundamental to normal metabolic function. Disturbances in the gut microbial ecosystem compromise mucosal integrity, increase intestinal permeability, and initiate a persistent inflammatory response that promotes insulin resistance and abnormal fat deposition. This sequence may be viewed alongside the progression from *Santarpana Ahara Vihara* to *Achaya purvaka Kapha Sanchaya*, *Bahudrava Sleshma* formation and *Dosha Dushya Anubandha* (*kapha*, *rasa*, *mamsa* and *meda dhatu*), which ultimately facilitates the involvement of *Meda* and the manifestation of *Sthoulya*. Together, these findings suggest that obesity develops through a sequence of events that starts in the gut and progressively influences metabolism and tissue function. This understanding shifts the focus towards maintaining intestinal integrity, restoring metabolic balance, and intervening early to prevent disease progression.

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