



Review Article

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ETIOLOGICAL AND PREVENTIVE PERSPECTIVES OF *STHAULYA* (OBESITY): AN INTEGRATIVE *AYURVEDIC* AND BIOMEDICAL NARRATIVE REVIEW

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ABSTRACT

Background: *Sthaulya* is an *Ayurvedic* clinical construct characterised by excessive *Meda* and *Mamsa*, impaired functional capacity and disturbed metabolic regulation. It overlaps with, but is not identical to, modern obesity. **Objective:** To critically synthesize *Ayurvedic* etiological and preventive concepts of *Sthaulya* with contemporary evidence on obesity. **Methods:** A focused narrative review of major *Ayurvedic* compendia and selected authoritative reports, guidelines, systematic reviews and landmark biomedical studies was undertaken. Evidence was organised around causation, pathophysiology, assessment and prevention; proposed cross-system correspondences were treated as conceptual rather than proven biological equivalences. **Results:** *Ayurvedic* descriptions emphasise excessive or inappropriate nourishment (*Santarpana*), heavy, unctuous and sweet foods, overeating, insufficient exercise, inappropriate daytime sleep, psychological and hereditary susceptibility, followed by *Kapha-Meda* increase, impaired *Agni* and *Srotorodha*. Biomedical evidence identifies interacting genetic, neuroendocrine, behavioural, environmental and adipose-tissue mechanisms. The strongest convergence concerns modifiable determinants: food quality and quantity, physical inactivity, sedentary time, sleep disruption, stress and an obesogenic environment. *Nidana Parivarjana* provides a useful organising principle for personalised and population-level prevention when combined with validated cardiometabolic assessment and non-stigmatising care. **Conclusion:** An integrative approach may enrich prevention by combining *Ayurveda's*

attention to routine, food qualities and individual context with biomedical risk stratification and evidence-based intervention. Literal translation of *Ayurvedic* constructs into biomedical mechanisms should be avoided, and better-designed interdisciplinary research is required.

Keywords: *Sthaulya*; obesity; *Nidana Parivarjana*; *Meda*; *Ayurveda*; prevention

1. INTRODUCTION:

Obesity is a chronic, relapsing and multifactorial disease in which excessive or dysfunctional adipose tissue impairs health. It is not adequately explained by personal choice or a simple failure of willpower. Biological susceptibility, food systems, socioeconomic conditions, sleep, stress, medications, the built environment and opportunities for physical activity interact across the life course.[1,4-6] This broader framing is important because prevention that focuses only on individual calorie restriction often produces modest and poorly sustained results.

The burden is substantial. In 2022, 2.5 billion adults were overweight, including more than 890 million living with obesity; approximately 16% of adults worldwide had obesity. Adult obesity prevalence more than doubled between 1990 and 2022, while obesity among children and adolescents aged 5-19 years increased fourfold.[1-3] India is simultaneously facing undernutrition and rapidly rising generalized and abdominal obesity, with marked variation by sex, residence, age and socioeconomic position.[7,8] South Asian populations also develop cardiometabolic risk at lower body-mass index (BMI) and waist measurements than many European-origin populations, making central adiposity and metabolic risk especially relevant.[9]

Ayurveda describes a clinically comparable, though not identical, construct under *Sthaulya* or *Atisthaulya*. The classical account places excessive *Meda* and *Mamsa*, impaired tissue nourishment, disturbed *Agni*, *Kapha-Meda* predominance and obstruction of channels within a coherent etiopathogenic model.[10-14] Modern obesity and *Sthaulya* should therefore not be treated as exact synonyms. They overlap in phenotype and risk factors, but they arise from different epistemological frameworks. A careful integrative review can identify useful convergences without forcing one-to-one equivalence.

This narrative review critically synthesizes *Ayurvedic* descriptions of *Nidana*, *Samprapti* and *Nidana Parivarjana* with biomedical evidence on energy regulation, adipose-tissue biology, behavioural determinants and prevention. Its purpose is conceptual and preventive; it does not claim that *Ayurvedic* categories have already been validated as biomedical mechanisms.

2. REVIEW APPROACH:

A focused narrative review was undertaken using major *Ayurvedic* compendia-*Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya* and *Bhavaprakasha*-with emphasis on passages concerning *Atisthaulya*, *Santarpanottha Vyadhi*, *Meda*, *Agni*, *Srotas*, diet, activity and preventive conduct.[10-14] Biomedical literature was selected from authoritative public-health reports, consensus statements, clinical guidelines, systematic reviews and landmark mechanistic

studies indexed in major scholarly databases. Priority was given to sources that directly informed epidemiology, pathophysiology, dietary and behavioural determinants, physical activity, sleep, psychosocial factors, *yoga* or preventive policy.[1-9,15-50]

Because this is a narrative rather than systematic review, no claim of exhaustive retrieval or formal risk-of-bias pooling is made. Sources were synthesized thematically. Classical concepts were first interpreted within their own textual context and only then compared with biomedical constructs. Proposed correspondences are presented as heuristic parallels, not proof of biological identity.

3. AYURVEDIC CONCEPTUALISATION OF *STHAULYA*

Sthaulya denotes pathological corpulence associated with excessive increase of *Meda* and *Mamsa*. Charaka includes *Atisthauilya* among the eight undesirable bodily constitutions because it is associated with diminished longevity and activity, difficulty in sexual function, weakness, offensive body odour, excessive sweating, hunger and thirst.[10] The often-cited description is:

“मेदोमांसातिवृद्धत्वाच्चलस्फिगुदरस्तनः।
अयथोपचयोत्साहो नरोऽतिस्थूल उच्यते॥” [10]

This description is clinically richer than body weight alone: it draws attention to regional laxity or pendulous fat, disproportionate tissue growth and impaired functional capacity. It also distinguishes a disease-prone phenotype from healthy robustness. Sushruta and later compendia similarly connect abnormal *Meda* accumulation with impaired movement, metabolic disturbance and complications.[12,14]

In the classical model, *Santarpana*-excessive or inappropriate nourishment-is a central context. Repeated exposure to heavy, unctuous and sweet foods, excessive intake, lack of exercise, daytime sleep and a life with minimal physical or mental exertion favours *Kapha* and *Meda* increase.[10,11] Hereditary susceptibility is expressed through *Beeja-dosha*, while individual constitution, digestive capacity, age, season and habitual adaptation influence whether exposure produces disease.

Agni is pivotal. Impaired digestive and tissue-level transformation is understood to permit maladaptive nourishment of *Meda* while other tissues receive inadequate or disordered nutrition. Increased *Meda* and *Kapha* obstruct the pathways of *Vata*, particularly within the abdominal region; the displaced or confined *Vata* may stimulate appetite, creating a cycle of frequent intake, continued deposition and progressive dysfunction.[10,13] This explanation is not a biochemical pathway, yet it represents a systems model linking appetite, metabolism, tissue distribution and behaviour.

The clinical assessment of *Sthaulya* is therefore multidimensional. Body habitus is considered alongside appetite, thirst, sweating, exertional capacity, sleep, bowel pattern, dietary habits, mental state and associated disorders. This approach has contemporary value

because BMI alone neither measures adipose distribution nor captures metabolic health, functional limitation or lived experience.[9,35,48]

Table 1. Core features of *Sthaulya* in classical *Ayurvedic* description

Domain	Classical formulation	Clinical implication
Phenotype	Excessive <i>Meda</i> and <i>Mamsa</i> ; pendulous abdomen, buttocks and breasts	Adiposity assessed with distribution and function, not body weight alone
Metabolic context	<i>Santarpana</i> , impaired <i>Agni</i> and disproportionate tissue nourishment	Chronic overnourishment with disturbed metabolic regulation
Dominant factors	<i>Kapha-Meda</i> increase with disturbed <i>Vata</i>	A self-reinforcing appetite-intake-adiposity pattern
Functional effects	Reduced enthusiasm, movement and endurance; sweating, hunger and thirst	Functional impairment and symptom burden require assessment
Prevention	<i>Nidana Parivarjana</i> with regulated diet, activity and routine	Remove maintainers of disease through personalised, sustainable change

Source: Synthesised from *Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya* and *Bhavaprakasha*. [10-14]

4. ETIOLOGICAL FACTORS: AN INTEGRATIVE ANALYSIS:

4.1 Dietary excess and food quality:

Ayurvedic Aharaja Nidana include frequent or excessive intake of *Guru* (heavy), *Snigdha* (unctuous) and *Madhura* (sweet) foods; overeating (*Atibhojana*); eating before the previous meal is digested (*Adhyashana*); and habitual use of foods that promote *Kapha* and *Meda*. [10,11,14] These terms describe more than nutrient composition: they combine food qualities, quantity, timing, digestibility and the eater's capacity.

Biomedical evidence similarly implicates sustained positive energy balance, but contemporary research shows that food form and environment influence intake. Sugar-sweetened beverages provide rapidly absorbable energy with limited satiety; diets rich in energy-dense fats can facilitate passive overconsumption; and ultra-processed foods are typically designed for convenience, palatability and rapid eating. [17,25-27] Irregular meal timing and frequent eating may also affect circadian and metabolic regulation, although optimal meal frequency cannot be prescribed uniformly. [28] The integrative message is not that every sweet or unctuous food is harmful, but that habitual excess relative to need and metabolic capacity is hazardous.

4.2 Physical inactivity and sedentary behaviour:

Avyayama-insufficient physical exertion-is repeatedly identified in the pathogenesis of *Sthaulya*. [10,13] Modern evidence distinguishes failure to meet exercise recommendations from sedentary behaviour such as prolonged sitting; both can contribute independently to cardiometabolic risk. [29,30] Mechanisation, screen-based work, motorised transport and reduced household or occupational activity collectively decrease daily energy expenditure.

Regular activity improves cardiorespiratory fitness, insulin sensitivity, blood pressure, mood and physical function even when weight loss is modest.[39,50]

4.3 *Divaswapna*, sleep and circadian disruption:

Divaswapna is classically considered *Kapha*-promoting in contexts where daytime sleep is inappropriate. Its meaning should not be reduced to a universal prohibition: *Ayurveda* recognises seasonal, age-related, occupational and illness-related exceptions. From a modern perspective, short, long, irregular or poor-quality sleep can disturb appetite regulation, glucose metabolism and behavioural control.[31] Shift work, late-night eating and circadian misalignment add complexity. The defensible integrative inference is that inappropriate sleep timing and poor sleep quality may reinforce a low-activity, dysmetabolic pattern; direct equivalence between *Divaswapna* and a single modern sleep variable has not been established.

4.4 Psychological and behavioural determinants:

Classical descriptions include freedom from worry or exertion (*Achintana*) among conditions that, when combined with overnourishment and inactivity, favour corpulence.[10] This should not be interpreted as blaming psychological wellbeing. Modern evidence instead shows bidirectional relationships among chronic stress, depression, sleep disruption, emotional eating and obesity.[32-34] Stress-related neuroendocrine responses, food reward, reduced activity and social disadvantage may reinforce one another. Prevention should therefore include compassionate behavioural support and avoid moral judgement.

4.5 Heredity, constitution and life-course susceptibility:

Beeja-dosha acknowledges inherited susceptibility, while *Prakriti* and individual variation influence response to diet and environment. Modern genetic studies demonstrate substantial heritability of adiposity and identify many variants affecting appetite, energy expenditure and fat distribution.[23,24] Genes are not destiny: most common obesity reflects polygenic susceptibility interacting with an obesogenic environment. Pregnancy, early-life nutrition, ageing, endocrine disease and certain medicines further modify risk. Neither framework justifies deterministic labelling; both support personalised risk assessment.

4.6 Environmental and social drivers:

An individual-level account is incomplete. Food price and marketing, urban design, working hours, safe spaces for activity, education, income, gender norms and access to care shape exposure and choice.[5,47] Preventive counselling is unlikely to succeed when the surrounding environment continually promotes high-energy food and inactivity. *Nidana Parivarjana* can therefore be extended from personal avoidance to household, institutional and policy-level reduction of causal exposures.

5. SAMPRAPTI AND BIOMEDICAL PATHOPHYSIOLOGY:

The *Ayurvedic Samprapti* can be summarised as repeated *Santarpana* and incompatible habits leading to *Kapha* aggravation, impaired *Agni*, disproportionate *Meda* formation and *Medovaha Srotodushti*. Progressive *Meda* accumulation produces obstruction (*Srotorodha*) and

altered *Vata* movement, which may intensify appetite and perpetuate intake.[10-14] The principal involved factors are *Kapha* with disturbed *Vata*; *Meda* as the dominant *Dushya*; *Agni* and tissue transformation; and channels concerned with nutrient transport and adipose tissue.

Modern pathophysiology likewise exceeds a simple storage model. Adipose tissue is an endocrine and immune organ that releases adipokines, cytokines and metabolites.[18,19] With hypertrophy and ectopic lipid deposition, adipose dysfunction can promote inflammation, insulin resistance, dyslipidaemia and vascular injury.[20,21,35,36] Leptin and other appetite signals participate in homeostatic regulation, while weight loss activates compensatory responses that favour regain.[22] These mechanisms explain why obesity is often relapsing and why long-term support is required.[4,48]

The two models converge at a high level: chronic overnourishment, impaired regulation, abnormal tissue accumulation, altered appetite and multisystem complications form reinforcing cycles. However, *Agni* is not synonymous with basal metabolic rate, *Ama* is not a validated biomarker, *Srotorodha* is not identical to atherosclerosis, and *Kapha* is not a hormone. Such literal substitutions weaken rather than strengthen integrative scholarship.

A more defensible use of correlation is functional. *Meda Vriddhi* may be discussed alongside excess adiposity; impaired *Dhatvagni* alongside disturbed tissue handling of nutrients; and *Srotorodha* alongside impaired transport or signalling. These are conceptual bridges for generating research questions, not established translations.

Table 2. Responsible use of Ayurvedic-biomedical conceptual parallels

<i>Ayurvedic construct</i>	Potential functional parallel	Required caution
<i>Santarpana</i>	Chronic overnutrition / energy-rich environment	Not equivalent to calorie count alone
<i>Meda Vriddhi</i>	Excess or dysfunctional adiposity	<i>Meda</i> has a broader classical meaning than adipose tissue
<i>Agnimandya</i> / <i>Dhatvagni</i> disturbance	Impaired digestive or tissue metabolic regulation	Not synonymous with basal metabolic rate or a single enzyme
<i>Srotorodha</i>	Obstructed transport, flow or signalling	Not identical to vascular occlusion
<i>Beeja-dosha</i>	Inherited susceptibility	Does not imply simple Mendelian causation
<i>Nidana Parivarjana</i>	Risk-factor removal and exposure reduction	Must address social and environmental constraints

6. ASSESSMENT AND RISK STRATIFICATION:

BMI remains a practical screening index, calculated as weight in kilograms divided by height in metres squared. WHO defines overweight in adults as BMI ≥ 25 kg/m² and obesity as BMI ≥ 30 kg/m². [1] For Asian Indians, lower action points have been proposed because diabetes and cardiovascular risk occur at lower BMI and waist values; these thresholds should be clearly identified as population-specific rather than labelled as WHO categories.[9]

BMI should be supplemented by waist circumference or waist-to-height ratio, blood pressure, glucose status, lipids, liver risk, sleep-apnoea symptoms, medications, functional

status and psychosocial assessment.[35-37,48] Clinical language should be person-first and non-stigmatising.[49] A person may have important metabolic risk without very high BMI, while another may have obesity without the same immediate complication profile.

From an *Ayurvedic* perspective, assessment may additionally document *Prakriti*, *Agni*, *Koshtha*, dietary qualities, meal timing, sleep, activity, appetite, thirst, sweating, bowel habits, mental factors and classical features of *Sthaulya*. These observations can guide individualised counselling, but they should be recorded with reproducible tools and should not replace validated cardiometabolic risk assessment.

Table 3. Adult anthropometric thresholds and interpretation

Framework	BMI / waist threshold	Interpretation
WHO	Overweight: $\geq 25 \text{ kg/m}^2$; obesity: $\geq 30 \text{ kg/m}^2$	Global adult screening categories [1]
Asian Indian consensus	Overweight: $23.0\text{--}24.9 \text{ kg/m}^2$; obesity: $\geq 25 \text{ kg/m}^2$; abdominal obesity: waist $\geq 90 \text{ cm}$ (men), $\geq 80 \text{ cm}$ (women)	Lower action points reflect cardiometabolic risk at lower adiposity [9]
Clinical assessment	BMI plus waist measure, complications, function and context	Avoid diagnosis or treatment decisions based on BMI alone [35-37,48]

7. PREVENTION THROUGH *NIDANA PARIVARJANA*:

7.1 Principle and scope:

Nidana Parivarjana-removal or reduction of causative and aggravating factors-is the logical foundation of prevention. Applied to *Sthaulya*, it requires identifying the specific pattern maintaining excess intake, inactivity, inappropriate sleep, stress or dysregulated routines in an individual. The aim is not abrupt deprivation but sustainable correction matched to capacity, comorbidity and context.

7.2 Dietary regulation:

A preventive pattern emphasises minimally processed foods, vegetables, pulses, whole grains, adequate protein and fibre, while limiting sugar-sweetened beverages, excessive fried foods and ultra-processed products.[25-27,46] Portion size and meal regularity matter, but prescriptions must remain nutritionally adequate and culturally feasible. In *Ayurvedic* terms, quantity should respect digestive capacity; meals should not be repeatedly layered over incomplete digestion; and *Guru*, *Snigdha* or *Madhura* foods should be moderated when they dominate the diet. No single food quality should be isolated from dose, preparation, season, activity and the person consuming it.

7.3 Physical activity and reduction of sitting:

Adults should progressively work toward evidence-based activity targets while reducing prolonged sitting.[39,50] Aerobic exercise produces dose-related, generally modest reductions in weight and waist circumference and offers broader cardiometabolic benefits.[40] Resistance training helps preserve or increase lean mass and improves body composition.[41] For deconditioned individuals, small increments-walking after meals, active transport,

household activity and scheduled movement breaks-may be safer and more sustainable than an aggressive initial prescription.

7.4 Yoga and mind-body support:

Yoga can combine postures, breathing, attention and relaxation, making it a potentially acceptable adjunct for some people. Meta-analyses suggest modest improvements in weight-related or cardiometabolic outcomes, but study heterogeneity and variable methodological quality limit certainty.[42-45] *Yoga* should complement, not replace, dietary, activity and medical care. Selection and progression should account for joint disease, severe obesity, cardiovascular risk, pregnancy and other clinical constraints.

7.5 Sleep, stress and behavioural care:

Consistent sleep opportunity, evaluation for obstructive sleep apnoea, alignment of meals and activity with daily rhythms, and management of stress can remove barriers to weight regulation.[31-34] Behavioural strategies include self-monitoring, realistic goals, problem solving, stimulus control, relapse planning and supportive follow-up.[37,48] Weight-neutral improvements-fitness, sleep, blood pressure, glycaemia, function and quality of life-should be recognised even when the scale changes slowly.

7.6 Community and policy prevention:

Schools, workplaces, primary care systems and local governments can improve food environments, provide safe opportunities for activity, limit harmful marketing to children and make prevention accessible across socioeconomic groups.[47] Such measures reflect a population-level interpretation of causal avoidance. They also reduce the tendency to locate responsibility solely within the individual.

8. AREAS OF CONVERGENCE AND CAUTION:

Both *Ayurveda* and biomedicine recognise that obesity develops through interacting dietary, behavioural, constitutional and environmental influences; both place prevention and routine regulation at the centre of long-term care. *Ayurveda* contributes a qualitative framework attentive to food properties, timing, digestive capacity, daily regimen and individual variation. Biomedicine contributes standardised anthropometry, mechanistic evidence, risk prediction, population surveillance and tested intervention frameworks.

Integration is strongest when it is additive and transparent: classical assessment can enrich history-taking; *yoga* and routine-based counselling can support behaviour change; and biomedical screening can identify complications requiring timely treatment. Integration becomes unsafe when metaphorical correspondences are presented as laboratory facts, when classical advice is universalised without exceptions, or when evidence-based treatment is delayed.

The current evidence base has important limitations. Much *Ayurvedic* obesity literature is conceptual, observational or based on small heterogeneous trials. Diagnostic constructs and outcome measures are often inconsistently operationalised. Future studies should prospectively define *Sthaulya* features, validate assessment instruments, register protocols, use adequate samples, report adverse events, include clinically meaningful outcomes and compare integrative programmes with credible controls. Mixed-methods research may clarify acceptability, adherence and the lived experience of personalised prevention.

9. IMPLICATIONS FOR PRACTICE:

A practical integrative prevention pathway begins with respectful screening for BMI, waist-related risk, comorbidities, medicines, sleep and functional limitations. The clinician then maps the person's dominant causal pattern: dietary excess or poor food quality; inactivity or prolonged sitting; disrupted sleep; stress or emotional eating; medical contributors; or environmental constraints. *Ayurvedic* history can add information about meal timing, perceived digestive capacity, habitual qualities of food and daily routine.

The plan should select a small number of high-yield, measurable changes, agree on follow-up and anticipate relapse. Referral is appropriate for suspected endocrine disease, eating disorders, severe sleep apnoea, major depression, obesity-related complications or consideration of anti-obesity medication or metabolic surgery. Lifestyle and *yoga* advice should be coordinated with, rather than positioned against, indicated medical care.[37,48]

10. CONCLUSION:

Sthaulya and modern obesity are overlapping but non-identical constructs. Classical *Ayurveda* depicts a self-reinforcing disorder of overnourishment, *Kapha-Meda* increase, impaired *Agni*, channel obstruction, altered appetite and reduced function. Biomedical science characterises obesity as a chronic relapsing disease shaped by adipose dysfunction, neuroendocrine regulation, genetics, behaviour and an obesogenic environment.

The most clinically useful convergence lies in prevention: moderation of excessive and poorly timed intake, improved food quality, regular movement, reduced sitting, appropriate sleep, stress support and sustained follow-up. *Nidana Parivarjana* offers a coherent organising principle, provided it is applied without blame and supported by validated risk assessment. Rigorous interdisciplinary research is needed before specific *Ayurvedic*-biomedical correlations can be treated as established mechanisms.

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critical revision. Atul Kumar contributed to the interpretation of classical *Ayurvedic* concepts and reviewed the manuscript. All authors read and approved the final manuscript.

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