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Śānto Agniḥ Mriyate: Understanding Sepsis and Multi-Organ Failure through Dhatvagni and Hormonal Regulation

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Abstract

- Ayurvedic perspective: Concept of agni 1) jatharagni 2) bhutagni 3) dhatvagni.
- Modern perspective: Hormonal regulation of metabolism in tissues (insulin, glucagon, thyroid, cortisol, GH, sex hormones).
- Rationale: Tissue-level metabolism in Ayurveda (dhatvagni) has conceptual overlap with hormone-controlled tissue energetics in modern physiology.
- Research gap: Lack of systematic mapping between dhatvagni and endocrine regulation.
- Objective: To propose a conceptual model linking dhatvagni with modern endocrine functions.

Metabolism is the sum of all chemical reactions in the body, encompassing both anabolic (building) and catabolic (breaking down) processes. Hormones, secreted by various endocrine glands, serve as the body's regulatory signals, orchestrating when and how energy is stored or utilised. Their precise balance ensures stable blood glucose, efficient energy use, and adaptation to stress, growth, and reproduction.

Keywords: Dhatvagni, Hormonal regulation, Metabolism, Endocrinology, Ayurveda, Integrative physiology

Aim: To correlate Ayurvedic dhatvagni with hormonal regulation of metabolism at tissue level.

Methods: Literature review & conceptual framework building.

Literature review

In Ayurveda

- Agni = not only digestive fire (Jatharagni), but also Bhūtāgni (elemental metabolism) and Dhātvagni (tissue-level metabolism).
- It represents all forms of metabolic energy, enzymatic activity, and tissue transformations that sustain life.

In Modern Physiology

- This can be compared to cellular metabolism and bioenergetics:

- ATP generation via mitochondrial oxidative phosphorylation.
- Hormonal regulation of basal metabolic rate (thyroid hormones), nutrient utilisation (insulin, glucagon), and energy release (cortisol, GH, catecholamines).

• If metabolism ceases (e.g., no oxidative phosphorylation, enzyme activity, or energy production) → death follows immediately.

• **Ayurvedic Concept of Dhatvagni [1]**

1. Rasa dhatvagni – nutrient assimilation in plasma.
2. Rakta dhatvagni – oxygen transport and haemopoietic metabolism.
3. Mamsa dhatvagni – muscle growth & repair.
4. Meda dhatvagni – lipid metabolism & adipose regulation.
5. Asthi dhatvagni – bone metabolism.
6. Majja dhatvagni – marrow & nervous tissue metabolism.
7. Shukra dhatvagni – reproductive tissue metabolism.

• **Modern Endocrine Regulation of Tissue-level Metabolism [2]**

• Insulin & Glucagon – carbohydrate, lipid, and protein metabolism.
• Thyroid hormones – basal metabolic rate, tissue oxygen consumption.
• Cortisol & Glucocorticoids – protein catabolism, gluconeogenesis.
• Growth Hormone – protein anabolism, lipolysis.
• Sex Hormones – tissue-specific growth & differentiation.
• PTH, Calcitonin, Vitamin D – bone metabolism.

Insulin – The Anabolic Hormone

• Source: β-cells of the pancreas (Islets of Langerhans)
• Function:
- Promotes glucose uptake into skeletal muscle and adipose tissue.
- Stimulates glycogenesis (glycogen storage in liver and muscle).
- Enhances lipogenesis (fat storage) and protein synthesis.
- Inhibits gluconeogenesis and lipolysis.

• Clinical relevance: Deficiency (Type1 diabetes) or resistance (Type2 diabetes) leads to hyperglycaemia and metabolic dysfunction.

Glucagon – The Catabolic Counterpart

• Source: α-cells of the pancreas.
• Function:
- Stimulates glycogenolysis and gluconeogenesis in the liver.
- Increases blood glucose during fasting.
- Promotes lipolysis to provide fatty acids as energy fuel.

• Balance with insulin: Together, insulin and glucagon form the primary glucose regulatory system.

Adrenal Hormones – Acute and Chronic Stress Regulators

Adrenaline (Epinephrine)

• Source: Adrenal medulla.
• Role:
- Triggers “fight-or-flight” response.

• Mobilises glycogen (glycogenolysis) and fats (lipolysis).
- Increases heart rate and oxygen delivery to tissues.

Cortisol

- Source: Adrenal cortex.
- Role:
  - Maintains glucose during prolonged stress by stimulating protein catabolism and gluconeogenesis.
  - Shifts fat distribution and suppresses the immune system.
  - Chronic elevation leads to obesity, insulin resistance, and muscle wasting.

Thyroid Hormones – The Metabolic Accelerators

- Source: Thyroid gland (T3, T4).
- Functions:
  - Increase basal metabolic rate (BMR).
  - Stimulate mitochondrial activity and oxygen consumption.
  - Regulate growth and development in children.
- Disorders:
  - Hypothyroidism → slowed metabolism, weight gain, fatigue.
  - Hyperthyroidism → weight loss, heat intolerance, tachycardia.

Growth Hormone (GH) – Balancing Growth and Energy

- Source: Anterior pituitary.
- Effects:
  - Stimulates protein synthesis and muscle growth.
  - Promotes lipolysis as an energy source.
  - Antagonizes insulin by reducing glucose uptake in tissues.
- Excess/deficiency:
  - Deficiency in children → dwarfism.
  - Excess in childhood → gigantism; in adults → acromegaly.

Appetite and Energy Regulator Hormones [3]

Leptin

- Source: Adipose tissue.
- Role:
  - Signals satiety to the hypothalamus.
  - Suppresses appetite and increases energy expenditure.
  - Leptin resistance → obesity.

Ghrelin

- Source: Stomach.
- Role:
  - Stimulates hunger before meals.
  - Increases growth hormone release.

Sex Hormones and Metabolism

Estrogen

- Enhances insulin sensitivity.
- Promotes fat storage in gluteofemoral regions.
- Deficiency (post-menopause) linked to central obesity and metabolic syndrome.

Testosterone

- Increases muscle protein synthesis and basal metabolic rate.

- Supports fat oxidation.
- Low levels associated with obesity and insulin resistance.
- **Hormonal Dysregulation in Disease**
- Diabetes mellitus → insulin deficiency/resistance.
- Cushing's syndrome → cortisol excess.
- Addison's disease → cortisol deficiency.
- Hypothyroidism/Hyperthyroidism → imbalance in BMR.
- Obesity → leptin resistance and altered ghrelin signalling.
- Mandāgni (hypofunction of Agni) - hypothyroidism, metabolic syndrome.
- Tikshṇāgni (hyperactive Agni) - hyperthyroidism, catabolic states.
- Vishamāgni (irregular Agni) - diabetes, stress-related metabolic fluctuations.
- Complete absence of Agni - biological death (cessation of metabolism).

शान्तोऽग्निः म्रियते - [4]

"Śānto 'gniḥ mriyate" is an Ayurvedic way of saying:

Life depends on metabolism; when metabolism stops, life ends.

- Śāntaḥ agniḥ → "When Agni becomes extinguished"
- Mriyate → "Death occurs"

Thus, the statement means: "When Agni ceases to function, life ceases."

Sepsis is a powerful modern clinical correlate for the Ayurvedic dictum "śānto 'gniḥ mriyate" and derangements of Agni.

- In sepsis, there is a collapse of metabolic fire (Agni) at both systemic (jatharagni) and tissue level (dhātvāgni).
- Classical Ayurveda describes that when Agni is extinguished, life cannot be sustained → "śānto 'gniḥ mriyate."
- Features of mandāgni (sluggish fire) and viṣamāgni (irregular fire) can be mapped to the dysregulated, chaotic metabolism in sepsis.

Modern Physiological Perspective [5]

- Sepsis is life-threatening organ dysfunction due to dysregulated host response to infection.
  - Metabolic failure → mitochondrial dysfunction, impaired oxidative phosphorylation.
  - Hormonal dysregulation →
    - Cortisol ↑ (stress response)
    - Catecholamines ↑ (shock compensation)
    - Insulin resistance ↑ → hyperglycemia
- Thyroid dysfunction (euthyroid sick syndrome) — decreased T_3 , altered metabolism
- Cytokine storm disrupts normal cellular fuel use.
- Ultimately → energy crisis in cells, lactate accumulation, multi-organ dysfunction, and if uncontrolled → death.
- This metabolic collapse = equivalent to Agni extinction.
- Sepsis = collapse of Agni at multiple dhatu levels:
  - Rasa (glucose handling),
  - Mamsa (muscle catabolism),

- Meda (lipid mobilization),
- Rakta (vascular dysfunction),
- Majja (neuroendocrine crisis).

- **Acute Sepsis Characteristics:** Hypercortisolism, catecholamine surge, RAAS activation, and hyperglycaemia.
- **Chronic Sepsis Characteristics:** Hormonal exhaustion, adrenal non-response, thyroid suppression, and wasting.
- Early sepsis = tikṣṇāgni (overactive catabolic metabolism).
- Late sepsis = mandāgni / agni nāsha (failure of all metabolic fire).
- This validates the dictum "śānto 'gniḥ mriyate" — death in sepsis is due to collapse of tissue-level metabolic regulation (both in Ayurveda and modern terms).

Discussion

- Similarities and differences between dhatvagni and hormonal regulation.
- Physiological basis of agnimandya (reduced metabolism) in hormonal disorders (e.g.

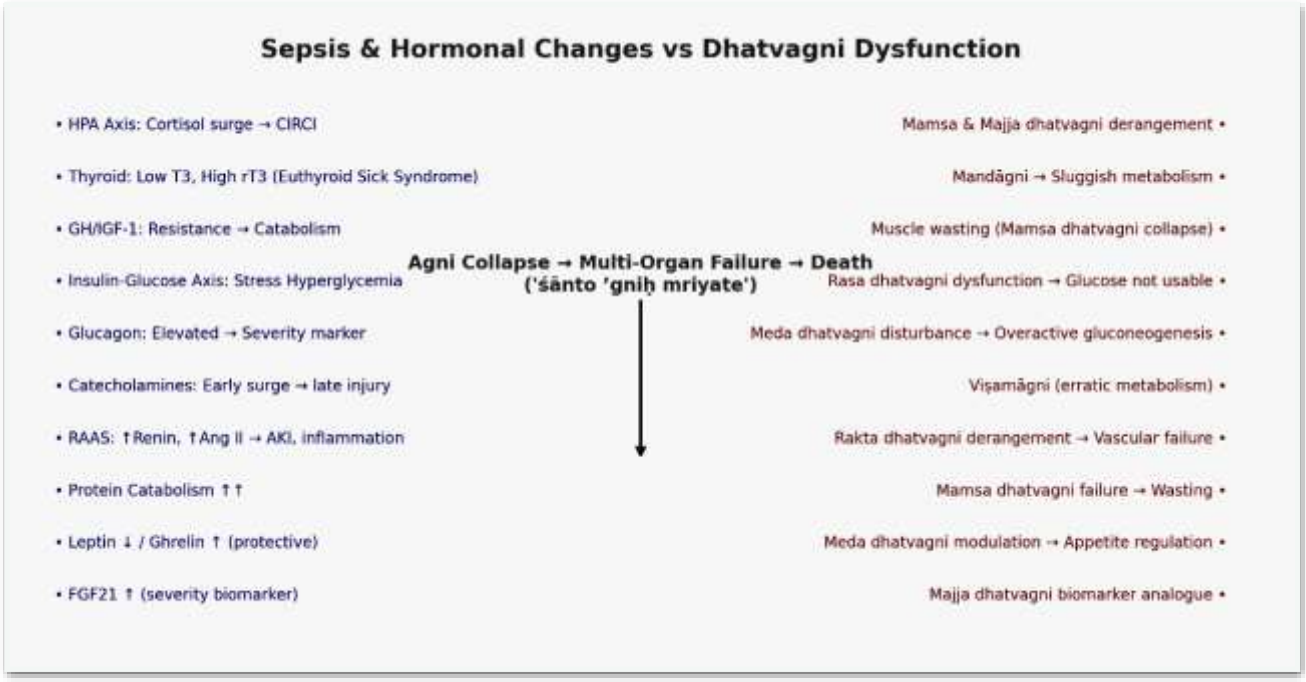

Figure 1 - Ayurveda's and contemporary changes of metabolism in sepsis

hypothyroidism, diabetes).

- Possible biomarkers for studying dhatvagni activity (e.g., metabolic rate, hormone levels, lipid profile).
- **Sepsis Definition:** Dysregulated host response to infection leading to multi-organ dysfunction.
- **Sepsis Mortality:** ~50% of hospital deaths linked to sepsis, with mortality rates increasing from 10-20% (mild) to nearly 50% (septic shock).
- **Ayurvedic Correlation:** Sepsis reflects the Ayurvedic concept of "śānto 'gniḥ mriyate" and the disruption of Agni.
- Integrative implications for preventive and therapeutic approaches.

Conclusion

Hormones are the commanders of metabolism, integrating signals from diet, energy demand, and stress to maintain homeostasis. Insulin and glucagon balance glucose, adrenaline and cortisol handle stress responses, thyroid hormones set metabolic rate, growth hormone ensures tissue development, while leptin and ghrelin fine-tune appetite. Disruptions in these pathways underlie many metabolic diseases, making hormone balance essential for health, longevity, and optimal performance. Sepsis disrupts various hormonal pathways, including the HPA, HPT, growth hormone/IGF-1, insulin-glucose, leptin, RAAS, and gonadal axes. Acute sepsis causes hypercortisolemia, hyperglycemia, thyroid suppression, and surges in catecholamines and glucagon, mobilising energy for infection. Chronic sepsis leads to hormonal exhaustion, insulin resistance, protein catabolism, and muscle wasting. FGF21 and ghrelin may serve as biomarkers and therapeutic targets. Endocrine alterations are both adaptive survival responses and contributors to multi-organ failure, making them crucial for prognosis and therapy in sepsis.

- Dhatvagni and hormonal regulation describe similar principles of tissue-specific metabolism through different knowledge systems.
- An integrative model may enhance understanding of metabolic disorders and guide holistic interventions.

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