

REVIEW ARTICLE

Critical Evaluation of Beejadusti in Prameha: A Review

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ABSTRACT

The ancient Indian medical system, Ayurveda, provides a comprehensive framework for understanding health and disease, incorporating genetic principles. In Ayurveda, genetics is conceptualized through terms such as Beeja, Beejabhaga and Beejabhagavayava which represent hereditary units analogous to modern genetic material. The concept of Prakriti (individual constitution) is also central, reflecting an individual's inherent biological profile established at conception, which influences both physical and mental characteristics throughout life. Ayurveda classifies certain diseases, such as Prameha, as hereditary conditions associated with genetic imbalances or defects. Prameha is characterized by metabolic disorders resembling Diabetes Mellitus and is considered a Kulaj Vikara attributed to Beeja Dosha (genetic disorders). Ayurvedic literature acknowledges that while genetic factors are crucial in the development of these conditions, lifestyle factors such as diet and exercise also significantly impact disease manifestation. This integrated perspective highlights the interaction between genetic predispositions and environmental influences in the management of health and disease.

KEYWORDS: Prameha, Beejadusti, Diabetes mellitus, Genetics, Prakriti, Ayurveda.

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INTRODUCTION

Acharya Charak identified Prameha as one of the eight major diseases (Ashtomahagad) in Ayurveda [1]. He explained the origin of Prameha in Nidansthan, stating that it is a hereditary disorder (Kulaj Vikar) and that Jataj Prameha (juvenile diabetes) is incurable [2]. If not properly managed, the disease becomes Asadhya (difficult to treat) in later stages. The defining characteristic of this disease is increased frequency and volume of urination, which is why it is called Prameha. Ayurveda classifies Prameha into 20 types: 10 types of Kaphaj Prameha, 6 types of Pittaj Prameha, and 4 types of Vataj Prameha. It is considered a life-threatening lifestyle disease affecting the Medovaha, Mutravaha, and Udakavaha srotas. Prameha is categorized as a Santarpanajanya Vyadhi (disease due to over-nourishment). Diabetes Mellitus is classified into Type 1 and Type 2 in modern medicine, and according to the WHO, around 220 million people have Type 2 Diabetes. It is a rapidly increasing health threat, often referred to as a "silent killer." India has the highest number of Diabetes cases worldwide, with the WHO recognizing it as the "Diabetes Capital of the World" [3]. Since Prameha is a Tridoshaj Vyadhi involving ten Dushyas (affected tissues), imbalances between doshas and dushyas can occur. Ayurvedic texts mention that Sahaj Prameha arises from defects in Beeja (genetic material). In Ayurveda, there is a belief that the visible world is limited, while the invisible is vast [4]. The invisibility is due to subtleness (Shaukshmya) [5], and any invisible entity can be perceived through inference [6]. Although structures like the nucleus, chromosomes, and genes are not visible to the naked eye, their existence can be inferred when hereditary diseases manifest in offspring or when abnormal organs develop. The concept of genetics has existed since the time of ancient Aacharyas, who referred to hereditary diseases using terms like Kulaj [7], Kulodbhava [8], and Sahaj Vikara [9]. These

concepts can now be understood in light of modern genetics, indicating that the roots of genetic knowledge were already present in *Ayurveda* and are referenced in various classical Ayurvedic texts.

MATERIAL AND METHODS

A literary descriptive method has been adopted in this research, with a critical examination of Ayurvedic and modern texts to reach reasoned conclusions.

REVIEW OF LITERATURE

Ayurvedic Aspects

Classical Ayurvedic texts offer comprehensive explanations regarding the etiologies, pathophysiology, types, symptoms, complications, and treatments of *Prameha*. The increased *Drava Guna* (liquidity) of *Kapha Dosha* is thought to contribute significantly to the onset of *Prameha* [10]. The disease affects multiple bodily components, including *Meda* (fat), *Mamsa* (muscle), *Kleda* (moisture), *Sukra* (semen), *Sonita* (blood), *Vasa* (fat tissue), *Majja* (bone marrow), *Lasika* (lymph), *Rasa* (plasma), and *Ojas* (vital energy) [11]. Among these, *Meda* is considered the primary site impacted [12]. The broad range of affected tissues indicates that *Prameha* involves a systemic disorder.

Classification of Prameha

On the basis of etiology: *Prameha* has been classified into two types by *Acharya Sushruta* [13].

- *Sahaj* (Hereditary)
- *Apathyanimitaja* (Acquired)

On the basis of *Doshas* predominance, *Prameha* is categorized into three major types.

- *Vataj Prameha*
- *Pittaj Prameha*
- *Kaphaj Prameha*

The *Dosha* predominance is solely detected by the physical characteristics of urine. Further *Prameha* is sub-classified into 20 types according to different Acharya. On basis of prognosis:

- *Sadhya* -These are new case without complications. They are curable ex:-*Kaphaj, Sthula, Apathyanimitaja* etc.
- *Yapya* -*Pittaj Prameha*.
- *Asadhya* - *Vataj, Jatapramehi*.

In *Ayurveda*, the terms *Sahaj* and *Jatah* refer to a genetic predisposition to diseases such as *Prameha* (Diabetes Mellitus) [14]. Hereditary diseases can be influenced by two main factors:

- *Beeja Dosha*, which is a defect in the sperm or ovum, can lead to genetic disorders or a predisposition to disease. In *Charak Samhita*, it is noted that excessive consumption of *Madhura Rasa* (sweet-tasting foods and drinks) by the parents can cause chromosomal damage in the sperm and ovum, increasing the likelihood of *Prameha* in the offspring.
- An unfavorable intrauterine environment due to the mother's diet, lifestyle, or emotional state during pregnancy can also impact fetal development. This can trigger the onset of disease in a child with a genetic predisposition. In the case of *Prameha*, overconsumption of *Madhura Rasa* by the mother during pregnancy may contribute to the development of *Prameha* in the child.

The mother's lifestyle, diet, and mental state during lactation, in addition to pregnancy, may further influence the onset of *Prameha* in infants. Similarly, a child with a genetic predisposition to *Prameha* is at higher risk if there is an excessive intake of *Madhura Rasa* during childhood.

Thus, both genetic factors and poor dietary and lifestyle habits—especially the overindulgence in sweet foods—can jointly lead to the development of hereditary *Prameha*. The descriptions of *Sahaj Prameha* in *Sushruta Samhita* and *Jatah Prameha* in *Charak Samhita* closely resemble what is now recognized as Type 1 Diabetes, also called juvenile-onset or insulin-dependent Diabetes. The term "*Jatah Pramehi Madhumehino*" in *Charak Samhita* relates to the early onset of Type 1 diabetes in children.

A review of classical Ayurvedic texts reveals several interesting concepts that align with modern genetics. These include:

Non-consanguineous marriage (*Atulya Gotriya*): Ayurveda emphasizes the importance of marrying outside one's own lineage (*Gotra*) to prevent genetic disorders. *Charaka* recommended that, for the birth of a healthy child, the male and female should come from different clans [15]¹⁵, advising against consanguineous marriages. Consanguineous marriage refers to a union between blood relatives who share a common ancestor. Numerous studies have shown that children born from such marriages have a higher risk of morbidity, mortality, and an increased incidence of congenital structural abnormalities [16].

Some studies shows that Parental consanguinity was not significantly associated with T1DM (Type 1 Diabetes Mellitus); however, children of first-cousin parents showed a higher risk of developing T1DM than did children of second-cousin parents. The presence of a family history of T1DM significantly differed between those with and without T1DM [17].

Maternal age and genetic disorders:

Acharya Susruta provided recommendations on the ideal age for conception for both parents, highlighting the importance of the mother's physical condition. He stated that mothers younger than 16 and fathers younger than 25 should refrain from conceiving to prevent the birth of children with undesirable traits [18]. He also cautioned against older women or those who have been trying to conceive for an extended period from attempting to have children [19]. Recent research indicates that maternal age plays a crucial role in the risk of various genetic abnormalities. This concept was first established in clinical medicine by Dr. Langdon Down, who identified Down's syndrome. It is now widely accepted that there is a significant association between increasing maternal age and the likelihood of this condition [20].

A study found that the risk of Type 1 Diabetes Mellitus in children increases by 25% for each five-year increase in maternal age. For example, a mother who gives birth at age 45 or older has a 3.11 times higher risk of having a child with type 1 diabetes than a mother who gives birth before age 20 [21].

A study found that the risk of Type 2 Diabetes Mellitus in children is highest for those born to very young or very old mothers, and lowest for those born to mothers around age 30 [22].

Phenotypic expression of genomes (*Prakriti* or Constitution):

Prakriti consists of the characteristics that an individual develops from the embryonic stage, influenced by the effects of physiologically normal *Doshas*. These traits persist and manifest as behaviors throughout life. The constitution (*Prakriti*) of the fetus (*Garbha*) is determined by several factors [23]:

- **Sukrasonita Prakriti (Sperm and Ovum):** The genetic contribution from both parents.
- **Garbhasaya Prakriti (Season and Uterine Conditions):** The timing and conditions of the uterus during pregnancy.
- **Matur Ahara-Vihara Prakriti (Maternal Diet and Lifestyle):** The dietary choices and regimen of the mother.
- **Mahabhuta Vikar Prakriti (Nature of the Mahabhutas in the Fetus):** The qualities of the five great elements that make up the fetus.

In Ayurvedic terms, *Janma Prakriti* remains constant and forms the foundation of psychophysiological constitution, while *Deha Prakriti*, which is dynamic, changes over time. The genotype corresponds to Ayurvedic *Janma Prakriti*, and the phenotype corresponds to *Deha Prakriti*. A disturbance in *Deha Prakriti* is referred to as *Vikriti* in *Ayurveda*, which aligns with disorders and diseases in modern medicine. Four major factors influence the phenotype or *Deha Prakriti* positively or negatively, depending on one's lifestyle: behavior, diet and digestion, stress, and environmental factors. These factors lead to changes in the phenotype that can affect the expression of the genotype (*Janma Prakriti*) without altering its fundamental structure [24]. Manohar has highlighted vivid accounts in ancient Ayurvedic texts concerning the inheritance of diseases and the genetic basis for the transmission of such conditions from parents to offspring [25].

The predisposition factors for *Prameha* include *Garbhakala Asatmyata* (intrauterine influences), *Shaitihilya* (lack of muscle and adipose tissue firmness), *Meda Asarata* (malfunctioning adipocytes), *Madhur Agraha* (preference for sweet foods), *Kapha Chaya* (accumulation of sero-mucoid substances), *Kapha Prakopa* (aggravation of biofilms), *Mansavahasrotadushti* (hepato-muscular infiltration), *Meda Vriddhi* (increased adiposity), *Avyayama* (lack of physical activity), *Divasvapna* (daytime sleeping), *Medovahasrotadushti* (inflammation of adipose tissues), and *Kleda Dushti* (excess extracellular fluid). The significance of these factors varies depending on an individual's *Prakruti* [26].

Studies have shown a link between *Prakruti* (Ayurvedic body constitution) and Diabetes. Research from the Central Research Institute (Ayurveda), Jaipur, found that *Kapha* or *Kapha* with *Vata/Pitta* types were more common in diabetic patients, with better treatment response to *Nisha Amalaki* in *Kapha* types. A Banaras Hindu University (BHU) study showed a correlation between blood glucose response to exercise and *Prakruti* [26].

Beeja, Beejabhaga, Beejabhaga Avayava

The term "*Beeja*" refers to a small entity capable of replicating itself, similar to how sperm and ovum serve as the seeds for progeny [27]. In *Ayurveda*, sperm provides the paternal component (*Pitruja Bhava*), while the ovum contributes the maternal component (*Matruja Bhava*) in the formation of the fetus. *Ayurveda* emphasizes six essential factors for the proper development and growth of the fetus. These procreative factors are *Matruja* (maternal), *Pitruja* (paternal), *Satvaja* (psyche), *Satmyaja* (habitual), *Rasaja* (nutritional), and *Atmaja* (soul). *Satvaja*, *Rasaja*, and *Satmyaja* are also derived

from the parents and can therefore be integrated into *Matruja* and *Pitruja Bhava* [28]. If either parent suffers from a disease that affects the quality or quantity of the nucleus in the sperm or ovum, it may result in the child inheriting the same condition. It is clearly stated that the *Beeja* is responsible for developing the entire body, and if a part of the *Beeja* becomes vitiated, the corresponding part in the child's body may also become diseased [29].

In *Charak Samhita*, *Acharya* explains that Patients who suffer from *Prameha* since birth (congenital) and those who are borne of *Prameha* parents (hereditary) are not curable because of the morbidity in their *Beeja* (genes). Similarly, other hereditary (*kulaj*/familial) ailments are considered as incurable [30].

In *Sushrut Samhita*, *Acharya* explains that there are two types of *Prameha*: *Sahaj* (inborn or congenital) and *Apathyaja* (caused by unhealthy lifestyle). *Sahaj Prameha* is caused by *Beejadasha*, defects in the reproductive elements (sperm or ovum) of the parents, while *Apathyaja Prameha* arises due to improper or harmful dietary habits [31].

Hereditary diseases (*Sahaj Vyadhi*):

In Ayurvedic classics, terms such as *Sahaj Vyadhi*, *Adibal Pravarita Vyadhi*, and *Kulaj Vikara* describe various hereditary diseases. *Sahaj Vyadhi* refers to diseases that are passed down through generations, such as various skin disorders (*Kustha*) and Diabetes (*Prameha*). These hereditary disorders can be categorized based on their maternal (*Matruja*) or paternal (*Pitruja*) origins [32]. When the pathogenesis of a disease affects the *Sukra Dhatus* (reproductive tissues or related components) in either males, females, or both, it can be transmitted to the next generation.

Once a weak *Dhatu* manifests in progeny, individuals may become more susceptible to developing related diseases with minimal exposure to causative factors. For instance, children of obese individuals or those with Type 2 Diabetes Mellitus may inherit insulin resistance and polyphagia. Due to this underlying condition, they are at risk of developing insulin-resistant Diabetes Mellitus even with slight exposure to risk factors. Moreover, the dietary habits and lifestyle of family members often remain consistent, which can lead to alterations at the epigenetic level [33].

Over two to three generations, these epigenetic changes can result in abnormalities at the genetic level, contributing to gene vitiation responsible for hereditary diseases. A genetic disease is defined as an inherited medical condition caused by a DNA abnormality. Since genes are transmitted from parents to offspring, any changes in the DNA within a gene are also inherited. Such mutations can arise spontaneously, and in Ayurveda, these conditions are classified as *Kulaj*. Each disorder has its unique genetic basis; specific mutations in certain genes can lead to particular genetic disorders, while variations within the same gene may result in different health issues or disorders. Genetic disorders may or may not be hereditary, but hereditary disorders are always genetic [34]. Furthermore, these hereditary diseases are often considered incurable [35].

MODERN ASPECTS

The term "genetics" originates from an Ancient Greek word that signifies 'genitive', 'generative', or 'origin'. A gene is defined as a distinct genomic region regulated by one or more promoters and distal regulatory elements, containing the information necessary for the synthesis of functional proteins or non-coding RNAs. These genes are interconnected through shared genetic information at the level of the final products (proteins or RNAs) [36]. Each gene is located at a specific position on a chromosome.

Genetics is the study of heredity, while genomics refers to the study of genes, their functions, and associated techniques. The key distinction between living and non-living entities lies in the presence of deoxyribonucleic acid (DNA) in living organisms. Among living species, the differences arise from the sequence of bases (adenine–thymine and guanine–cytosine) in the DNA. All nucleated cells contain DNA, and the arrangement of DNA forms genes. It is estimated that humans possess approximately 20,000 genes, with about 400 of these—roughly 2%—being coding genes that actively express themselves; these are known as the genotype. The genotype represents the stable portion of an individual's genetic makeup that determines specific characteristics and typically remains unchanged unless affected by toxic damage.

The genotype is crucial for the development of an individual's phenotype, which encompasses their physical traits, development, and behaviour. Actions taken throughout one's life, whether consciously or unconsciously, can influence the phenotype, and notable changes in expression can affect future generations, potentially resulting in genetic disorders. To enhance understanding, genetic diseases can be classified into four types.

1. Chromosomal abnormality
2. Single gene defect
3. Multifactorial and
4. Mitochondrial.

On the basis of disease manifestation time these can again categorised as

- Presented at the time of birth e.g. Down syndrome
- Act as predisposing factor and disease occur at its time with even little indulgence of causative factor like manifestation of Type 2 DM with positive family history.

Table 1: Common gene proportion in relatives at different degree of relation [37]

Relationship type	Relation to each other	Proportion of gene they have in common
-	Identical twins	100%
First degree relatives	Brothers and sisters, non-identical twins	50%
Second degree relatives	Uncles, Aunts, nephews, nieces and grand parents	25%
Third degree relatives	First cousins, half uncles and aunts, half nephews and nieces	12.5%

Family and twin studies suggest that diabetes has a heritability ranging between 20% and 80%. First-degree relatives of individuals with Type 2 Diabetes Mellitus (T2DM) are about three times more likely to develop the condition compared to those without a family history of the disease [38]. Although a family history of Diabetes on either the maternal or paternal side raises the risk, the Framingham Offspring Study found that children of mothers with Diabetes have a slightly higher likelihood of developing impaired glucose tolerance than those whose fathers have Diabetes [39]. Twin studies on T2DM show a higher concordance rate among monozygotic twins compared to dizygotic twins, highlighting the significant genetic contribution to the disease. In contrast, studies on Type 1 Diabetes (T1DM) reveal that monozygotic twins have a concordance rate of 40%-50% in population-based research [40].

Genetic Factors in T1DM and T2DM [41]

Type 1 Diabetes Mellitus (T1DM):

- T1DM is linked to genetic susceptibility loci, primarily the HLA complex on chromosome 6, which was identified through genetic linkage studies.
- Non-HLA genes like INS, CTLA4, and PTPN22 also contribute to T1DM risk.

Type 2 Diabetes Mellitus (T2DM):

- Genes like TCF7L2, CAPN10, and IRS1 have been identified in T2DM patients through genome-wide association studies (GWAS).
- Variants in PPARG, KCNJ11, and HNF1A are also associated with T2DM.

Role of Genetic Linkage Studies and GWAS:

Linkage analysis and genome-wide association studies (GWAS) have helped identify critical loci and genes associated with diabetes. Over 250 loci related to T2DM and more than 60 loci linked to T1DM have been discovered through GWAS.

Epigenetics and Environmental Influences:

Epigenetics refers to heritable changes in gene function without altering the nucleotide sequence, influenced by environmental factors. Epigenetic changes can be inherited from one cell generation to the next and in some cases, can be inherited through the generations. DNA methylation and histone modifications can affect gene-environment interactions. Intrauterine DNA methylation is of particular interest in the etiology of T2DM [42], as it may contribute to later-life diabetogenic effects. A study by Dabelea et al [42] found increased diabetes and obesity in offspring exposed to intrauterine diabetes compared to siblings born before the mother's diabetes onset.

Polygenic Risk Scores (PRS) [41]:

Polygenic risk scores (PRS) have gained attention for predicting T2DM, though early studies showed genetic data only slightly improved risk prediction when combined with clinical factors like age and family history. Recent advancements in PRS from large, multi-ancestry cohorts have improved predictive power, particularly in identifying pre-diabetes and T2DM in women with a history of gestational diabetes. However, more research is needed to assess the utility of PRS in guiding preventive therapies for reducing future T2DM incidence.

DISCUSSION

The concept of *Beejadusti*, or genetic predisposition, in *Prameha* (Diabetes Mellitus) as outlined in *Ayurveda* aligns remarkably with modern understandings of genetic disorders and hereditary diseases.

Ayurveda attributes diseases such as *Prameha* to defects in the *Beeja* (sperm and ovum), suggesting that abnormalities in these reproductive elements, due to lifestyle and dietary choices of the parents, can lead to a predisposition for disease in offspring. This concept can be compared to genetic mutations or defects in contemporary biomedical terms.

1. *Beeja Dosha* and Modern Genetics: The idea that indulgence in *madhura rasa* (sweet-tasting foods) by parents causes chromosomal damage and leads to diseases like *Prameha*, as mentioned in *Charak Samhita*, is analogous to modern concepts of genetic mutations and hereditary risk factors for Type 1 Diabetes (T1DM). Genetic studies have shown that T1DM has a significant hereditary component, with a higher concordance rate in monozygotic twins (40%-50%) compared to dizygotic twins, indicating a strong genetic basis. Similarly, family and twin studies of Type 2 Diabetes (T2DM) have demonstrated that first-degree relatives are at a significantly higher risk of developing the condition, which mirrors the Ayurvedic understanding that *Beeja dosha* can be passed from parents to offspring.
2. Further supporting this is the identification of specific genetic loci associated with Diabetes, such as HLA loci in T1DM and TCF7L2 in T2DM. These discoveries validate the ancient notion of inherited susceptibilities, though Ayurveda attributes the cause to lifestyle and diet-induced *Beeja* defects rather than spontaneous genetic mutations.
3. Intrauterine Environment and Epigenetics: *Ayurveda* goes beyond genetic predisposition by also emphasizing the influence of the intrauterine environment on disease manifestation. The notion that a mother's dietary habits, particularly the overconsumption of *madhura rasa*, can trigger *prameha* in a fetus predisposed to the disease finds a striking parallel in modern epigenetic research. Studies suggest that intrauterine exposure to diabetes or a poor maternal diet can lead to epigenetic alterations, such as DNA methylation and histone modification, which may predispose offspring to T2DM and obesity.
4. For instance, research has shown that offspring exposed to a diabetic intrauterine environment have a higher risk of developing diabetes later in life. This can be seen in studies where children born to mothers with diabetes are at increased risk for both T1DM and T2DM. The Ayurvedic concept of *Jatah prameha* and its correlation to childhood onset of Diabetes echoes these findings, where early exposure to unwholesome maternal conditions triggers disease development.
5. Postnatal Influences and Early Life Risk Factors: *Ayurveda's* understanding of postnatal influences is also relevant in the context of early-life environmental factors contributing to diabetes risk. The idea that excessive intake of *madhura rasa* during infancy and childhood in genetically predisposed individuals can precipitate *Prameha* aligns with the modern understanding of lifestyle and environmental factors in the development of diabetes. Current research shows that obesity and poor dietary choices in early life can contribute to insulin resistance and beta-cell dysfunction, thereby increasing the risk of developing T2DM in those already genetically predisposed.
6. *Beejadusti* and Polygenic Risk Scores (PRS): Modern genetic tools, such as polygenic risk scores (PRS), offer insights into an individual's genetic susceptibility to diseases like diabetes, much like *beejadusti* offers a framework in Ayurveda for understanding hereditary diseases. PRS combines multiple genetic loci to estimate a person's risk of developing T2DM, highlighting the importance of inherited traits. However, while these scores have improved the accuracy of diabetes risk prediction, especially in multi-ethnic cohorts, they offer only marginal improvement when compared to traditional risk factors like family history, which aligns with the Ayurvedic emphasis on the interplay of genetic and environmental factors.
7. Integration of *Beejadusti* and Modern Science: The critical evaluation of *Beejadusti* in *Prameha* shows that Ayurveda's approach to hereditary diseases is not only holistic but also closely mirrors modern concepts of genetic inheritance and epigenetics. Ayurveda's recognition of both genetic predisposition and environmental triggers, such as diet and lifestyle, provides a more integrative understanding of disease etiology. This is similar to modern medicine's growing recognition of the gene-environment interaction, where both genetic predisposition and external factors contribute to the development of diseases like diabetes.

CONCLUSION

Prameha, described as *Adibala pravritta*, *Kulaj vikara*, and *Sahaj vyadhi*, develops due to *Beejadusti* (genetic or hereditary defects). These *Sahaj vikara* (hereditary/genetic diseases) are traditionally considered incurable. The primary causative factors influencing *Beeja* (sperm and ovum) include unhealthy lifestyle choices, poor dietary habits, advanced parental age, and imbalanced doshas at the time of conception. Similarly, in modern science, genetic and hereditary factors are recognized as key

etiological contributors to Diabetes Mellitus, with epigenetic mechanisms further influencing its development.

According to the foundational Ayurvedic principle of “*swasthasya swasthya rakshanam aturashya vikar prashamanam*” (maintaining the health of the healthy and curing the ill), prevention of *Sahaj prameha* is possible. By incorporating *nidan parivarjana* (avoidance of causative factors), *sharirshodana* (body purification) during reproductive age and preconception, along with *garbhini paricharya* (maternal care), lifestyle modifications, and dietary adjustments in the pre- and postnatal phases, the risk of *Sahaj prameha* can be reduced, leading to the birth of healthier progeny.

REFERENCES

1. Agnivesh, A., Shukla, V., & Tripathi, R. D. (2002). *Charaka Samhita* (Chikitsasthana, Adhyaya 6/8, p. 187). Chaukhamba Sanskrit Pratishthan.
2. Agnivesh, A., Shukla, V., & Tripathi, R. D. (2002). *Charaka Samhita* (Indriyasthana, Adhyaya 9/9, p. 848). Chaukhamba Sanskrit Pratishthan.
3. World Health Organization. (n.d.). *World Health Organization India*. <https://www.whoindia.org/>
4. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Sutrasthana, Adhyaya 11/7, p. 69). Chaukhamba Surbharati Prakashan.
5. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Sutrasthana, Adhyaya 11/8, p. 69). Chaukhamba Surbharati Prakashan.
6. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Sutrasthana, Adhyaya 11/22, p. 71). Chaukhamba Surbharati Prakashan.
7. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Chikitsasthana, Adhyaya 6/57, p. 449). Chaukhamba Surbharati Prakashan.
8. Vagbhata, A., & Gupta, A. (2009). *Ashtanga Hridaya with Vidyotini commentary* (Nidanasthana, Adhyaya 10/38–39, p. 505). Chaukhambha Prakashana.
9. Vagbhata, A., & Gupta, A. (2009). *Ashtanga Hridaya with Vidyotini commentary* (Sharirasthana, Adhyaya 3/77, p. 401). Chaukhambha Prakashana.
10. Agnivesh, A., & Acharya, Y. T. (2013). *Charaka Samhita with Chakrapani Datta commentary* (Pramehanidana Adhyaya, p. 212). Chaukhamba Prakashan.
11. Agnivesh, A., & Acharya, Y. T. (2013). *Charaka Samhita with Chakrapani Datta commentary* (Pramehanidana Adhyaya, p. 213). Chaukhamba Prakashan.
12. Sushruta, A., & Shastri, A. D. (2012). *Sushruta Samhita with Ayurveda Tattva Sandipika Hindi commentary* (Vol. 1, Pramehanidana Adhyaya, p. 326). Chaukhamba Sanskrit Sansthan.
13. Sushruta, A., & Shastri, A. D. (2012). *Sushruta Samhita with Ayurveda Tattva Sandipika Hindi commentary* (Vol. 1, Pramehachikitsa Adhyaya, p. 75). Chaukhamba Sanskrit Sansthan.
14. Chandola, H. M., & Kajaria, D. (2020). Prameha Nidana Adhyaya. In S. K. Khandel, P. Godatwar, Y. S. Deole, & G. Basisht (Eds.), *Charak Samhita: New edition* (1st ed., p. 37). CSRTSDC.
15. Sharma, R. K., & Dash, B. (2007). *Caraka Samhita* (Vol. 2, Sharira Sthana, Chapter 2, Verse 3). Chowkhamba Sanskrit Series Office.
16. Mueller, R. F., & Young, I. D. (1998). *Emery's elements of medical genetics* (10th ed.). Churchill Livingstone.
17. Albishi, A. A., AlAmri, E., & Mahmoud, A. A. (2022). Relationships among consanguinity, family history, and the onset of type 1 diabetes in children from Saudi Arabia. *Primary Care Diabetes*, 16(1), 102–106.
18. Sushruta, A., & Acharya, Y. T. (Eds.). (2012). *Sushruta Samhita* (Sharira Sthana, Chapter 10, Verses 54–55). Chowkhamba Sanskrit Sanstha.
19. Sushruta, A., & Acharya, Y. T. (Eds.). (2012). *Sushruta Samhita* (Sharira Sthana, Chapter 10, Verse 56). Chowkhamba Sanskrit Sanstha.
20. Mueller, R. F., & Young, I. D. (1998). *Emery's elements of medical genetics* (10th ed.). Churchill Livingstone.
21. Bingley, P. J., Douek, I. F., Rogers, C. A., & Gale, E. A. (2000). Influence of maternal age at delivery and birth order on risk of type 1 diabetes in childhood: Prospective population-based family study. *BMJ*, 321(7258), 420–424.
22. Lammi, N., Moltchanova, E., Blomstedt, P., et al. (2007). The effect of birth order and parental age on the risk of type 1 and type 2 diabetes among young adults. *Diabetologia*, 50, 2433–2438. <https://doi.org/10.1007/s00125-007-0843-5>
23. Sharma, R. K., & Dash, B. (2007). *Caraka Samhita* (Vol. 2, Vimana Sthana, Chapter 8, Verse 95). Chowkhamba Sanskrit Series Office.
24. Sharma, H., & Wallace, R. K. (2020). Ayurveda and epigenetics. *Medicina*, 56(12), 687. <https://doi.org/10.3390/medicina56120687>
25. Manohar, P. R. (2016). Medical genetics in classical Ayurvedic texts: A critical review. *Indian Journal of History of Science*, 51, 417–422. <https://doi.org/10.16943/ijhs/2016/v51i2.2/48455>
26. Vaidya, A. D. (2014). Prakruti genomics and prameha-proclivity: Relevance to metabolic syndrome. *Molecular Cytogenetics*, 7(Suppl. 1), 13. <https://doi.org/10.1186/1755-8166-7-S1-13>
27. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Sharirasthana, Adhyaya 3/17, p. 315). Chaukhamba Surbharati Prakashan.

28. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Sharirasthana, Adhyaya 4/4, p. 316). Chaukhamba Surbharati Prakashan.
29. Chandola, H. M., & Kajaria, D. (2020). Prameha Chikitsa Adhyaya. In M. S. Baghel, Y. S. Deole, & G. Basisht (Eds.), *Charak Samhita: New edition* (1st ed., p. 79). CSRTSDC.
30. Shastri, A. D. (2014). *Sushruta Samhita* (Vol. 1, Chikitsa Sthana, Chapter 11, Prameha Chikitsa, p. 75). Chaukhamba Sanskrit Pratishthan.
31. Sushruta, A., & Acharya, Y. T. (2017). *Sushruta Samhita with Dalhana commentary* (Sutrasthana, Adhyaya 24/6, p. 96). Chaukhamba Surbharati Prakashan.
32. Shankar, S., Kumar, D., & Srivastava, R. K. (2013). Epigenetic modifications by dietary phytochemicals: Implications for personalized nutrition. *Pharmacology & Therapeutics*, 138(1), 1–17. <https://doi.org/10.1016/j.pharmthera.2012.11.002>
33. Löwy, I. (2019). How diseases became “genetic.” *Ciência & Saúde Coletiva*, 24(10), 3607–3617. <https://doi.org/10.1590/1413-812320182410.19102019>
34. Agnivesh, A., & Acharya, Y. T. (2017). *Charaka Samhita with Chakrapani Datta commentary* (Chikitsasthana, Adhyaya 6/57, p. 449). Chaukhamba Surbharati Prakashan.
35. Pesole, G. (2008). What is a gene? An updated operational definition. *Gene*, 417(1–2), 1–4. <https://doi.org/10.1016/j.gene.2008.03.010>
36. Prescott, C. A., & Kendler, K. S. (1995). Twin study design. *Alcohol Health & Research World*, 19(3), 200–205.
37. Ali, O. (2013). Genetics of type 2 diabetes. *World Journal of Diabetes*, 4(4), 114–123. <https://doi.org/10.4239/wjd.v4.i4.114>
38. Sun, H., Saeedi, P., Karuranga, S., Pinkepank, M., Ogurtsova, K., Duncan, B. B., Stein, C., Basit, A., Chan, J. C. N., Mbanya, J. C., Pavkov, M. E., Ramachandaran, A., Wild, S. H., James, S., Herman, W. H., Zhang, P., Bommer, C., Kuo, S., Boyko, E. J., & Magliano, D. J. (2022). IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. *Diabetes Research and Clinical Practice*, 183, 109119. <https://doi.org/10.1016/j.diabres.2021.109119>
39. Kyvik, K. O., Green, A., & Beck-Nielsen, H. (1995). Concordance rates of insulin dependent diabetes mellitus: A population-based study of young Danish twins. *BMJ*, 311(7010), 913–917. <https://doi.org/10.1136/bmj.311.7010.913>
40. Goyal, S., Rani, J., Bhat, M. A., & Vanita, V. (2023). Genetics of diabetes. *World Journal of Diabetes*, 14(6), 656–679. <https://doi.org/10.4239/wjd.v14.i6.656>
41. Simmons, R. A. (2007). Developmental origins of beta-cell failure in type 2 diabetes: The role of epigenetic mechanisms. *Pediatric Research*, 61(5, Pt. 2), 64R–67R. <https://doi.org/10.1203 /pdr. 0b013 e3180457623>
42. Dabelea, D., Hanson, R. L., Lindsay, R. S., Pettitt, D. J., Imperatore, G., Gabir, M. M., Roumain, J., Bennett, P. H., & Knowler, W. C. (2000). Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: A study of discordant sibships. *Diabetes*, 49(12), 2208–2211. <https://doi.org/10.2337 /diabetes.49.12.2208>

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