AYUSHDHARA
ISSN: 2393-9583 (P)/ 2393-9591 (O)
An International Journal of Research in AYUSH and Allied Systems

Review Article

EVO-AYU: INTEGRATING AYURVEDA WITH EVOLUTIONARY BIOLOGY

Shakeel H Jamakhane[1]*, Mahantesh R Sajjanashetty[2], B.H. Katti[3], V.V. Goudar[4]
*[1]Post Graduate Scholar, [2]Professor and HOD, [3]Professor, [4]Lecturer, Department of PG Studies in Ayurveda Samhita Siddhanta, Shri Vijay Ayurvedic Medical College and PG Research Centre, Ilkal, Karnataka, India

Article info
Article History:
Received: 02-01-2022
Revised: 28-01-2022
Accepted: 06-02-2022
KEYWORDS:
Natural Selection, Evolution, *Jaraa,* Antagonistic pleiotropy, Genetic conflict.

ABSTRACT

Evolution refers to a change in the relative frequency of alternative alleles in a population or species. Evolutionary biology studies evolution at two levels- micro-evolution and macro-evolution. Micro-evolution studies the processes that change allele frequency over time within a population. Macro-evolution studies the tree of life. Using the concepts from evolutionary biology to understand human health and disease is the basis of evolutionary medicine. Evolutionary medicine has improved our understanding of infectious diseases, genetics, ageing etc. Would an integration of evolutionary ideas with *Ayurveda* be equally rewarding? *Ayurveda* accepts *Saankhya* philosophy's ideas about the origin of life. *Satkaarya Vaada,* which forms the basis of *Ayurvedic* understanding of disease and health, doesn't provide satisfying answers to the cardinal problem of evolutionary biology - the appearance of design in the living world. *Ayurvedic* ideas about inheritance also contradict the germ-plasm theory of inheritance. Yet, *Ayurveda* has much to contribute to the study of phenotypes. *Ayurvedic* concepts of *Deha-prakriti, Saara, Desha* etc. provide exciting opportunities to test evolutionary principles within the *Ayurvedic* framework. Integrating *Ayurveda* with Evolutionary biology could help us rethink *Ayurvedic* concepts in the light of evolution.

INTRODUCTION

Charaka Samhita recognizes that *Shareera* and *Manas* experience disease and health.[1] The susceptibility of *Shareera* and *Manas* to external influences i.e., *Hetu* is considered to be the cause for both disease and health. [1] The status of *Swaasthya* is defined as the state of equilibrium of *Dosha-aadi*. Diseases emerge when *Dosha-aadi* are deranged. [2] Restoring and maintaining the equilibrium of *Dosha-aadi* is the aim of *Ayurveda.*[3]

Ayurveda does well at recognizing the proximate mechanical causes of disease and health. But it leaves the questions of evolutionary significance relatively untouched.

Access this article online
Quick Response Code
https://doi.org/10.47070/ayushdhara.v9i1.901

Questions such as- Why are *Shareera* and *Manas* vulnerable to disease? Why don't we live forever? Why do we age? Why hasn't natural selection weeded out *Tridosha*? Why does *Vata Dosha* produce more *Nanatmaja Vikara*? How do *Dosha-prakiriti* interact? Why do we have eight types of *Saara-purusha*? Why do we not have *Vikaara-vighaata Bhaava* for every disease? Could *Roga* be the cause of *Sukha*? Applying evolutionary thinking to these riddles could help us better understand *Ayurvedic* concepts.

Evolution101

Evolution refers to a change in the relative frequency of alternative alleles in a population or species. The study of the evolution of life involves two big ideas - Microevolution and Macroevolution. While microevolution studies the processes that cause changes in the relative frequency of alternative alleles, macroevolution studies the historical constraints to the evolution of life.

Evolution is the historical occurrence of change; the change happens through many processes. Natural selection is one such process. Evolution by natural selection produces adaptations (adaptive

evolution). This happens whenever three conditions are satisfied [4]:

1. When a trait varies among individuals.
2. Variation in trait affects how many successful offspring an individual has (non-zero correlation between trait and reproductive success).
3. Genes that vary among individuals influence at least some of the variation in the trait (Heritability).

When variation in trait doesn't affect the number of successful offspring an individual has evolution occurs through random drift. Random drift leads to Neutral evolution.

Evolution can be fast or slow. The rate of evolution depends on- the size of the population and the strength of natural selection. When a large population is exposed to a strong selective pressure, evolution by natural selection happens rapidly. E.g., evolution of antibiotic resistance.

Processes like mutations, meiosis (crossing-over and independent assortment of chromosomes) and random mating etc. produce genetic variation. Natural selection operates on this variation. Selection could be - directional, stabilising or disruptive. It could also be frequency dependent. Deleterious alleles experience purifying selection. Beneficial alleles experience positive selection.

Selection chiefly operates at the level of genes, but it also operates at the level of individuals, groups and species (multilevel selection).

A gene's effect are its phenotypes. Genes are difference-makers.[5] One genotype can produce an entire range of phenotypes depending on the environment (reaction norm). Traits are thus a function of both genes and the environment.

Fitness refers to reproductive fitness, not survival. Natural selection optimises individuals for reproductive success (fitness). Natural selection doesn't operate beyond the reproductive age.

Genes with benefits in ancestral environment but costs in present environment experience evolutionary mismatch. Genes that increase fitness early in life but produce detrimental effects later in life are retained by natural selection (antagonistic pleiotropy). Differences in parental investment in offspring leads to parent-offspring conflict.

1. Why are *Shareera* and *Manas* vulnerable to diseases?

Charaka Samhita rightly points out that *Shareera* and *Manas* are vulnerable to diseases as both of them are susceptible to external influences i.e., *Hetu.*[1] Diseases could be of *Nija* origin or *Aagantuja* origin.[6]

Natural selection optimises individuals for reproductive success. Any trait with a reproductive advantage should then get selected. Getting rid of *Nija Vyadhi* could provide substantive reproductive advantage. Why has natural selection then not removed the causes of *Nija Vyadhi*?

The impossibility of eliminating *Nija Vyadhi*

An important insight from Evolutionary Biology is that *Shareera* is a bunch of trade-offs. The possibility of improving a trait is constrained by its effect on other traits. (Imagine why humans don't have wings like angels!) And evolution by natural selection has not left many spaces for simple, trade-off free improvements.

Also, the large-scale structure of an organism is determined by hard-to-change developmental patterns with deep evolutionary history. To put simply, things that change slowly constrain things that change rapidly.

Natural selection optimises organisms for reproductive fitness. That means - Evolution doesn't care if the organism suffers from a disease - if the gene that causes the disease somehow improves the lifetime reproductive success of the individual (the number of successful offspring that individual has).

So, natural selection works to eliminate *Nija Vyadhi* only to the extent that the benefits of weeding out the cause of the disease (let's say, the *Rookshata* of *Vata Dosha*) outweigh the costs associated with eliminating that trait. It seems that the *Rookshata* of *Vata Dosha* has benefits that outweigh the costs associated with it.

Natural selection replaces the rosy view of perfection in nature with a realistic view - life is a bunch of compromises.

What would happen if *Nija Vyadhi* (intrinsic mortality) were completely eliminated?

Weeding out all the imperfections of *Shareera* and *Manas* would eliminate *Nija Vyadhi*. But that doesn't eliminate the possibility of death. The more we live, the more we are exposed to *Nidaana* for *Aagantuja Vyadhi*. That means, the probability of dying by accidental causes increases as we live longer lives.

At some age, when the cumulative exposure for accidental causes (*Aadi-bhautika Dukha*) increases the chances of death (extrinsic mortality), there is no reason to invest more into eliminating the causes of *Nija Vyadhi*. Instead, it makes sense to use the available resources to improve the reproductive fitness even if doing so leads to *Nija Vyadhi, Jara* and *Mrityu*.

Also, as death from extrinsic causes becomes more and more inevitable the strength of selection becomes weaker (because increased death rate decreases the population size at that age. Strength of

selection is directly proportional to the size of population). This further decreases the impact of natural selection on eliminating the *Nija Vyadhi*.

The insight here is – the unavoidable exposure to *Aadi-bhautika Nidaana* makes the *Shareera* susceptible to *Aadhyaatmika Vyadhi*.

This is also the (partial) reason for why we don't live forever, and why do we age.

2. Why Do We Age?

The standard *Ayurvedic* explanation for *Jaraa* is that it is a *Svaabhaavika Vyadhi*.[7] Which means that *Jaraa* is an unavoidable disease the cause of which is unascertained. Calling *Jaraa* as the effect of *Kaala*, or *Svabhaavaja* doesn't take us too far at understanding or curing it.

Why do we age? Aging is the outcome of senescence. Senescence, as a process, is similar to *Sheerana,* the gradual decline of *Shareera*. Our organ systems all seem to wear out at about the same rate, on average. Senescence makes us steadily more vulnerable to diseases like cancer, stroke, infections, accidents, auto-immune diseases etc.

This begs the question – why would natural selection not eliminate the causes of *Jaraa*?

Antagonistic Pleiotropy

When a gene has more than one effect, it is called as pleiotropic.

In a 1957 paper, George Williams came up with the pleiotropic theory of senescence.[8] According to this theory, aging is the by-product of selection for reproductive performance. That is, genes that improve performance early in life (and thereby increase reproductive fitness) at the cost of detrimental effects later in life - get selected. There is a trade-off between survival and reproduction. His reasoning is[9]:

"Imagine that there is a gene that changes calcium metabolism so that bone heals faster, but the same gene also causes slow and steady calcium deposition in the arteries. Such a gene might well be selected for, because many individuals benefit from its advantages in youth, while few will live long enough to experience the disadvantages of arterial disease in old age. Even if the gene caused everyone to die by age 100, it would still spread if it offered even minor benefits in youth."

Disposable soma theory: Natural selection favours reproduction over survival

Disposable soma theory generalises Williams's theory of senescence. According to this theory, organisms possess finite resource budget which is to be divided between growth, somatic maintenance and reproduction. Natural selection favours reproduction over survival.

These observations are important to answer the questions related to *Dosha*. *Dosha* are characterised by their ability to cause *Shareera Dooshana*. Why would natural selection not get rid of *Dosha*?

3. Why hasn't natural selection weeded out the *Tridosha*?

Dosha are considered to be the primary causes of *Nija Vyadhi*. They are recognised for their ability to cause disease. *Dosha* cause two types of diseases – *Saamanyaja Vyadhi* and *Naanatmaja Vyadhi*.

Evolutionary Biology posits that each trait comes with its own costs and benefits. The costs are of two types:

1. Direct costs
2. Vulnerabilities

Direct costs of a trait are the costs of the maintenance for that trait. These are difficult to avoid as they are inherent to the trait. Vulnerabilities are rare, negative effects of a trait incurred by a fraction of the population. Natural selection tries to reduce the absolute value of direct costs and reduce the frequency of vulnerabilities.

An example for direct costs and vulnerabilities is the synovial joints. The manoeuvrability of synovial joints makes them susceptible to erosion of cartilage, bone friction and damage. Hence Osteo-arthritis is a direct cost of the trait synovial joint. Synovial joints are encased in synovial membrane. Unlike soft tissue, the inflammatory exudation in a synovial joint cannot spread out. This makes the synovial joints susceptible to rheumatoid arthritis. Rheumatoid arthritis is the vulnerability associated with the synovial joint.

Naanaatmaja Vikaara are the direct costs of each of the *Tridosha*. And *Saamaanyaja Vikaara* are the vulnerabilities associated with the *Dosha*.

It is clear that the costs associated with *Tridosha* are quite high. Why has natural selection retained such costly traits?

Natural selection has tried to reduce the costs associated with *Tridosha.* The physiological range of variations in *Dosha* don't cause diseases. *Dosha* keep undulating within the physiological range. Only when the *Dosha* are deranged to an extent that they have reached *Prasara* and *Sthaana-samshraya Avastha* do they cause disease.

Dosha are retained by natural selection because they play an important role in development of organisms. *Dosha* decide the *Prakriti* of the individual early in life. They also help maintain physiological processes of the body. The benefits associated with *Tridosha* far outweigh the costs associated with them.

Tridosha are high-cost high-benefit traits of the *Shareera*.

4. The 80:40:20 Ratio: Why does *Vata Dosha* produce more *Nanatmaja Vikara*?

There are at least 80 direct costs (*Nanatmaja Vikara*) mentioned for *Vata Dosha* compared with 40 for *pitta* and 20 for *Kapha*.[10] Why does *Vata Dosha* causes many *Nanatmaja Vikara*?

The answer to this question lies in the physiological variation of *Dosha* with age. *Kapha Dosha* peaks in the *Baalya-avastha*. *Pitta Dosha* peaks in the *Yauvana-avastha*. *Vata Dosha* peaks in *Vriddha-avastha*.[11]

Things that happen beyond the reproductive age are of no significance for evolution. Strength of selection decreases with age. Natural selection operates until the end of the reproductive age.

Vata Dosha starts to peak only after the end of the reproductive age. Most of the *Vataja Nanatmaja Vikara* occur during the old age. That means, the direct costs of increased *Vata Dosha* have not been eliminated by natural selection. Of the *Tridosha*, direct costs of *Vata* are the least regulated by natural selection.

Direct costs of *Kapha* and *Pitta* affect the reproductive fitness of individual because *Kapha* and *Pitta* peak early in life. Evolution has weeded out most of the direct costs of *Kapha* and *Pitta* through purifying selection. *Kapha* is associated with the least direct costs. *Pitta* has moderate costs associated with it because *pitta* peaks during *Yauvana-avastha* when the *Kaala-krita Bala* is the highest. So, direct costs of *Pitta* do not produce as much a disadvantage as do the direct costs associated with *Kapha*.

The antagonistic pleiotropic effects of *Vata Dosha* make it the worst offender of the *Tridosha*.

4. How do *Dosha Prakriti* interact?

Dosha-Desha Sambandha

Dosha Prakriti are characterised by a range of phenotypes. That is, individual *Prakriti* is a mosaic of traits. The traits constituting a *Prakriti* vary within a physiological range. (E.g., not all *Kapha Prakriti* individuals are equally fat)

Reaction norms and phenotypic plasticity:

Reaction norm is a property of the genotype. It is the response of development to the changing environment. That is, a genotype can produce more than one phenotype as a response to the changing environment.

The sensitivity of the phenotype to the changes in the environment is referred to as phenotypic plasticity. The plasticity of a phenotype is limited by developmental constraints. Old, hard-to-change developmental patterns are less plastic than new phenotypes.

When plotted on a graph (trait vs environment) the slope of a reaction norm indicates the plasticity of the phenotype.

Reaction norms, phenotypic plasticity and the *Dosha Prakriti*

The phenotypes associated with each *Prakriti* produce reaction norms in response to the changing environment. That is, a *Kapha Prakriti Purusha* exhibits phenotypes of *Kapha Prakriti* as a function of changing environment.

The phenotypes which are most plastic (sensitive to the changes in the environment) vary substantially in response to the changing environment. Trying to plot the reaction norm for the phenotypes of each *Prakriti* and identifying the phenotypes that are most sensitive to the changing environment will help us identify the ways in which *Vata-aadi Prakriti* adapt to the changing environment.

Evolutionary mismatch (Mismatch to modernity) and *Dosha Prakriti*

Evolutionary mismatch refers to a type maladaptation. When a gene is beneficial in one environment but produces negative consequences (costs) in different environment it produces phenotypes which are maladapted to the latter environment. This happens because adaptation takes time. Evolution is slow for a sexually reproducing diploid with a long generation time (e.g., Homo sapiens).

Allergies and autoimmune disorders are examples for mismatch to modernity. Autoimmune diseases affect developed countries disproportionately more than they affect the developing countries. This happens because of the lack of exposure to organisms that were part of human evolutionary history - leading to disordered immune regulation. In a sense, allergies and autoimmune disorders are indicators of demographic transition. People migrating from low-incidence countries to high incidence countries acquire immune disorders with a high incidence at the first generation.[12]

***Dosha-Desha Sambandha* and evolutionary mismatch:**

Dosha Prakriti are specifically adapted to *Desha*. *Vata Prakriti* is best adapted to *Jaangala Desha, Pitta Prakriti* is adaptive to *Saadhaarana Desha* and *Kapha Prakriti* is best adapted to *Aanoopa desha. Prakriti* are optimised for *Desha.*

An evolutionary mismatch perspective would predict that *Kapha Prakriti Purusha* migrating from *Aanoopa Desha* to *Jaangala Desha* would be more susceptible to auto-immune disorders than the other

two *Prakriti*. Also, *Vata-prakriti Purusha* migrating from *Jaangala Desha* to *Aanoopa Desha* would be more prone to infectious diseases than the other two *Prakriti*. This could be the reason for rarity of *Eka-dosha Prakriti. Dwandwa Prakriti* limit the negative consequences of evolutionary mismatch.

Genetic conflict and *Dosha Prakriti:*

When the incentives of genes are not aligned it results in genetic conflict. Genetic conflict could arise when gene transmission is asymmetrical (e.g., conflict between cytoplasmic and nuclear genomes). Genetic conflict also ensues when selection at a lower level reduces fitness at higher level (e.g., gynodioecy in plants).

Genetic conflicts also occur between parents, and parent and offspring.

Genetic conflict between parents occurs because reproductive strategies of male and female are different. Male can father several children while female bears each child. Both parents use genetic imprinting to influence foetal development. Genetic imprinting occurs during gametogenesis. Imprinted genes are not expressed in the foetus. Father turns off genes that down-regulate growth. Mother turns off genes that up-regulate growth. That is, father tries to extract more than the mother is prepared to give.

Parent and offspring conflict results because the foetus is selected to extract more from the mother than the mother is ready to provide. This is because mother shares only 50% of her genes with the foetus. She would conserve her body to reproduce more. Foetus, on the other hand, shares only 50% of genes with (future) siblings. Getting more nutrients would benefit all of his 100% genes now. The result - foetal tissue secrets hormones that manipulate mother's metabolism. The symptoms of this conflict are high blood pressure and diabetes in mother.

***Dosha Prakriti* and *Prakriti-Prakriti* conflict**

Similar conflicts could occur between parent and offspring of different *Dosha Prakriti*.

The conflict occurs when the mother is of *Vata Prakriti* and the foetus is of *Kapha* (or *Pitta*) *Prakriti.* The reasoning is - if the mother is of *Vata Prakriti*, her body's energy reserves are quite limited. As the foetus is of *Kapha prakriti*, the energy demands of the foetus are higher than what the mother is ready to give. This makes foetus put in extra effort to acquire nutrients by - increasing the blood pressure, increasing the amount of nutrients (glucose) available in the blood. The mother becomes susceptible to pre-eclampsia, eclampsia and gestational diabetes.

Thus, when *Vata Prakriti* mother bears *Kapha Prakriti* foetus, she would be susceptible to pre-eclampsia, eclampsia and gestational diabetes.

Thrifty phenotypes and obesity in *Kapha Prakiti purusha*

Thrifty phenotypes result when early life events fail to predict the late life environments. The idea is – nutritional stress early in life switches one's physiology to being good at conserving energy, but it is unsuitable if there is adequate food. Thrifty phenotypes make people susceptible to obesity, cardiovascular diseases etc.

When *Vata Prakriti* mother bears *Kapha Prakriti* foetus, the foetus experiences nutritional deficit early in life. This makes such *Kapha Prakriti* individuals susceptible to obesity, cardiovascular diseases etc. later in life.

5. Why are there eight types of *Saara Purusha?*

Natural selection optimises individuals for reproductive success. Which means that, given some time *Shukra Saara purusha* would replace all the other *saara purusha* in the population.

But this hasn't happened. Why?

The reason for the existence of *Rasa-aadi Saara Purusha* is that each of the *Saara Purusha* provides reproductive advantage in certain environments.

1. *Rasa Saara Purusha* would not be bothered by the tropical climate.
2. *Rakta Saara Purusha* would possess reproductive advantage in high altitudes (high-altitude adaptation).
3. *Mamsa Saara Purusha* would be more agile than others.
4. A *Meda Saara Purusha* would store more energy and thereby improve his reproductive fitness. (Remember that throughout human evolutionary history food was scarce resource.)
5. *Asthi Saara Purusha* would recover from bone injuries faster than others.
6. A *Majja Saara purusha* would have stronger immunity to evade the threat of infectious diseases.
7. A *Satwa Saara purusha* would lead the tribe.

Saara Purusha interact with the environment. Certain environments favour specific *Saara Purusha*. Therefore, *Shukra Saara Purusha* is not the only types of *Saara Purusha*.

***Meda Saara Purusha* as an example for mismatch to modernity**

Throughout the human evolutionary history *Meda Saara Purusha* possessed great reproductive advantage over other *Saara Purusha.* This is because food was a scarce resource.

But modern lifestyle allows people to experience excess energy intake while reducing the physical labour. *Meda Saara Purusha* are susceptible to

this energy-intake and energy-expenditure mismatch. This makes *Meda Saara Purusha* susceptible to obesity, diabetes and cardiovascular diseases etc.

Similarly, antagonistic pleiotropic effects of each *Saara Purusha* may make them susceptible to specific diseases during the old age. E.g., *Asthi Saara purusha* benefits from fast healing bones during the reproductive age (hence get selected) but suffer from calcium deposition in arteries leading to atherosclerosis later in life.

6. Why do we not have *Vikaara-vighaata Bhaava* for every disease?

Vikaara-vighaata Bhaava refers to the factors that suppress the processes of disease emergence and progression.

Three factors are identified as causes of diseases - *Nidaana, Dosha* and *Dooshya.* When these factors are congruent, *Vyadhis* occur. The degree of congruence between the *Vyadhi Utpaadakara Bhaava* (*Nidaana-aadi*) depends on three elements- *Paraspara Anubandha, Kala* and *Bala*. The presence and absence of *Vikaara-vighaata Bhaava* affects the degree of congruence between *Nidaana-aadi*.

Vikaara utpaadakara bhaava and *vikaara vighaata bhaava* could interact in the following manner:

Table 1: Interactions between *Vikaara-Vighaata Bhaava* and *Vikaara Utpaadakara Bhaava* (*anubandha*)

		Vikaara-Vighaata Bhaava		
		Alpa	***Madhyama***	***Uttama***
***Vikaara-utpaadkara bhaava* (*Anubandha*)**	*Anubandha*	Severe disease	Mild disease	No disease
	Ananubandha	No disease	No disease	No disease

Table 2: Interactions between *Vikaara-Vighaata Bhaava* and *Vikaara Utpaadakara Bhaava* (*Kaala*)

		Vikaara-Vighaata Bhaava		
		Alpa	***Madhyama***	***Uttama***
***Vikaara-utpaadkara bhaava* (*Kala*)**	*Kala-prakarsha*	Chronic disease	No or mild disease	No disease
	Sheeghra anubandha	Acute disease	Mild disease	No or mild disease

Table 3: Interactions between *Vikaara-Vighaata Bhaava* and *Vikaara Utpaadakara Bhaava* (*Bala*)

		Vikaara-Vighaata Bhaava		
		Alpa	***Madhyama***	***Uttama***
***Vikaara-utpaadkara bhaava* (*Bala*)**	*Abaliyamso Anubandha*	Mild disease	No or mild disease	No disease
	Baliyamso anubandha	Severe disease	Mild disease	No or mild disease

Based on the aforementioned interactions, the question should be - Why do we not possess *Uttama Vikaara-vighaata bhaava* for every disease?

***Vikaara Vighaata Bhaava* and the evolution of life-histories**

Life-history traits are the traits that are directly associated with reproduction and survival. (e.g., number of offspring, lifespan, growth rate etc.)

Life-history traits experience evolutionary trade-offs. Trade-off refers to any evolutionary change that increases fitness in a trait or context but causes decrease in fitness in other trait or context.

Vikaara-vighaata Bhaava are costly traits. Spending resources on *Vikaara-vighaata Bhaava* impacts other life-history traits (e.g., growth rate, size at maturity etc.). Therefore, evolution of *Vikaara-vighaata Bhaava* is context-dependent. For example, when extrinsic mortality (*Aadi-bhautika Vyadhi*) is high, it makes sense to invest most resources into reproduction.

Natural selection optimises individuals for reproductive success. If suffering from a minor illness doesn't impact reproduction, evolution acts slowly. Evolution doesn't care about *Uttama Vikaara-vighaata Bhaava* for every disease. *Uttama Vikaara-vighaata Bhaava* for a disease evolves as a response to selection. The rate of evolution depends on the size of the population and the strength of selection.

Sukha Saadhya Vyaadhi are either minor illnesses or diseases to which we have evolved *Uttama Vikaara-vighaata Bhaava*.

Vikaara-vighaata Bhaava* as *Vikaara Utpaadakara Bhaava

Like any other trait *Vikaara-vighaata Bhaava* have two types of costs- direct costs and vulnerabilities.

Some *Vikaara-vighaata Bhaava* are beneficial in one context but cause disease in other context. That is, a *Vikaara-vighaata Bhaava* could also be *Vikaara Utpaadakara Bhaava*.

For example, sickle-cell anaemia is a disease caused by a gene that is also useful. A person who is heterozygous for this gene is protected from malaria (the gene changes the structure of haemoglobin in a way that speeds the removal of infected cells from circulation). Homozygotes, however, get sickle-cell disease. Sickle-cell disease is an example for heterozygote advantage. In the absence of malaria, the sickle-cell gene loses its status as *Vikaara-vighaata Bhaava* and soon undergoes negative selection.

Some *Vikaara-vighaata Bhaava* have costs and benefits at different stages of the life cycle. This is the principle behind the antagonistic pleiotropy theory. According to the theory, *Vikaara-vighaata Bhaava* early in life becomes *Vikaara Utpaadakara Bhaava* later in life.

For example, a gene named P53 increases the odds of reproduction early in life but deleterious effects later in life (It helps the cancer cells to grow and spread in the body).[13]

Another gene named DR3 decreases the chances of miscarriage, therefore gets selected. But it is also known to cause childhood-onset diabetes.

Vikaara-vighaata Bhaava that were useful in ancestral environment, but cause diseases in the present environment (mismatched to modernity).

For example, high prevalence of Tay-Sachs disease in Ashkenazic Jews is because heterozygotes for the Tay-Sachs gene provided protection against Tuberculosis (historically a major selective force in Ashkenazic Jews).

Gender as a *Vikaara-vighaata Bhaava*

Even gender could be a *Vikaara-vighaata Bhaava*. Females for haemophilia never suffer from the disease. Here, the X chromosome acts as a *Vikaara-vighaata Bhaava*.

7. Could *Roga* be a cause for *Sukha?*

Ayurveda provides details about *Nidaanaarthakara Roga* but doesn't explore cases where a *Roga* protects the person from other severe *Roga*.

Sickle-cell anaemia, Tay-Sachs disease, G6PD deficiency are some of the examples for *Roga* protecting the individual from more severe *Roga*. It has also been showed that worm-infestation early in life reduces the chances of autoimmune diseases later in life. This means, *Roga* could be a cause of *Sukha*. *Roga* could be an evolved response for another severe disease.

CONCLUSION

Integrating *Ayurveda* with Evolutionary Biology could have far-reaching consequences. An evolutionary perspective on *Ayurveda* predicts that *Aayu* evolves (among other reasons) as a response to *Aadi-bhautika Dukha; Jaraa* could result from the antagonistic pleiotropic effects of *Dosha-aadi; Tridosha* are retained by natural selection because of their impact on development; *Naanaatmaja Vikaara* are direct costs of *Tridosha;* interactions between *Prakriti* and *Desha* could produce *Vyadhi; Prakriti-Prakriti* conflict between the mother and foetus could lead to pre-eclampsia, gestational diabetes (in mother), and obesity and CVDs (in child); each of the eight *Saara Purusha* provides reproductive advantage in specific environments; *Vikaara-vighaata Bhaava* is a costly trait, sometimes *Vikaara-vighaata Bhaava* could produce *Vyadhi*; *Roga* could also be the cause for *Sukha*.

REFERENCES

1. Trikamji J, editor. Commentary Ayurveda Dipika of Chakrapanidutta on Charaka Samhita of Charaka, Sootra Sthana; Deerghanjeeviteeya Ahyaaya. Reprint ed., Ch.1. Verse 55. Varanasi: Chaukhambha Sanskrit Sansthan; 2017. p. 15.
2. Trikamji J, editor. Commentary Ayurveda Dipika of Chakrapanidutta on Charaka Samhita of Charaka, Sootra Sthana; Khuddakachatushpaada Ahyaaya. Reprint ed., Ch. 9. Verse 4. Varanasi: Chaukhambha Sanskrit Sansthan; 2017. p. 62
3. Trikamji J, editor. Commentary Ayurveda Dipika of Chakrapanidutta on Charaka Samhita of Charaka, Sootra Sthana; Deerghanjeeviteeya Ahyaaya. Reprint ed., Ch.1. Verse 53. Varanasi: Chaukhambha Sanskrit Sansthan; 2017. p. 14.
4. Lewontin, R. C. The Units of Selection. Annual Review of Ecology and Systematics, vol. 1, Annual Reviews, 1970, pp. 1–18, http://www.jstor.org/stable/2096764.
5. Krimsky S, Gruber J. Genetic Explanations: Sense and Nonsense. Cambridge, MA and London, England: Harvard University Press; 2013. https://doi.org/10.4159/harvard.9780674067769
6. Sreekumar T. Editor. Astanga Hrdaya of Vagbhata Sūtrasthana-1, Sootra Sthana; Ayuskamīyam Ahyaya, 4th ed., Ch. 01. Verse 20. Thrissur: Publication Department Harishree Hospital; 2013. P. 47.
7. Trikamji J, Ram N, editors. Susruta Samhita of Susruta, Sootra Sthana; Vyadhisamuddesheeyam Adhyaaya. Reprint ed., Ch. 24. Verse 7. Varanasi: Chaukhambha Sanskrit Sansthan; 2015. p. 114.
8. Williams, George C. Pleiotropy, Natural Selection, and the Evolution of Senescence. Evolution, vol. 11,

no. 4, [Society for the Study of Evolution, Wiley], 1957, pp. 398–411, https://doi.org/10.2307/2406060.

9. Nesse, Randolph M, and George C. Williams. Why We Get Sick: The New Science of Darwinian Medicine. New York: Times Books, 1994.
10. Trikamji J, editor. Commentary Ayurveda Dipika of Chakrapanidutta on Charaka Samhita of Charaka, Sootra Sthana; Maharoga Ahyaaya. Reprint ed., Ch. 20. Verse 10. Varanasi: Chaukhambha Sanskrit Sansthan; 2017. p. 113.
11. Sreekumar T. Editor. Astanga Hrdaya of Vagbhata Sūtrasthana-1, Sootra Sthana; Ayuskamīyam Ahyaya, 4th ed., Ch.01.Verse 8. Thrissur: Publication Department Harishree Hospital; 2013. P. 28.
12. H Okada, C Kuhn, H Feillet, J-F Bach, The 'hygiene hypothesis' for autoimmune and allergic diseases: an update, Clinical and Experimental Immunology, Volume 160, Issue 1, April 2010, Pages 1–9, https://doi.org/10.1111/j.1365-2249.2010.04139.x
13. Erica Ungewitter, Heidi Scrable, Antagonistic pleiotropy and p53, Mechanisms of Ageing and Development, Volume 130, Issues 1-2, 2009, Pages 10-17.https://doi.org/10.1016/j.mad.2008.06.002.

Cite this article as:
Shakeel H Jamakhane, Mahantesh R Sajjanashetty, B.H. Katti, V.V. Goudar. Evo-Ayu: Integrating Ayurveda with Evolutionary Biology. AYUSHDHARA, 2022;9(1):80-87.
https://doi.org/10.47070/ayushdhara.v9i1.901
Source of support: Nil, Conflict of interest: None Declared

***Address for correspondence**
Dr Shakeel H Jamakhane
Post Graduate Scholar,
Department of P.G. Studies in Samhita & Siddhanta,
Sri Vijay Mahantesh Ayurvedic Medical College and PG Research Centre, Ilkal, Karnataka, India.
Email: shakishu1996@gmail.com
Phone: 8073812693