



The Ecological Risk Index and Environmental Health

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Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

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ABSTRACT

Background: The correlation between health and the environment has been acknowledged since ancient times. Values of environmental parameters situated beyond the ecological range produce vulnerabilities and latent pathological mechanisms or generate other reactions. This phenomenon can be called ecological pathogeny.

Objective: The aims of this paper are to prove the existence of the abovementioned phenomenon logically and intuitively using a graphical approach and to define a method for calculating the potential pathological risk index for a given person in non-ecological environments.

Methods: The method consists of a theoretical analysis followed by a synthesis, beginning with the history of medical concepts and mathematical statistics.

Results and Conclusion: a) Given the relationship between environmental parameters situated beyond the ecological range and several pathological states there is a need to introduce the notions of ecological pathogeny and pathology; b) It is possible to find a graphical pattern that suggests the origin of ecological pathology; c) We can quantify the ecological risk index in non-ecological conditions; and d) The concept of ecological pathology involves the cooperation of every academic specialty as well as social and political courts concerned with the human-environment interactions, including nourishment.

Keywords: Ecological pathogeny; public health; graphic pattern; ecological risk index.

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1. INTRODUCTION AND AIMS

It is the nature of general thinking, especially philosophy, to embody terms of maximum generality and the sciences that apply these terms in specific situations. Medicine is among these sciences, suggesting that “ever since the Middle Ages, doctors will be given the title of philosopher-doctor” [1]. The present title of Ph.D. awarded to scientists in different fields “certifies the tight connection between philosophy (read theorising) and those particular sciences” [1].

Approximately 2300 years ago, Aristotle observed that “it is the things we use the most and most frequently for our bodies that contribute greatly to our health; and the action of water and air is of such nature” [2]. Nourishment can be included in this category, as well [3,4].

Human health is a relationship between the human individual and the environment in which the individual lives (i.e. the ecological system). Because human health is a relationship, a change in one of the underlying parameters produces a change in the result. The change of the human ecological environment thus produces a change in human health.

Hans Selye, cited by Kopin, states that environmental factors influence the dynamic balance of life and introduces the term “diseases of adaptation” [5]. By placing pressure on this balance through recurring impacts, environmental factors can develop pathogenic mechanisms, as demonstrated by psychosomatics and by the study of stress. The operation of these factors can also uncover pathogenic vulnerabilities.

One must rationally distinguish between four factors, each playing a different role: a) Pathogenetic vulnerabilities; b) Risk factors for phenotypic expression; c) Onset factors; d) Maintenance factors.

According to W. Osler: “...all scientific truth is conditioned by the state of knowledge at the time of its announcement” [6].

The aims of this paper are to obtain a theoretical proof of the existence of pathogenic relations regarding the systemic relationship of man with the non-ecological environment and to search for a method to calculate a potential pathological risk

index in non-ecological environments. The ecological environment is defined as the set of all parameters of a human being's environment that have values within the limits of human adaptation. The non-ecological or patho-ecological environment is defined as a deviation of one or more environmental factors from the range of values to which humans are adapted through phylogenetic and ontogenetic development. For example, temperatures of -80 degrees Celsius or +60 degrees Celsius are clearly beyond the adaptive range of any human being. A methodology to quantify the relative risk (RR) in punctual, non-ecological conditions has been developed so far [7]. A demonstration that there is also a possibility to quantifiably evaluate a potential pathological risk for a given person in a multiparametrical non-ecological environment is meant.

2. METHODS

The first measured personal observations were those concerning the impact of non-ecological nutrition on the incidence and prevalence of Alzheimer's disease in a given population. This data has been published [8]. Going through scientific literature, measured medical observations of other non-ecological environmental factors and their impact on human health have been found [3,9-11]. Empirical medical observations need a notional conceptualization in order to be considered scientific and must be articulated into a theoretical system that is compatible with the medical paradigm in its unity. In consequence, starting from the conclusive ideas of the papers mentioned above, proceeding in a logical, prospective manner, a more general form for these conclusions was synthetically given, a general form of the relationship between man and environment and finally, between health and disease. These conclusions, one step more general than the empirical observations' conclusions, have allowed their organization in a relations system that could be expressed through a diagram. Further, these observations were addressed in the primary way, as Pascal and Fermat did in the XVII century. The two analyzed the winning chances in gambling, setting the foundations of mathematical statistics. Thus, a mathematical approach of the general relationship environment-man, from the health point of view was found to be possible. This is a path that differs from previous approaches and it needs to be developed.

3. RESULTS AND DISCUSSION

The complex systemic relationship between man and the environment can be expressed in terms of the following theoretical pattern. The arrows indicate the direction of influence.

The following observations apply to the consideration of the opposition between health and disease.

1. The components of the relationship are man and his environment.
2. The environment can be: a) Ecological; according to the definition above, this environment is suitable for the optimal functioning of all somatic and psychic human parameters; or b) Non-ecological; according to the definition above, this environment is overwhelming, stressful or exceeds a human's adaptive capacity.
3. The non-ecological environment is situated beyond the ecological range in varied degrees. For example, it can be monoparametric (e.g., extremely high luminosity) (denoted here as bp1), biparametric (e.g., extremely high luminosity and extremely loud noise) (denoted here as bp2), or triparametric (denoted here as bp3). In this notation, p can have any value from 1 to n. Therefore, any patho-ecological transgression can be denoted as bp1, bp2, bp3,....., bpn.
4. Man as a system is the result of his genetic background and ontogenetic conditions, including all conditions that do or do not facilitate the expression of the genetic background.
5. The environment acts on ontogenetic phenomena. In turn, these phenomena can choose or partially affect the environment of the organism.
6. The environment influences the organism by facilitating or inhibiting the phenotypic expression of the genetic background.
7. The genetic background directly influences ontogeny. In turn, ontogeny produces feedback that influences the expression of the genetic background.
8. Through ontogeny, the genetic background indirectly influences the trajectory of the phenotype in its environments.
9. The potential of humans to function depends directly on these interrelationships.
10. The final result of the function of the environmentally integrated phenotype is actually the state of health or illness.

These ten assertions are proven findings of the biological and medical sciences, whether they are proven simultaneously or separately. One knows that genetic vulnerabilities exist, that they may occur separately or together and that they are clinically (pathologically) expressed through synthetic summation regardless of the ontogenetic conditions or environmental pressure [12]. The term "genetic background with critical mass" will be used to refer to this genetic background. It is also known that certain genetic vulnerabilities cannot be expressed clinically in the absence of pressure from ontogeny and the environment. These vulnerabilities are expressed clinically as diseases only in the case of metabolic or cellular damage. The term "genetic background without critical mass" will be used to refer to this genetic background. The same type of distinction applies to all vulnerabilities that are potentially pathogenic. One example would be the low epileptogenic potential generated by a minor to medium cranial trauma occurring during childhood. This potential may not exhibit epileptic expressions during life or may be expressed clinically only if another factor having a negative impact on the brain (e.g. alcohol) is ontogenetically added. This example illustrates the concept of vulnerability without critical mass. However, if the trauma is major, regardless of the ecological life conditions, the person experiences epileptic seizures. In this case, the vulnerability occurs at the critical mass level.

The same principles apply to pathogenic mechanisms, which are functional expressions of the vulnerabilities from which the mechanisms are derived. These mechanisms constitute the actual disease and its accompanying symptoms. In life, nothing can be understood, including diseases, unless a continuous transformation is considered. Death is the only stationary state.

The mechanisms of diabetes mellitus will be used to illustrate the concept of a pathogenic mechanism. Through the progressive accumulation of chemical compounds, the vulnerabilities are expressed in the blood and transformed into mechanisms. If the diet is adequate to control glycemia, then pathogenic mechanisms without critical mass are involved. If oral medication alone is needed to maintain glycemia within normal limits, then pathogenic mechanisms with a light critical mass are involved. If insulin addiction is present, then a mechanism with a heavy critical mass is present.

These notions are not sufficiently exact to be represented digitally. They are, however,

fundamental. Accordingly, they give meaning to a very large class of applicable notions. They are not meant to enhance knowledge. Rather, they enhance the understanding of what is already known. This characteristic allows us to exercise a complete freedom of association.

Each entity associated with the relationships in Fig. 1 may occur in several states that can be quantified (taking the model of the initiators of probability) as follows [13]:

A1. Environment	
A1.a. Ecological	0 points
A1.b. Non-ecological (b.p.1, b.p.2,..., b.p.n)	1 point
B1. Genetic basis	
B1.a. without any genetic vulnerabilities	0 points
B1.b. with genetic vulnerabilities but without critical mass	0.5 points
B1.c. with genetic vulnerabilities and with critical mass	1 point
B2. Ontogeny	
B2.a. harmonious	0 points
B2.b. with minor developmental accidents, without critical mass	0.5 points
B2.c. with significant developmental accidents, with critical mass	1 point
B3. Acquired vulnerabilities	
B3.a. without acquired vulnerabilities	0 points
B3.b. with acquired vulnerabilities, without critical mass	0.5 points
B3.c. with acquired vulnerabilities, with critical mass	1 point
B4. Pathogenic mechanisms	
B4.a. without pathogenic mechanisms	0 points
B4.b. with pathogenic mechanisms, without critical mass	0.5 points
B4.c. with pathogenic mechanisms, with critical mass	1 points

The non-ecological environment can:

- a) Trigger pathological mechanisms without critical mass that, once initiated, may induce mechanisms with critical mass
- b) Create other vulnerabilities
- c) Involve pathogenic mechanisms, maintain or accelerate them

According to the notation proposed for the ecological environment, the risk of contracting a disease ranges between 0 and 4 points. Four possible factors are involved: Genetic factors, ontogenetic factors, acquired vulnerabilities and pathogenic mechanisms. In contrast, the risk of disease for the non-ecological environment ranges between 1 and 5 points. All 5 factors are affected: The environment, genetic factors, ontogenetic factors, acquired vulnerabilities and pathogenic mechanisms. These simple regularities allow the definition of a specific level for each person. Each person therefore occupies their own level, ranging from 0 to 5 points, depending on the specific circumstances associated with that person. It should be noted that at any individual level, the person's score will be at least 1 point higher in a non-ecological environment. Therefore, the risk of contracting the disease increases at least 20%. If a particular change affects the ontogeny, thus moving the person from B2.b. to B2.c. and the acquired vulnerabilities from B3.b. to B3.c, the score increases up to 2 points. This increase corresponds to a disease risk of 40%. If the pathological mechanisms are affected, then the level increases by 2.5 points, i.e., the risk of disease increases by 50%. We can conclude that the non-ecological environment raises the person's risk of contracting the disease by 20-50%. The exact amount by which the risk increases will depend on the individual case. If the increase in the risk is 20%, then one person in five will risk contracting the disease if that person is placed in a non-ecological environment. If this increase is 50%, then one of every two people will risk contracting the illness if the person is placed in a non-ecological environment. This increased risk of illness will be called the potential disease-specific risk statistical factor, or, stated more concisely, the Ecological Risk Index (E.R.I.). Of the above it is shown that $E.R.I. = A1+ B1+ B2+ B3+ B4$ but, because all the B factors are interspecific interdependent I propose that they form an average.

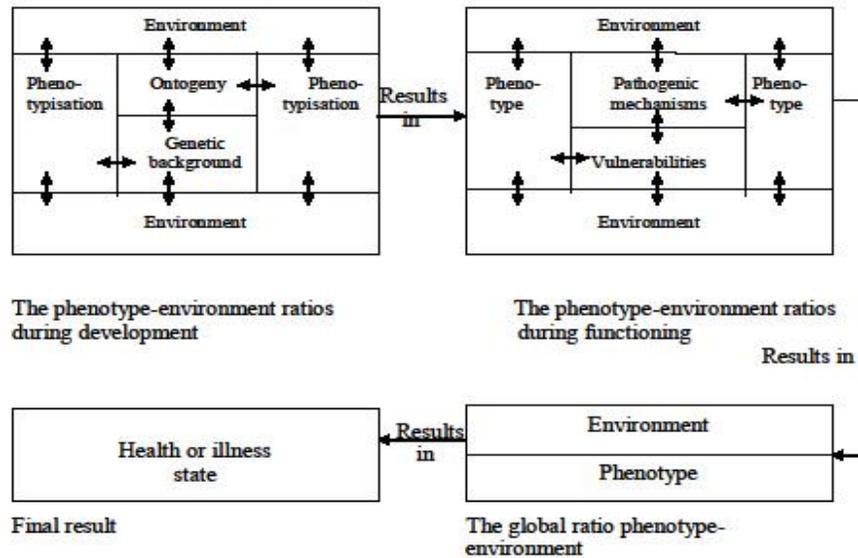


Fig. 1. Diagram of the dynamic relationship between humans and the environment in terms of health or disease

This results in $E.R.I. = A1 + \left(\frac{B1 + B2 + B3 + B4}{4} \right)$. This risk increases if the non-ecological environment is affected by more than one parameter (i.e. biparametrically, triparametrically, n-parametrically affected). We may hypothesize that if current trends continue, man will completely overreach his naturally inherited habitat and that this natural habitat will be replaced by an artificial habitat (e.g. sound pollution). Natural nourishment might be completely replaced by artificial nourishment obtained from genetically engineered plants. The changes associated with this replacement might include the complete industrialization of food production and the conservation of food resources. Furthermore, space travel will require a change to artificial nourishment. Isolation from birth from the natural and original environment of man transforms immune pathology into a significant public-health issue. The effects of background noise (e.g. radio and television) and visual backgrounds (e.g. television and computer screens) have not yet been documented. It is known that genetically engineered foods are a factor in the incidence and prevalence of Alzheimer's Disease [8].

What consequences will ensue for humans after the process of ecological mutation is complete? These consequences may be far-reaching and

may affect the processes of living, breathing, feeding and feeling. Are humans ready for this type of mutation? Of course, these mutations do not make people ill in the classical sense, but they contribute to a person's vulnerability. Under primitive conditions, natural selection would have acted. However, is the intervention of natural selection at all acceptable? Will the accommodation of a species occur through self-selection? The different types of questions about the ecological aspect of the human future are both numerous and extremely serious. I believe that we must introduce and accept the concepts of ecological pathology and ecological disease, and that a thorough scientific study of ecopathology through the establishment of institutes or departments dedicated to the subject is needed. The term "ecological pathology" refers to any pathology with preexisting vulnerability and pathogenic mechanisms activated by ecological factors, thereby increasing their incidence and prevalence, and also to illnesses that emerge through mechanisms generated by ecological factors.

These theoretical results are consistent with other theoretical results that indicate that the parameters of the non-ecological environment can be viewed as stressing factors whose effects are [14] "...progressively destructive and time-dependent, from primary processes and chronic phenomena to chronic illness".

4. CONCLUSION

In conclusion, the human environment is becoming increasingly less ecological in different ways. If environmental parameters exceed their homeostatic limits, they may transform vulnerabilities without critical mass and pathogenic vulnerabilities without critical mass into vulnerabilities and mechanisms with critical mass, respectively and thus produce illness. We can intuitively express the man-environment relationships that have this effect in graphical form. The evolution of the essential human context implies giving a new dimension to the pathogeny of human illnesses by introducing the new concept of ecological pathogeny or ecological illnesses in recognition of their ecological origin. A Potential Ecological Risk Index can be calculated. This chapter is important for giving coherence and unity to the conceptual frameworks and disciplinary perspectives of curative and preventive medicine. The chapter is needed to regularize and systematize the perspectives of all academic disciplines as well as social and political courts that address human habitats and human existence.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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