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DNA, Diseases; Stress

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Abbreviations: HPA: Hypothalamic Pituitary Adrenal; TNF-α: Tumor Necrosis Factor Alpha; DNA: Deoxyribonucleic Acid; ACTH: Adrenocorticotropic Hormone; IL: Interleukin

Introduction

The age of the genome, the age of the phenome, the age of the proteome, the era of epigenetics. Now, when one closes one's eyes and imagines that "Mitochondrial Eva" (primitive African woman), whose multiple motives, made her migrate to Europe; in what time? And so to the rest of the Continents. What happened to you? What were their needs in possibly an inhospitable environment, dangerous insecurities, with changes in the food habitat, causing a "stress" surely chronic, as well as acute? It is possible that those changes in the external environment, were conditioning the changes in the internal environment, activating the "stress", a hormonal alteration, of neurotransmitters as a threat to homeostasis, against which the organism, to survive, reacts with a large number of adaptive responses and modulation of the immune system. The stress of the Latin "stringere": squeezing or fatigue material conditions a physiological reaction of biological organisms, like the human, through different mechanisms that in defense allows facing a threatening situation for the individual, or that requires a greater demand for the. The first can be considered "pure stress", the second "eutress", it can be eutress because it can motivate it positively (this point cannot be discarded even in the primitive era), but in both definitions it is a challenge. The organism responds by means of a neurobiological, endocrine and immunological response that determines responses of social and psychological behaviors. Thus, the sympathetic nervous system promotes escape in the individual, and generates peripheral vasoconstriction, mydriasis, tachycardia, tachypnea, slowing of intestinal motility, etc. The homeostatic balance will promote the activation of the parasympathetic system which has a tendency to return the body to more normal physiological conditions.

For the understanding of the neuroendocrine-immune function in stress, it is necessary to study several mediators with immune function, as well as hormones of the hypothalamic-pituitary-adrenal (HPA) axis (adrenocorticotropic hormone (ACTH) and cortisol.) The prolactin hormone is not usually mentioned, which is a hormone that also increases in the state of stress and somatostatin that modulates various immune functions, such as the proliferation of lymphocytes. The hormones involved in stress inhibit the maturation of lymphocytes, responsible for specific immunity. Mediators with immune function include two cytokines: interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α) and also include leptin (which also acts as a proinflammatory peptide) and enkephalin.

Metabolically, these changes promote increased blood glucose (hyperglycemia fleeting or with some

permanence), the coagulation factors, free amino acids and immune factors. The blood is thrombolytic so the risk of stroke and other thrombosis are possible. The mechanisms associated with immune function are particularly relevant with respect to mood disorders, such as depression, and with respect to somatic diseases with affective components; chronic diseases of the immune system. The psychological reactions that stress causes have three components: emotional, cognitive and behavioral. In this regard it has been observed that prolactin (PRL) and oxytocin (OT) are pituitary hormones essential for lactation, but also promote behavior sexual. La OT estimula los comportamientos sociales, como el reconocimiento, el enfoque y el aprendizaje, pero se sabe menos sobre la PRL en estos comportamientos. Dado que la PRL y la OT tienen funciones complementarias en la reproducción, se ha planteado la hipótesis de que la PRL aumenta el reconocimiento social, el enfoque y el aprendizaje (observado en el comportamiento de las ratas al exponerse a la PRL y a la bromocriptina alternativa).

La HPL fisiológicamente aumentada por el estrés (sin referirse a los adenomas o carcinomas), promovería en la descendencia femenina en la edad adulta una disminución en la producción de PRL. Además, los niveles altos de prolactina pueden causar galactorrea, impotencia, disminución del deseo sexual (disminución de la libido) e infertilidad. Además, la hiperprolactinemia en hombres no tratados puede hacer que produzca menos o ningún esperma. Hay causas fundamentales que causan este trastorno hormonal: Por un lado, hay causas fisiológicas que pueden elevar temporalmente o permanentemente los niveles de prolactina, incluidos el embarazo, el estrés, el ejercicio excesivo (peregrinación) o la falta de sueño. Es posible pensar entonces, que en la era primitiva no se reproducían con frecuencia.

Thus, the emotional responses that occur in people affected by stress are the following: depression, sadness, irritability, apathy, indifference, emotional instability, etc. It is said that the stressors arrive through the organs of the senses (sight, hearing, touch, taste, smell), which then come the emotions, and with this the homeostatic, metabolic and biological changes.

So, let's turn our gaze towards that primitive woman (homo sapiens - "Mitochondrial Eva"), passing through threatening situations, climatic changes (environmental stimuli), food changes or lack of food, threat perception (by an animal, another homo sapiens, neanderthals, group pressure, fire etc.), all situations that forced the brain trigger of "stress", producing changes in the internal environment and this, may have conditioned that gene expression was modified? Either, with the participation of modifying genes for a given gene expression and later when the populations were settled and constituted, the variations in the DNA were presenting the haplogroups? If the population is smaller because they did not reproduce frequently, the appearance of haplogroups could be more feasible. Is it the stress that determines the gene variants, which would activate modifiers genes for a given gene expression, modifying it? Is stress the one that conditioned the variants that determine the haplogroups / haplotypes / subhaplotypes that would seem to influence the presentation of a disease, either exacerbating it or delaying it? Stress as a factor of disease, disease as a producer of stress.

Understanding the presentation of a disease in our times, involves a study of search, observation and analysis, which from the middle of the last century to date, has not culminated with the analysis of the proteome, after having gone through the genome and the phenome. From where it is taking the look towards the epigenetics. Nor for the disadvantages of an epidemiological disease, where some people die and others do not. There is still a lot to study.

Perhaps it is important to look at this "Mitochondrial Eva", migrating through the different continents (continental drift), had to adapt to a different external environment, causing a "stress" that could also have caused changes in their internal environment and modified the gene expression that she brought. Either, the environment, the culture, the food, the socialization and the mating of those who arrived before, of those who arrived later. In an adaptive survival that could have initiated the activity of modifier genes, for a given gene expression. As well as the modulation (in the meaning of its meaning: "change the factors that affect a procedure that allows different results"), which Haplogroups and Haplotypes could be giving, present in the Genome that each individual loads from their ancestors and the place where he has lived and lives.

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