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EC ENDOCRINOLOGY AND METABOLIC RESEARCH

Editorial

Ectodysplasin A (EDA): A Potential Biomarker of Metabolic Syndrome

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Received: January 25, 2021; **Published:** February 26, 2021

Ectodysplasin A (EDA) is a protein produced in humans by the EDA gene. Ectodysplasin A is a transmembrane protein of the TNF family that assumes an important role in the human production of ectodermal tissues including the skin [1,2]. It is identified by the receptor for ectodysplasin A [3-5].

The protein generated by this gene, which can be produced by furin to produce a stored shape, is a type II membrane protein [3,6,7]. The expressed protein, which relates to the tumor necrosis factors family, acts as a homotrimer and may even be involved in signal transduction during the development of ectodermal organs. The distinction of anatomical placodes, components of scales, feathers and hair follicles in vertebrates was shown to be related in accordance to c-Met [8]. Anhidrotic ectodermal dysplasia seems to be the origin of mutations in this gene, which is sometimes identified as X-linked hypohidrotic ectodermal dysplasia (HED). For this gene, multiple transcript variants have been identified that encode various different isoforms [9-11].

Inhibition of germline mutations in EDA in all vertebrates observed to date leads to the genetic condition designated HED. HED is an existence disorder in individuals that contributes to hyperthermia, particularly in infants caused by a lack of or dramatically reduced sweating [12]. In addition to our knowledge of the creation of ectodermal protrusions and the etiology of genetic disorders, EDA study extends our knowledge of the growth of many types of vertebrates, particularly humans. Experiments on HED mouse and dog species have led to one of the most impressive advancements in the study of integrated molecular genetics by showing that many of the HED-associated characteristics are improved by short-term management of neonates with an artificial ligand [12].

The EDA pathway, which would be effective mostly during formation of ectodermal tissues, like teeth, hairs, feathers and mammary glands, and what is necessary for the good of the developmental system controlling the quantity, size and density of all these systems, has been detected by comparing human subjects impaired by anhidrotic/hypohidrotic ectodermal dysplasia [13]. It includes three main gene products: EDA, a tumor necrosis factor (TNF) ligand-the alpha family, EDAR, a TNF alpha receptor-related receptor, and EDARADD, a specific adaptor. To regulate target genes, this main mechanism focuses on downstream NF-κB pathway activation [13]. Given its impact on human disease, the EDA pathway is a 'promising mechanism' that could enable evolutionary responses in ectodermal protuberances that are known hot-spots of costs to change as sophisticated interactions with the setting [13].

Lately, several studies indicate that EDA is a hepatokine that can lead to decreased insulin levels of the skeletal muscle in obesity [14]. By maintaining a balance among lipid accumulation and removal, EDA exacerbates steatosis. This would be a possible non-alcoholic fatty liver disease (NAFLD) biomarker [15]. NAFLD is now accepted in Western countries as among the most frequent form of chronic liver disease. Insulin resistance is an important element in NAFLD pathogenesis, which is known to become the hepatic insulin resistance or

obesity aspect [16]. Actually, NAFLD is not a component of the metabolic syndrome (MetS) diagnostic criteria; but, the development of NAFLD has some similar mechanisms with the development of MetS as they share the insulin resistance pathophysiological basis. It is also known that the liver manifestation of MetS is NAFLD [17,18]. The existence of at least three of the proposed criteria is necessary to classify MetS and sometimes it is appropriate for the classification of MetS to have only one laboratory meaning, changed by diet or drugs [17,18].

These have long always been known that, contrary to the common opinion that adipose tissue acts as an inert organ for storing extra energy as fat, adipose tissue is probably one of the main and most active organs of the human body and plays an essential part in controlling energy homeostasis [19]. Adipose tissue is among the first tissues to react to variations in nutritional status, like elevated consumption of food, fasting, reduced temperature exposure, and physical activity. In addition to lipolysis and metabolism of fatty acids, comprehensive renovation is also needed in various aspects, such as cell size and morphology, angiogenesis, normoxia/hypoxia responses, whitening/browning characteristics, immune characteristics [20].

Although these studies support the role of liver-derived EDA in the development of commonly associated NAFLD metabolic dysfunctions, clinical outcomes are complicated by marked differences in adiposity between groups of patients. In addition, there is an insufficient understanding of the relationship between the levels of EDA and the severity of NAFLD liver disease, especially in obesity, where the incidence of any degree of NAFLD relative to lean individuals is markedly increased. Therefore, EDA could be a potential biomarker of metabolic syndrome too.

Disclosure Statement

The author declare that there are no conflicts of interest.

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Volume 6 Issue 3 March 2021