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Letter to Editor
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Synergistic Actions of Thyroid-Adipokines Axis during Development

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Thyroid hormones (THs) are involved in the programming of early prenatal period [1-37]. Moreover, adipokines [leptin (LEP), resistin (RETN), adiponectin (ADP), inflammatory cytokines [interleukin-6 (IL-6), monocyte chemoattractant protein-1 (MCP-1) and tumor necrosis factor-α (TNF-α)], and chemokines [interleukin-8 (IL-8)] play an important role during the fetal development and early life [12,32,33]. THs also control the actions of LEP [8,9], interferon-γ [38], ADP [8,9,39], TNF-α [8,26], and growth factors [8,28,32,33,35,40,41] by non-genomic mechanisms. Alternatively, LEP, ADP, and TNF-α modulate the functions of thyroid axis and insulin sensitivity [8]. Also, adipokines can regulate the hypothalamic-pituitary-thyroid axis (HPTA), thermogenesis, body weight, basal metabolic rate and appetite [42]. The nuclear receptors such as thyroid receptors (TRs), peroxisome proliferator-activated receptor-α (PPARα), and liver X receptor (LXR) are vital for the triiodothyronine (T3) regulation of cholesterol metabolism and transcription of lipogenic and lipolytic genes [42]. In white adipose tissue (WAT), the expression and secretion of ADP was decreased throughout the pregnancy [13,43]. Moreover, the expression of LEP and RETN was observed in the developing placenta [44] with reducing the insulin level [45]. Also, the coordination in the secretion these cytokines is involved in the availability of energy during the gestation [46].

On the other hand, hyperleptinemia was noticed in hypothyroid state [47,48] due to the degradation of leptin [8,14,17,28,49], or stimulation the HPTA in rats [8,14,17,28], canine and human [48,49]. Also, a reduction in the body weight due to the disturbance in the actions of THs, leptin, adiponectin may reflect the decline in the general health during the development [8,17,28,50-53]. In hypothyroidism, LEP and ADP caused a dyslipidemia [54], particularly atherogenic dyslipidemia [55]. In parallel, Tsuji et al. [56] stated that there are links between the fetoplacental insufficiency, THs alterations, and immunosuppression. This state might be associated with the cardiovascular dysfunctions [57]. This reduction results from the disruption of the HPTA, the energy expenditure and cytokine networks [8,12,28], and inducing oxidative stress [58], and cell death [59]. Thus, any disorders in the thyroid-adipokines axis might alter the fetoplacental communications. This can inhibit the passage of maternal nutrients to fetuses and delay the critical development. Further additional studies are needed to detect the signaling of this axis during the gestation in human and animals.

Conflict of Interest

The author declares that no competing financial interests exist.

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