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Letter to the Editor

Letter to the Editor: Nutritional thrift can be associated with precocious puberty and premature adrenarche in children born small for gestational age

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Dear Editor,

I read the review article by Hwang et al., entitled "Long-term care, from neonatal period to adulthood, of children born small for gestational age" in Clinical Pediatric Endocrinology with great interest (1). In this review, the authors reported that infants born small for gestational age (SGA) tend to have short stature, neurocognitive dysfunction, and metabolic syndrome. These children are also prone to have precocious puberty, premature adrenarche, and faster progression of puberty (1). The underlying mechanism for precocious puberty in SGA children remains unclear. The mismatch between prenatal and postnatal weight gain in SGA children might lead to energy imbalance inducing puberty. Therefore, these children may be at an increased risk of precocious puberty or premature adrenarche.

Leptin is a paradoxical starvation hormone (2). Leptin is secreted by the adipose tissue in accordance with the nutritional status. Leptin levels signal the hypothalamus regarding the body energy level or nutritional status. Leptin also stimulates gonadotropin-releasing hormone (GNRH) expressing neurons in the hypothalamus for fertility (2). Infants born SGA tend to economize in their intrauterine life because of inadequate nutrition from the placenta. Due to this, their organs such as the heart, pancreas, kidney, liver, and spleen receive inadequate blood supply and these fetuses grow in a restricted environment. If there is a mismatch between prenatal food supply and postnatal food intake, SGA infants cannot tolerate extra energy; the fat formed due to this excess energy might not be safely stored in the visceral organs and subcutaneous adipose tissue (3, 4). It is hypothesized that this mismatch might be associated with the presence of hypertrophic and hyperplasic fat cells in their bodies. These fat cells can produce more leptin and send incorrect messages to the hypothalamus regarding the energy level; this suggests that these children have extra energy in their bodies because of hepato-visceral fat. Leptin from the fat cells also modulates GNRH release for inducing puberty. In SGA children, this extra energy may be used for starting puberty, due to which precocious puberty may occur in these children. Normally, an energy balance exists between the prenatal and postnatal periods. As long as the energy balance is positive because of central adiposity, puberty onset will be faster, resulting in exaggerated precocious adrenarche or earlier onset of pubertal development.

The mismatch between prenatal and postnatal weight gain may change the biological clock of puberty because of energy imbalance due to central adiposity. As long as SGA children maintain energy restriction in later life, they may not develop puberty earlier. It is believed that early onset of puberty or faster maturation in children with SGA may be an adaptive mechanism for using the excess energy present due to hepato-visceral fat.

Conflict of interest: The authors declare there is no conflict of interest in this paper.

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