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Commentary

Fat in infants – Facts & implications

Each year, around 41 million people die due to non-communicable diseases (NCDs) as per the World Health Organization reports[1]. Raised blood pressure, overweight/obesity, hyperglycaemia and hyperlipidaemia are the important metabolic risk factors which increase the risk of NCDs. The foetal origin hypothesis by Barker & Osmond[2], suggests that NCDs like coronary heart disease, type 2 diabetes mellitus and hypertension originate based on the responses of a foetus to undernutrition which can cause permanent changes in the structure and function of the body. According to this hypothesis, when the foetus is deprived of nutrition during crucial periods of development, the foetus can recourse to adaptive survival strategies, thus reorganizing the course of normal development. If the same individual is exposed to contrasting nutritional circumstances during his or her later life, these adaptations may become maladaptive. Intrauterine growth restriction or clinically abnormal thinness at birth strongly predicts the subsequent occurrence of hypertension, hyperlipidaemia, insulin resistance, type 2 diabetes and ischaemic heart disease[3]. To decrease the incidence of NCDs, it is important to understand the intricacies of foetal nutrition and how malnutrition may alter the physiology and metabolism. Based on the pathophysiologic findings, interventions can be initiated to decrease the damage.

It has been suggested that research related to weight gain and catch-up growth of preterm and small for gestational age (SGA) infants will help in devising better nutritional strategies. Animal studies have shown rapid catch-up growth of adipose tissue with low prenatal protein and postnatal high fat and calorie diet. During early postnatal days, SGA infants accumulate more fat than appropriate for gestational age (AGA) infants[4]. SGA infants have a rapid increase in skinfold thickness before their catch-up growth in weight. They also have rapid increase in insulin-like growth factor-1 and lipoprotein lipase concentrations indicating a rapid increase in neonatal fat deposition[5]. Better management of intrauterine undernutrition and later neonatal growth is important for better future outcome. Obesity is a key parameter for NCDs and it would be prudent to assess parameters related to fat mass especially during infancy.

In this context, the Chandigarh study by Kaur *et al*[6] on growth pattern of skinfold thicknesses in term symmetric and asymmetric SGA infants gain importance. This study included a total of 200 full-term SGA (symmetric SGA: male 50, female 50; asymmetric SGA: male 50, female 50) and 100 AGA infants born consecutively. Infants with birth weight within 10^{th} to 90^{th} percentile of intrauterine growth curves were considered as AGA, while those weighing $<10^{th}$ percentile as SGA. Ponderal Index (PI) was used to categorize infants into symmetric SGA (PI $\geq$2.2 g/cm^3) and asymmetric SGA (PI <2.2 g/cm^3). Triceps, sub-scapular, biceps, mid-axillary and anterior thigh skinfold thicknesses using Harpenden's skinfold caliper were measured at 1, 3, 6, 9 and at 12 months. Care was taken to minimize observer bias and inter-observer variation. Mean and standard deviation were computed for different skinfold thicknesses measured among male and female symmetric SGA, asymmetric SGA and AGA infants at each age level. Infants who dropped out were replaced with other age- and sex-matched infants. The attrition rate varied from two to 6.7 per cent. They have observed rapid fat deposition during the first three months and gradual reduction thereafter among SGA infants. AGA age infants continued to increase fat deposition till six months and then reduced subsequently[6].

Some of the drawbacks of this study were: (*i*) these infants were recruited during 2006–2008. It would have been interesting if they had followed them up till adolescence since obesity is becoming major issue during this period; (*ii*) feeding pattern,

educational and socio-economical levels also might have changed during this period although most of them were breastfed till five months of age; (*iii*) authors have not looked at blood levels of insulin-like growth factor-1 and lipoprotein lipase levels associated with fat metabolism. A simultaneous measurement of hormones or total body fat estimation by magnetic resonance imaging or dual-energy X-ray absorptiometry would have added value to the study. Future longitudinal studies may be required to confirm the hypothesis that these SGA infants are actually at risk for metabolic disorders later in life.

As one envisages the quantity of fat and distribution among infants, it would be worthwhile to analyze the fat phenotypes and their physiological aspects. Adipose tissue is not just a mere energy store, it is likely to be a potential target for intervention in several metabolic pathologies. Adipocytes and their precursors act as key metabolic regulators with their ability to integrate different systemic stimuli and responding with a specific endocrine secretion and modulating the energy balance[7]. White adipose tissue characterized by accumulation of triglycerides, is usually affected by malfunctions related to metabolic pathways. Brown adipose tissue (BAT) has a thermogenic function characterized by dense vascularity and sympathetic innervation. It consists of adipocytes filled with small lipid droplets and mitochondria specialized in dissipating energy derived from fatty acid oxidation. Apart from thermoregulation, BAT has also been demonstrated to act as an endocrine organ characterized by a specific brown adipokine secretion. Stimulating BAT content and activity may represent a suggestive target for the treatment of obesity and metabolic disorders[8-10]. Besides classical brown adipocytes, an additional type of uncoupling protein-1-expressing adipocytes with thermogenic properties has also been characterized[11-13]. These cells appear postnatally in white adipose depots through an adipogenic process called browning, following specific inductive stimuli and have been named inducible beige/brite adipocytes[14]. Classical BAT, developing from the same dermomyotome is confined to a discrete anatomic distribution (interscapular and perirenal) during neonatal life and dramatically regress in adults. It is still not clear whether classical brown and beige adipose cells play different functions in controlling metabolic processes. Describing the molecular signatures and functional properties of these two phenotypes of adipocytes and documenting the differences in their adipogenic processes are important areas of research and the findings will provide useful inputs for the development of effective metabolic therapeutic strategies in future[7].

***Conflicts of Interest*:** None.

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References

1. World Health Organization. *Management of noncommunicable diseases*. Available from: *https://www.who.int/activities/management-of-noncommunicable-diseases*, accessed on January 2, 2020.
2. Barker DJ, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* 1986; *1* : 1077-81.
3. Nair L, Nair MK, Chacko DS. Markers of fetal onset adult diseases. *Indian Pediatr* 2009; *46* Suppl : s48-54.
4. Okada T, Takahashi S, Nagano N, Yoshikawa K, Usukura Y, Hosono S. Early postnatal alteration of body composition in preterm and small-for-gestational-age infants: Implications of catch-up fat. *Pediatr Res* 2015; *77* : 136-42.
5. Yoshikawa K, Okada T, Munakata S, Okahashi A, Yonezawa R, Makimoto M, *et al*. Association between serum lipoprotein lipase mass concentration and subcutaneous fat accumulation during neonatal period. *Eur J Clin Nutr* 2010; *64* : 447-53.
6. Kaur H, Bhalle AK, Kumar P. Growth pattern of skinfold thicknesses in term symmetric & asymmetric small for gestational age infants. *Indian J Med Res* 2021; *154* : 461-6.
7. Di Franco A, Guasti D, Squecco R, Mazzanti B, Rossi F, Idrizaj E, *et al*. Searching for classical brown fat in humans: Development of a novel human fetal brown stem cell model. *Stem Cells* 2016; *34* : 1679-91.
8. Peirce V, Vidal-Puig A. Regulation of glucose homoeostasis by brown adipose tissue. *Lancet Diabetes Endocrinol* 2013; *1* : 353-60.
9. Villarroya J, Cereijo R, Villarroya F. An endocrine role for brown adipose tissue? *Am J Physiol Endocrinol Metab* 2013; *305* : E567-72.
10. Wang GX, Zhao XY, Lin JD. The brown fat secretome: Metabolic functions beyond thermogenesis. *Trends Endocrinol Metab* 2015; *26* : 231-7.

11. Harms M, Seale P. Brown and beige fat: Development, function and therapeutic potential. *Nat Med* 2013; *19* : 1252-63.

12. Betz MJ, Enerbäck S. Human brown adipose tissue: What we have learned so far. *Diabetes* 2015; *64* : 2352-60.

13. Sidossis L, Kajimura S. Brown and beige fat in humans: Thermogenic adipocytes that control energy and glucose homeostasis. *J Clin Invest* 2015; *125* : 478-86.

14. Rosenwald M, Wolfrum C. The origin and definition of brite versus white and classical brown adipocytes. *Adipocyte* 2014; *3* : 4-9.