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Effect of an early fat-rich diet on immune cell functions and redox status of adolescent mice

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The number of children consuming hypercaloric diets has been rapidly increasing in developed countries during the last decade. This fact promotes a high rate of obesity during adolescence, which in turn leads to worsened health during this period[(1)]. In a model of premature ageing in mice oxidative stress and an impairment of relevant functions of peritoneal immune cells have been shown in very young animals, which may be related to premature immunosenescence[(2)]. Therefore, we proposed obesity as a model of premature immunosenecence[(3)] and we have suggested that diverse factors of lifestyle, such as nutrition and stress situations among others are involved in the homeostatic features of subjects, and thus in the health maintenance during their whole life[(4)]. The aim of this work was to study several function and oxidative stress biomarkers in peritoneal immune cells of obese adolescent mice fed with a fat-rich diet during childhood. Young female ICR mice ($n = 20$) (9 weeks old) were divided into two groups: 1) Controls (C): fed *ad libitum* with a maintenance diet (Harlan) and 2) fed *ad libitum* with a fat-rich diet (Harlan) (4 weeks with 9% fat and 2 weeks with 60% fat). Peritoneal cell suspensions were obtained from both groups of adolescent mice (15 weeks of age) and the following functional biomarkers were analysed: macrophage phagocytosis (both phagocytic and efficiency index), NK activity against tumour cells and proliferative response to mitogens (ConA and LPS). The following redox biomarkers were also evaluated: xanthine oxidase (XO) (an enzyme producing oxidants such as superoxide anion) and catalase (CAT) (an antioxidant enzyme) activities and glutathione (antioxidant) levels. A significant decrease in macrophage phagocytosis ($P<0.001$), NK activity ($P<0.05$) and proliferative response of lymphocytes T to ConA ($P<0.01$) were shown. However, no differences were obtained in the redox parameters evaluated. In conclusion: adolescent mice fed during their childhood with a fat-rich diet showed impairment in several relevant immune functions, which may predispose them to a high risk of infections and developing cancer. However, surprisingly immune cell redox status was not affected in these mice. Thus, although obesity is an oxidative stress situation, the impairment of immune functions seems to be previous to redox alterations.

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