

Increase in the secretion of pro-inflammatory cytokines by peritoneal leucocytes in a genetic mouse model of obesity and metabolic syndrome

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Metabolic syndrome is characterised by a low-grade chronic inflammation involving white adipose tissue⁽¹⁾. Db/db mice show a mutation in the leptin receptor gene and are a model of genetic obesity as well as metabolic syndrome⁽²⁾. Previous studies have shown an impairment of the lymphoproliferative response in these obese mice at two months of age, which seems to show a premature immunosenescence. It is well known that with ageing there is an imbalance in the secretion of cytokines^(3,4) with higher levels of pro-inflammatory (PI) (such as TNF-alpha and IL-1beta) compared to the anti-inflammatory (AI) (for example, IL-10) cytokines. For this reason, we have investigated the secretion of these cytokines by leucocytes in young obese mice. Female genetically obese mice (db/db), lean heterozygotic mice (db/+) and wild type mice (+/+) fed with a standard diet were used. The peritoneal leucocytes of these animals at two months of age were obtained and maintained in a culture supplied with lipopolysaccharide (LPS) of *E.coli*. After 24 hours the culture medium was collected and the secretions of IL-10, IL-1beta and TNF-alpha were measured by luminometry. The results showed that the secretions of IL-10 were similar in the three groups studied. However, the secretions of TNF-alpha and IL-1beta were higher ($p < 0.05$ and $p < 0.01$, respectively) in obese mice compared to wild control animals. In conclusion, young obese mice showed high levels of PI cytokines and this early PI/AI imbalance might contribute to the chronic inflammatory status associated with obesity and to the development of premature oxi-inflamm-aging in these animals.

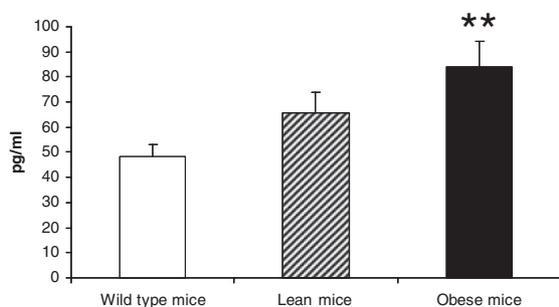


Figure 1. IL-1 BETA.

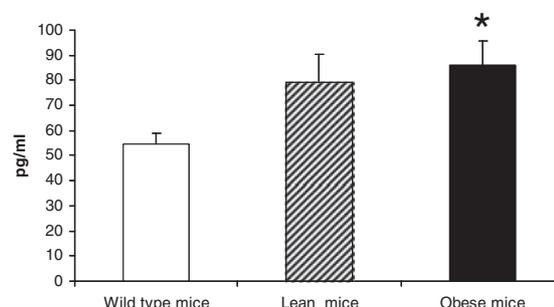


Figure 2. TNF-ALPHA.

Data are expressed as mean \pm SE * $p < 0.05$; ** $p < 0.01$ with respect to the Wild type group.

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