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Fat-rich diet-induced obese mice show premature immunosenescence

O. Hernández[1], L. E. Díaz[2], G. Baccan[2,3], A. Gheorge[2], B. Zapatera[2], A. Marcos[2] and M. De La Fuente[1]

[1]*Department of Physiology. Faculty of Biology, Complutense University of Madrid,* [2]*Department of Metabolism and Nutrition. Institute of Food Science, Technology and Nutrition (ICTAN), Spanish National Research Council (CSIC), Madrid, Spain and* [3]*Institute of Health Sciences (Federal University of Bahia), Salvador, Brazil*

Obesity, a modern pandemic, is associated with a chronic grade of oxidation-inflammation, which may produce an acceleration of the immune system impairment. Since the age-related changes on the immune system seem to be similar to those found in obese subjects, we have proposed obesity as a model of premature immunosenescence[(1)]. This fact could be one of the reasons to develop an early ageing rate related to obesity as well as a high risk of morbidity and mortality. The aim of the present study was to confirm the premature immunosenescence of obese adult mice fed with a fat-rich diet, analysing several functions in peritoneal immune cells such as chemotaxis, NK activity and lymphocyte proliferation, which have been proposed as biomarkers of the ageing rate and predictors of longevity[(2)]. Young female ICR mice ($n = 16$) (14 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 high fat diet (HFD) containing 60% fat (Harlan). After 14 weeks (when animals were adults and HFD group developed obesity features), mice were sacrificed and peritoneal cell suspensions were obtained from both groups (HFD and C). The following functions were evaluated in peritoneal leukocytes: lymphocyte and macrophage chemotaxis, phagocytosis of inert particles by macrophages, anti-tumour NK activity and lymphocyte proliferative response to mitogens such as ConA and LPS (Table). The results showed that chemotaxis capacity of both macrophages and lymphocytes, NK activity and the proliferative response of lymphocytes to both mitogens (ConA and LPS) were significantly lower in HFD mice than in C group. Since an age-related decrease of these functions has been already shown[(2)]) we can confirm that adult mice developing obesity after having been fed with a fat-rich diet during their adolescence show a premature immunosenescence, which predispose them in the adulthood to a high risk of infection and tumours. This premature impairment of key immune functions could be related to a shorter life span, suggesting a status of premature ageing in obese mice.

Table. Functions in peritoneal immune cells

		CONTROL	HFD	
FUNCTIONS	N	Mean ±SD	Mean±SD	*P*
Macrophage chemotaxis Index	8	963±113	554±64	0.001
Lymphocyte chemotaxis Index	8	1005±109	620±100	0.001
Phagocytosis Index	8	251±69	231±49	0.507
Phagocytic efficacy Index	8	68±6	64±10	0.329
Natural killer activity (% Lysis)	8	67±23	46±12	0.010
Proliferative response ConA	8	6551±1299	2773±1035	0.001
cpm) LPS	8	8521±2394	2394±1736	0.001

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