HIGH FAT DIET CONSUMPTION IMPAIR EARLY $\alpha_7$nAChR EXPRESSION IN SPLEEN OF SWISS MICE.

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Antiinflammatory cholinergic pathway can control the inflammatory response. This neural circuit is responsible for converting signals from the brain to peripheral tissues, subunit-dependent manner $\alpha_7$ nicotinic acetylcholine receptor ($\alpha_7$nAChR). High-fat diet-induced obesity and insulin resistance (IR) are associated with inflammation, but the relation between these metabolics disorders and cholinergic anti-inflammatory pathway are unclear. To determine factors that may cause IR, we have performed a time-course study in mice fed a high fat diet (HFD). Swiss male mice were fed (3 days, 5, and 16 weeks) with standard chow or HFD (60% fat). Body weight, fasting glucose, epididymal fat pad mass, spleen inflammatory cytokines and $\alpha_7$nAChR expression were evaluated. Body mass, fasting glucose and epididymal fat pad mass were significant increased in HFD compared to SC mice. IL-1$\beta$ expression was increased after 5 and 16 weeks (2.1-fold and 2.6-fold, respectively) in HFD compared to SC mice. TNF-$\alpha$ gene expression was 1.5-fold higher in HFD than SC mice (5 weeks). $\alpha_7$nAChR gene expression was reduced (3.2-fold) in HFD compared to SC mice (3 days), but for 5 and 16 weeks did not observe difference between groups evaluated. These results show that HFD consumption modulates early (3 days) spleen $\alpha_7$nAChR gene expression compared to proinflammatory cytokines expression (5 and 16 weeks). In this condition the inflammatory response could be elevated and promote damage to tissues.

Acknowledgments: CAPES, CNPq and FAPESP.

Key words: cytokines, $\alpha_7$nAChR, mice.