Tuberculosis (Tb) is a public health problem with around 1.4 million deaths per year [1]. In the lungs is observed an intense influx of cells to the site of infection where they can form structures called granulomas [2]. It has been observed the differentiation of macrophages (MØ) in “foamy cells” in granulomas [3]. The foam aspect of MØ is a reflex of intracellular lipid accumulation [3,4,5]. Lipid body structural features, including lipid and protein composition may vary according to the cell type, activation state and inflammatory environment and thus may determine different cellular functions for lipid bodies [6]. Obesity is another health problem worldwide, causing the deaths of almost 3 million of people [1]. It is associated with chronic inflammatory response of white adipose tissue due to infiltration of MØ, responsible for overexpression of TNF-α and IL-6 [7]. Our objective was to evaluate the involvement of obesity in influx and activation of cells in experimental infection with M. bovis BCG in mice, aiming to clarify the physiopathology of Tb and the role of metabolic disorder in bacterium replication.

C57BL6 mice were divided in 2 groups fed with high-sugar diet or common chow. After 90 days, the mice were intrapleurally infected by BCG and the control received saline. (Animal euthanasia ethical approval: #109/2012 CEUA/UFJF). The leukocyte influx and lipid body enumeration was performed at 24h after infection. It was observed an increase in the mass of fat in the high-sugar diet (Mean ± SEM: from: 0.112 ± 0.017 in control to 0.219 ± 0.002 in high-sugar group; n=10) and a significant reduction in influx of leukocytes into the pleural cavity (2.05 ± 0.366 in control to 20.100 ± 5.460 in infected on common chow group; from: 4.480 ± 0.615 in control to 8.080 ± 2.168 in infected on high-sugar diet group; n=5) as well as the neutrophils and eosinophils migration in obese mice. Also, there was less lipid body formation in obese compared with normal animals (from 1.200 ± 0.060 in control to 3.920 ± 0.738 in infected group on common chow; from1.713 ± 0.081 in control to 1.460 ± 0.102 in infected on high-sugar diet group). Our data suggest that the largest quantity of adipose tissue disadvantage the BCG. The fact the reduction in the lipid body formation and leukocyte migration in obese individuals indicated a correlation of obesity under the progression of experimental infection with M. Bovis.


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Fig. 1: Leukocyte migration in control individuals, performed at 24h after infection, showed mononuclear cells (arrow). Bar = 50 μm

Fig. 2: Leukocyte influx in infected individuals, performed at 24h after infection, showed neutrophils (black arrow indicated) and eosinophils (white arrow indicated) migration. Bar = 50 μm

Fig. 3: Lipid bodies (black arrow) observed on mononuclear cells from the pleural cavity of the infected group. Bar = 50 μm