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Endotoxin induces pulmonary inflammation in obese mice

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Obesity is a global public health concern that has been related to an increased risk of several chronic diseases, such as cardiovascular diseases, type II diabetes mellitus, obstructive sleep apnea. As overweight has become more prevalent, the effect of obesity on acute lung injury incidence and outcome has gained more attention in recent years. Despite this negative correlation, some investigations have revealed a controversial correlation, termed "obesity paradox" in which overweight with established cardiovascular disease have a better prognosis. To elucidate further on this issue, we conducted this study with a diet-induced obesity murine model. Obese mice, adult C57BL/6J mice fed a high-fat diet for 12 weeks, received normal saline or endotoxin (lipopolysaccharide, 10 mg/kg, intraperitoneally administered) (denoted as the Obese and LPS group, respectively). After 48 hours of administration of normal saline or endotoxin, mice were euthanized. The level of lung injury (injury score, tissue water content, and leukocyte infiltration in lung tissues) in the LPS group was significantly higher than in the Obese group (p=0.0002; =0.02; and =0.0001, respectively; Figure 1). The levels of pulmonary cytokines (tumor necrosis factor-α [TNF-α], interleukin-6 [IL-6], and interleukin-1β [IL-1 β]) in the LPS group were also significantly higher than in the Obese group (p=0.03;=0.0003; =0.0007; respectively; Figure 1D-F). Moreover, the level ofpulmonary inducible nitric oxide synthase (iNOS, indicator of pro- inflammatory M1 phase macrophage polarization) in the LPS group was significantly higher than in the Obese group (p=0.0087; Figure 1G). The expression levels of nuclear factor-κB (NF-κB) and hypoxia-inducible factor-1α(HIF-1α) in lung tissues in the LPS group were significantly higher than in the Obese group (p=0.003 and <0.0001, respectively; Figure 2A), too. Similar pictures were observed in the levels of oxidation and apoptosis in lung tissues, as the levels of malondialdehyde (MDA) and DNA fragmentation (assayed using the terminal deoxynucleotidyl transferase dUTP nick end labeling [TUNEL] method and the count of TUNEL-positive cells) in the LPS group were significantly higher than in the Obese group (p<0.0001 and =0.001, respectively; Figure 2B-C)

In conclusion, data from this study collectively demonstrate that endotoxin induces significant inflammation in obesity mice.

Biography

Milton Chiang currently is a Ph.D. candidate in the department of the International Master/Ph.D. program in Medicine, Taipei Medical University, Taiwan. He got his specialty in Internal Medicine at Francisco Marroquin University, Guatemala, and Medical Degree in Rafael Landivar University, Guatemala. His research focuses on obesity, inflammation, and its therapeutic.

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