

Diet and exercise restore immune function in obesity

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(PhysOrg.com) -- Boston University scientists say that moderate daily exercise and dietary control might reverse immune dysfunctions found in people with obesity.

Overeating and a sedentary lifestyle are well-known risk factors for obesity, which is linked to <u>hypertension</u>, <u>heart disease</u>, diabetes, gum disease, certain cancers, and <u>asthma</u>.

Research has suggested that a change in immune function is a predecessor to all these diseases and researchers at Boston University Henry M. Goldman School of Dental Medicine (GSDM) have previously shown that obesity causes immune defects that make it hard to fight infection.

Until now, little was known about how diet and exercise affects immunity in obese people.

Researchers worked with diet-induced obese mice in four groups:

- lean mice on a standard chow diet
- obese mice on a high fat diet
- obese mice on a high fat diet on a moderate exercise plan for four weeks, and
- obese, high fat diet mice given moderate exercise and a four-week standard chow diet



Moderate daily exercise and dietary control dramatically restored immune function. Obese <u>mice</u> saw damaged cytokines—signaling molecules that help immune cells talk to each other—repaired and an improved ability to fight <u>gum disease</u> as measured by bone loss.

"The study underscores the necessity to correct two important factors in obesity—diet and exercise—to improve markers of immune dysfunction and bone loss," says senior author Dr. Salomon Amar. "The correction of one factor only may not lead to any tangible changes."

More information: The study, Signaling mechanisms in the restoration of impaired immune function due to diet-induced obesity, is a collaboration of GSDM Director of Anti-Inflammatory Therapeutics Dr. Salomon Amar, Boston University School of Medicine Professor Dr. Susan Leeman, and researcher Dr. Qingde Zhou. The study appears in the *Proceedings of the National Academy of Sciences* and is available online at www.pnas.org/content/108/7/2867

Provided by Boston University

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