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OBESITY

INFLUENZA IMMUNITY IMPAIRED IN OBESITY

Obesity could reduce the ability of the immune system to mount responses to influenza vaccines, according to researchers from the University of North Carolina, Chapel Hill, USA. "There are two components of the immune response that you want to stimulate with vaccination," explains senior investigator Melinda Beck, "one is antibody production and the other is $CD8^+$ T cell function."

In a previous study, Beck and colleagues observed that 60% of obese mice died after challenge with an influenza virus compared with 4% of lean mice. "We wanted to extend our study to humans, but because we couldn't infect people with influenza, we decided to investigate responses to influenza vaccination in healthy-weight, overweight and obese adults," says Beck.

The 2009–2010 inactivated trivalent influenza vaccine was given to 499 study participants, who returned after 28–35 days for measurement of influenza antibody titers. $CD8^+$ T cell function was assessed by measuring activation status as well as interferon γ and granzyme B expression. A total of 74 participants were followed up for 12 months.

Although BMI correlated positively with antibody titers 1 month after vaccination, high BMI was associated with a greater decline in levels of influenza antibody after 12 months than low BMI. Measures of $CD8^+$ T cell activation and functional responses to *ex vivo* influenza virus challenge showed impaired T cell function in individuals with obesity compared with healthy-weight participants.

These results have important implications for vaccine development and influenza immunity programs. Vaccines designed to stimulate $CD8^+$ T cell memory could have reduced efficacy in obese populations, and the inability to maintain long-term antibody responses might mean that people with obesity could be at risk of influenza even when vaccinated.

The researchers plan to determine whether vaccinated individuals with obesity are really at greater risk of influenza than those of healthy weight and aim to investigate a possible mechanism. "One hypothesis is that increased leptin production associated with obesity could have a deleterious effect on immune signaling," concludes Beck.

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