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Contagious obesity? Identifying the human adenoviruses that may make us fat

Human adenoviruses may cause human obesity, but more research is needed before a screening test and vaccine become reality. Meanwhile, one researcher advises, 'Eat right, exercise, wash your hands'

There is a lot of good advice to help us avoid becoming obese, such as "Eat less," and "Exercise." But here's a new and surprising piece of advice based on a promising area of obesity research: "Wash your hands."

There is accumulating evidence that certain viruses may cause obesity, in essence making obesity contagious, according to Leah D. Whigham, the lead researcher in a new study, "Adipogenic potential of multiple human adenoviruses in vivo and in vitro in animals," in the January issue of the American Journal of Physiology-Regulatory, Integrative and Comparative Physiology published by the American Physiological Society.

The study, by Whigham, Barbara A. Israel and Richard L. Atkinson, of the University of Wisconsin, Madison, found that the human adenovirus Ad-37 causes obesity in chickens. This finding builds on studies that two related viruses, Ad-36 and Ad-5, also cause obesity in animals.

Moreover, Ad-36 has been associated with human obesity, leading researchers to suspect that Ad-37 also may be implicated in human obesity. Whigham said more research is needed to find out if Ad-37 causes obesity in humans. One study was inconclusive, because only a handful of people showed evidence of infection with Ad-37 ‑not enough people to draw any conclusions, she said. Ad-37, Ad-36 and Ad-5 are part of a family of approximately 50 viruses known as human adenoviruses.

Researchers now must:

- identify the viruses that cause human obesity

- devise a screening test to identify people who are infected
- develop a vaccine

Screening test and vaccine still a long way off

The Whigham et al. study prompted an editorial in the same issue of AJP-Regulatory, Integrative and Comparative Physiology by Frank Greenway, professor in the Department of Clinical Trials, Pennington Biomedical Research Center, Louisiana State University, Baton Rouge.

"If Ad-36 is responsible for a significant portion of human obesity, the logical therapeutic intervention would be to develop a vaccine to prevent future infections," Greenway wrote. "If a vaccine were to be developed, one would want to ensure that all the serotypes of human adenoviruses responsible for human obesity were covered in the vaccine."

"If one could predict the potential of an adenovirus to cause human obesity by using an in vitro assay or even by animal testing, screening of the approximately 50 human adenoviruses might be accelerated, shortening the time required for vaccine formulation," Greenway wrote. "Human antibody prevalence in obese and lean human populations appears to be the only reliable method to screen adenoviruses for their potential to cause obesity in humans at the present time," he noted.

Obesity contagion theory slow to catch on

The notion that viruses can cause obesity has been a contentious one among scientists, Whigham said. And yet, there is evidence that factors other than poor diet or lack of exercise may be at work in the obesity epidemic. "The prevalence of obesity has doubled in adults in the United States in the last 30 years and has tripled in children," the study noted. "With the exception of infectious diseases, no other chronic disease in history has spread so rapidly, and the etiological factors producing this epidemic have not been clearly identified."

"It makes people feel more comfortable to think that obesity stems from lack of control," Whigham said. "It's a big mental leap to think you can catch obesity." However, other diseases once thought to be the product of environmental factors are now known to stem from infectious agents. For example, ulcers were once thought to be the result of stress, but researchers eventually implicated bacteria, H. pylori, as a cause.

"The nearly simultaneous increase in the prevalence of obesity in most countries of the world is difficult to explain by changes in food intake and exercise alone, and suggest that adenoviruses could have contributed," the study said. "The role of adenoviruses in the worldwide epidemic of obesity is a critical question that demands additional research."

Ad-37 third virus implicated in animal obesity

The theory that viruses could play a part in obesity began a few decades ago when Nikhil Dhurandhar, now at Pennington Biomedical Research Center at LSU, noticed that chickens in India infected with the avian adenovirus SMAM-1 had significantly more fat than non-infected chickens. The discovery was intriguing because the explosion of human obesity, even in poor countries, has led to suspicions that overeating and lack of exercise weren't the only culprits in the rapidly widening human girth. Since then, Ad-36 has been found to be more prevalent in obese humans.

In the current study, Whigham et al. attempted to determine which adenoviruses (in addition to Ad-36 and Ad-5) might be associated with obesity in chickens. The animals were separated into four groups and exposed to either Ad-2, Ad-31, or Ad-37. There was also a control group that was not exposed to any of the viruses. The researchers measured food intake and tracked weight over three weeks before ending the experiment and measuring the chickens' visceral fat, total body fat, serum lipids, and viral antibodies.

Chickens inoculated with Ad-37 had much more visceral fat and body fat compared with the chickens infected with Ad-2, Ad-31 or the control group, even though they didn't eat any more. The Ad-37 group was also generally heavier compared to the other three groups, but the difference wasn't great enough to be significant by scientific standards.

The authors concluded that Ad-37 increases obesity in chickens, but Ad-2 and Ad-31 do not. "Ad-37 is the third human adenovirus to increase adiposity in animals, but not all adenoviruses produce obesity," the study concluded.

There is still much to learn about how these viruses work, Whigham said. "There are people and animals that get infected and don't get fat. We don't know why," she said. Among the possibilities: the virus hasn't been in the body long enough to produce the additional fat; or the virus creates a tendency to obesity that must be triggered by overeating, she said.

Mass screening for these viruses is impractical right now because there is no simple blood test available that would quickly identify exposure to a suspect virus, Whigham et al. said. More work is needed to develop such a test, Whigham said.

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Source, funding and disclosure

"Adipogenic potential of multiple human adenoviruses in vivo and in vitro in animals," by Leah D. Whigham and Richard L. Atkinson of the Departments of Medicine and Nutritional Sciences at the University of Wisconsin, Madison, and Barbara A. Israel of the Department of Pathobiological Sciences, University of Wisconsin, Madison, is in the January issue of the American Journal of Physiology - Regulatory, Integrative and Comparative Physiology published by the American Physiological Society.

Research was supported by grants from the National Institute of Diabetes and Digestive and Kidney Diseases, and the Beers-Murphy Clinical Nutrition Center, University of Wisconsin. Atkinson, now at the Virginia Commonwealth University, owns all shares of Obetech LLC, a company that markets assays to detect infection with human adenovirus-36 and owns patent rights for these assays.

Editor's note: The media may obtain a copy of Whigham et al. by contacting Christine Guilfoy, American Physiological Society, 301-634-7253 or cguilfoy@the-aps.org.

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