

Obesity and sedentary behavior: Which is chicken, which is egg?

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This is an image of a weight scale. Credit: CDC/Debora Cartagena

If you dread the prospect of hauling your lazy rear end to the gym more often in 2017, new research suggests the extra weight you've been carrying around may be to blame.

That may sound obvious. But the relationship between excess weight and inactivity is anything but. Behind the twin epithets "fat and lazy" lie

some unkind - and unclear - beliefs about whether inactivity leads to [weight gain](#) or whether weight gain leads to inactivity.

A new study offers evidence that diet-induced obesity alters the brain's functioning in ways that suppress the natural impulse to move around.

Many other powerful factors influence our inclination to exercise - not least having the safe spaces, leisure time and social encouragement to do so. But new research in [mice](#) confirms that obesity disrupts the proper functioning of a key docking station for dopamine, a brain chemical that affects our moods, appetites and motor control.

The result: The chubby mice became couch potatoes.

When researchers fattened mice up on high-fat chow, they saw the activity of a specific class of dopamine receptor in the brain's striatum (a center of movement control and reward-seeking behavior) fall. Along with that change, they observed that the obese mice adopted more sedentary habits than their lean peers.

When researchers experimentally turned down or knocked out that brain receptor's activity in lean mice who were fed normal chow, those mice too lost the impulse to run on their running wheels or zip around their cages. They did not, however, become obese.

And when researchers took [obese mice](#) and experimentally "turned up" the receptor's faulty signaling, they saw the chubby mice step up the frequency of their [physical activity](#).

The new research, published Thursday in the journal *Cell Metabolism*, suggests that inactivity is not a natural cause of obesity. The activity of the affected dopamine receptor varies considerably among mice and presumably in humans, and it's clearly not the case that the lazy among

us all get fat.

Rather, inactivity appears to be a downstream consequence of excess weight, the new findings suggest. As such, it may not only encourage further weight gain. Since we know that regular exercise can prevent or mitigate the effects of obesity-related diseases, lack of exercise may foster the development of such conditions as Type 2 diabetes, hypertension and worrisome cholesterol.

The research also suggests that carrying [excess weight](#) may subtly interfere with the rewards we are meant to get by physical activity.

Not all of us feel joy at the prospect of mounting the elliptical or stair climber for a solid aerobic workout. But free of the spandex and the guilt and the goading of muscled trainers, properly fed experimental mice will happily run for hours on a running wheel. It's when they avoid the wheel that lab technicians wonder what's wrong.

Humans are likely no different - though now we have cars and computers and televisions to induce us to stay and sit.

About 30 percent of Americans 6 years old and over are thought to live a completely sedentary lifestyle, meaning they do not engage in any [regular physical activity](#). And 8 in 10 American adults don't meet government recommendations for activities that build aerobic fitness and strength. Adults who are inactive pay \$1,437 more per year in health care costs than physically active adults.

That's a problem for all Americans. But it's a particular loss for the roughly 1 in 3 who are obese and who need to exercise if they are to drive down their added risk of diabetes and heart disease. Restoring their zeal for movement could go a long way to helping protect such people from those diseases. But knowing what's sapping that zeal may be a first

step.

More information: *Cell Metabolism*, Friend et al: "Basal ganglia dysfunction contributes to physical inactivity in obesity"
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