Is Taking a GLP-1 Drug Cheating?

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The question behind the question

People taking semaglutide or tirzepatide, sold as Ozempic, Wegovy, Mounjaro and Zepbound, often report a particular discomfort. Not about side effects or cost, but about legitimacy. They feel they are taking a shortcut around something they should have managed themselves. Friends and family sometimes say so out loud.

That feeling rests on an assumption worth examining: that body weight is under voluntary control, and that a medication which reduces appetite substitutes for effort the person should have supplied.

The assumption is wrong, and the reasons are not controversial.

Appetite is a regulated system, not a decision

Body weight is centrally regulated. Signals released from the gut, the pancreas, and fat tissue are integrated in the brain, mainly in the hypothalamus, and those signals govern food intake and energy expenditure.

Sumithran P et al., New England Journal of Medicine 2011;365:1597-1604.

This inverts the usual picture. Hunger is not generated by choice and then overridden by willpower. It is produced by a control system that receives hormonal inputs and sets the strength of the drive to eat. Conscious effort works on top of that system, not in place of it.

One of those inputs is glucagon-like peptide-1, usually written GLP-1. It is released by cells in the intestinal lining when food arrives. It slows the emptying of the stomach, increases the sensation of fullness, prompts the pancreas to release insulin, and suppresses the release of glucagon.

Madsbad S, Diabetes, Obesity and Metabolism 2014;16:9-21.

What happens when you lose weight by dieting

This is the finding that does the most damage to the willpower story, and it comes from a study with an unusually clean design.

Fifty people with overweight or obesity completed a ten-week very-low-energy diet and lost roughly 13.5 kg on average. Their appetite hormones were measured three times: before the diet, immediately after it, and again at 62 weeks.

A year after the weight loss, the hormonal picture had not returned to where it started. Leptin, peptide YY, cholecystokinin, insulin, ghrelin, gastric inhibitory polypeptide and pancreatic polypeptide all remained significantly different from their pre-diet values. Ghrelin, which drives hunger, remained elevated. So did the participants' own reported hunger.

Sumithran P et al., New England Journal of Medicine 2011;365:1597-1604 (n = 50).

A year after losing the weight, these people were hungrier than before they started. Nothing about their character had changed. Their physiology had.

The authors put it plainly: the signals that encourage weight regain do not revert after diet-induced weight loss, and long-term strategies are needed to counteract that change.

Related work found that the drop in energy expenditure after large weight loss can persist for years. The body defends its former weight from both directions. It burns less and it wants more.

Fothergill E et al., Obesity 2016;24:1612-1619.

Where GLP-1 fits

GLP-1 signalling runs weaker in people with obesity than in people whose weight regulation is working. This has been measured in a large standardized study spanning normal weight through obesity, and it holds whether or not the person has any problem with blood sugar.

Færch K et al., Diabetes 2015;64:2513-2525 (the ADDITION-PRO study, 1,462 participants).

So the signal that tells the brain a meal has arrived and fullness should follow is arriving faint.

What the medication does

Semaglutide and the related drugs activate the same receptor that natural GLP-1 activates. They are engineered to resist the enzyme that destroys natural GLP-1 within about two minutes, which is why they can be given weekly instead of by continuous infusion.

Functionally, the medication supplies a regulatory signal the body is not producing at sufficient strength. Appetite falls not because the person has acquired discipline but because the system that generates hunger is finally receiving the input it needs.

This is why the experience patients describe is so consistent, and so strange to them. The constant background negotiation about food goes quiet. That is not willpower arriving late. It is a control loop closing.

Why “cheating” does not apply

Set the biology aside for a moment. The concept itself does not fit.

Cheating means gaining an unfair advantage in a contest. Managing your own health is not a contest. There is no opponent, and no prize awarded for suffering more on the way to the same outcome.

Corrective lenses are not cheating at seeing. Insulin is not cheating at glucose regulation. A hip replacement is not cheating at walking. In each case a bodily function is not being performed adequately and a medical intervention performs it instead. Nobody asks the patient to first prove they tried hard enough without it.

Obesity attracts the accusation and short-sightedness does not. That distinction is not a scientific one. It is an inherited moral story about body weight that the physiology does not support.

What this means about stopping

The same explanation says something about how these drugs are likely to be used, and patients hear it as bad news when it is not.

If the medication supplies a signal the body is not producing at sufficient strength, then stopping returns you to the state in which it was not being produced. The weight comes back. This is not a failure of the drug, and it is not evidence that the loss was artificial.

It is what maintenance treatment looks like. Nobody expects blood pressure to stay down after the pills stop, or thyroid hormone to be a course you finish. Insulin is the closest parallel: a signal the body cannot supply in sufficient quantity, replaced for as long as that remains true.

Weight returning after you stop is the argument working, not failing. A signal that has to be supplied has to keep being supplied.

A natural question is whether a lower maintenance dose can hold the new weight. This is not yet established practice: no trial has tested a maintenance regimen, and guidelines are silent on it. Finding your maintenance dosage is something you need to do with your doctor's support.

If someone tells you it is cheating

Most of the time the remark is not an argument. It is a reflex, and it does not survive a question.

The useful question is what they think you are cheating at. There is no contest here, and once that is said out loud the accusation usually has nowhere to go.

If they want the substance, it fits in three sentences. Your body regulates weight with hormones, not with resolve. One of those hormones runs weak in people carrying extra weight. The medication supplies it, the way glasses supply focus a lens no longer provides.

And if they say the weight will come back if you stop: yes, that is how maintenance treatment works. It is true of blood pressure medication and of insulin, and nobody calls those cheating either.

You do not owe anyone a demonstration that you suffered enough first.

Sources

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World Health Organization. Guideline on the use of GLP-1 medicines in treating obesity. Geneva: World Health Organization; December 2025.

This paper is educational and is not medical advice. Decisions about starting, continuing, or stopping any medication belong with you and your physician.