National Eating Disorder Information Centre

BULLETIN


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# Insulin Resistance and Eating Disorders

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Eating disorders are a culmination of genetic, epigenetic, biological, psychological, sociocultural, and individual factors. Volumes have been written and said about the contribution of some of these factors, such as cultural messages about appearance ideals. In comparison, some factors have not been examined to a great extent. For example, what if an underlying physical dysfunction in a body – insulin resistance – significantly increases the risk of the onset of disordered eating behaviour? It's similar to the age-old question, "which came first – the chicken or the egg?" Which came first the eating disorder or the insulin resistance? Regardless of the answer, these two diagnoses have the possibility of occurring hand in hand.[1,2]

## WHAT IS INSULIN RESISTANCE?

According to research, metabolic syndrome diagnoses are on the rise. In 2012, it was estimated that at least 1/3 of Americans over the age of 18 meet the criteria for metabolic syndrome.[3] Metabolic syndrome is a compilation of abnormalities, including insulin resistance, abdominal fat accumulation, high blood pressure, and dyslipidemia (elevated triglyceride levels, elevated LDL cholesterol levels, and/or low HDL levels).[4] Insulin resistance occurs when the body's ability to metabolize and use carbohydrates is impaired – not completely broken, but dysfunctional. After a person consumes carbohydrates, as the carbohydrates travel down the digestive tract, they are broken down into glucose. In order for it to enter a cell, glucose needs to be paired with insulin, which acts like a "key" and "unlocks" the cell. In insulin resistance, the "key" – that is, insulin – is unable to "unlock" the cell, which leads to excess insulin and excess glucose in the blood stream, leaving the body lacking in energy, and craving carbohydrates because the glucose is not getting into the cells. If we think logically about this, carbohydrate or glucose is the primary source of fuel for almost every cell in the body, and it plays a vital role in the production of serotonin, a neurotransmitter involved in, among others, mood regulation, digestive system functioning, and perception of hunger and satiety. Insulin resistance therefore has the possibility of reducing patients' serotonin production, leaving them more prone to greater anxiety and depression, and less energy production, leading to more fatigue, and more carbohydrate cravings.[5]

Insulin-resistant bodies metabolically present as starving bodies. Mostly this occurs because individuals with insulin resistance often consume large amounts of carbohydrates, which the body is unable to use. We know that starving bodies break down lean body mass and store fat mass as a protective mechanism. So now we have a patient, typically in a larger body, that is in starvation mode, storing fat, and craving carbohydrates; this leads to eating disordered behaviours such as restriction, and bingeing, as well as negative emotions, such as shame and guilt, surrounding food and bodies.

1 Zuniga-Guajardo S, Garfinkel PE, Zinman B. Changes in insulin sensitivity and clearance in anorexia nervosa. *Metabolism*. 1986;35(12):1096-1100. doi:10.1016/0026-0495(86)90021-1

2 Hudson J, Javaras K, Pope H. The Challenges of Metabolic Syndrome in Eating Disorders. *Psychiatr* Ann. 2020; 50: 346-350. doi: 10.3928/00485713-20200713-02

3 Moore, J. X., Chaudhary, N., & Akinyemiju, T. (2017). Metabolic Syndrome Prevalence by Race/Ethnicity and Sex in the United States, National Health and Nutrition Examination Survey, 1988-2012. *Preventing chronic disease*, 14, E24.

4 Day C. Metabolic syndrome, or What you will: definitions and epidemiology. *Diab Vasc Dis Res*. 2007;4(1):32-38. doi:10.3132/dvdr.2007.003

5 Baranyi A, Amouzadeh-Ghadikolai O, von Lewinski D, et al. Revisiting the tryptophan-serotonin deficiency and the inflammatory hypotheses of major depression in a biopsychosocial approach. PeerJ. 2017;5:e3968. Published 2017 Nov 2. doi:10.7717/peerj.3968

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## INSULIN RESISTANCE AND BINGE EATING DISORDER

Let's take a look at some of the similarities between some binge eating disorder patients[6] and insulin-resistant patients. While it is important to note that not all individuals with binge eating disorder present with the same symptoms, it is common for binge eating disorder patients who may have underlying insulin resistance to present with the following symptoms.

| | |
|---|---|
| **BED patients** | • Hungry all the time<br>• Crave carboydrates<br>• Tired, fatigued, joint pain |
| **IR patients** | • Hungry all the time, or can go long periods of time without eating<br>• Crave carbohydrates<br>• Tired, fatigued, joint pain |

Individuals with binge eating disorder often struggle with strong guilt and shame, and use food to cope with their emotions. Many patients with binge eating disorder engage in daily restriction, which can lead to binge eating, but if they are insulin resistant, it is easy to keep using food as a tool for coping, as they rarely get full, which allows for large amounts of intake. Assessing for and treating underlying insulin resistance can be a valuable component in treating binge eating disorder. Equipping someone with an insulin resistant body with "off" switches, so it is able to signal that it is full, can help them start to employ healthier coping mechanisms versus only relying on food. Treatment also involves working with a multidisciplinary team to develop healthy coping skills.

## INSULIN RESISTANCE AND ATYPICAL ANOREXIA NERVOSA

Let's take a look at the similarities between some atypical anorexia patients and insulin resistant patients.[7] It is important to note that not all atypical anorexia patients exhibit these symptoms, and that each patient should be evaluated on an individual basis.[8]

| | |
|---|---|
| **Atypical AN patients** | • Restrict intake<br>• Larger body masks disordered behaviours<br>• Anxiety and depression due to lack of serotonin production |
| **IR patients** | • Ability to go long periods of time without eating or being hungry<br>• Gain weight easily, typically in a larger body or hallmarked by significant weight gain<br>• Anxiety and depression due to body not processing carbohydrates efficiently |

Individuals with atypical anorexia comprise the most underdiagnosed population with eating disorders. Could it be the case for some that they started using eating disordered symptoms as a result of their bodies being insulin resistant and not working normally? They are often the patients who have been sickest the longest due to their larger body size masking their symptom use. These patients often have high levels of shame instilled from the societal ideal of "thin", and have a very distorted idea of what a normal amount of food entails. Metabolically, these patients often present as underfed, partly because they are underfed, and, when insulin resistance is present, partly because of the body's inability to use what it is being given. While it is important to assess and treat these patients for insulin resistance, it is also important to encourage regular eating and help them understand that all bodies need and deserve food.

## DIAGNOSIS OF INSULIN RESISTANCE

There are several scientific methodologies to determine insulin resistance, including the Euglycemic Insulin Clamp, QUICKI (quantitative insulin sensitivity check index), or the HOMA-IR method (Homeostatic Model of Assessment for Insulin Resistance). The Euglycemic Insulin clamp is

6 Chao A, Grey M, Whittemore R, Reuning-Scherer J, Grilo CM, Sinha R. Examining the mediating roles of binge eating and emotional eating in the relationships between stress and metabolic abnormalities. *J Behav Med*. 2016;39(2):320-332. doi:10.1007/s10865-015-9699-1

7 Jaworski M, Panczyk M, liwczy ski AM, et al. A Ten-Year Longitudinal Study of Prevalence of Eating Disorders in the General Polish Type 2 Diabetes Population. *Med Sci Monit*. 2018;24:9204-9212. Published 2018 Dec 18. doi:10.12659/MSM.912253

8 Ilyas A, Hübel C, Stahl D, et al. The metabolic underpinning of eating disorders: A systematic review and meta-analysis of insulin sensitivity. *Mol Cell Endocrinol*. 2019;497:110307. doi:10.1016/j.mce.2018.10.005

the gold standard, but is also the most expensive test.[9] QUICKI and HOMA-IR are both validated methods, but the HOMA-IR is more widely used because it is the most affordable method available today. The HOMA-IR method uses a fasting glucose level and a fasting insulin level to determine the responsiveness of the insulin.[10]

$$\text{HOMA-IR} = \frac{\text{Fasting Glucose X Fasting Insulin}}{22.5}$$

The important thing to note is that even when the fasting insulin level and the fasting glucose level are both within the normal range, the HOMA-IR level can still indicate insulin resistance. The normal ranges specified below can vary between populations based on age and ethnicity.[11]

**Healthy Range:** 1.0 (0.5 - 1.4)

Less than 1.0 means insulin sensitive

Above 1.9 indicates early insulin resistance

Above 2.9 indicates significant insulin resistance

Other common factors that can, but do not always, co-exist with insulin resistance are Vitamin D and B-12 deficiencies, elevated inflammatory factors, dyslipidemia, elevated cholesterol levels, elevated triglyceride levels, and ovarian cysts or polycystic ovarian syndrome (PCOS).

## TREATMENT OF INSULIN RESISTANCE

A large part of the medical community believes insulin resistance is considered pre-diabetes and does not require pharmacological treatment, and instead can be managed via diet and exercise, until Hemoglobin A1C levels meet or rise above the threshold for a diabetes diagnosis. Insulin-resistant bodies do not metabolize food normally; with the impairment in their metabolic functioning, they are prone to weight gain, which is not readily countered even with restrictive dieting methods or intense exercise regimens. The weight gain that commonly occurs with insulin resistance can be distressing for affected individuals, especially when it occurs despite efforts to combat it. Untreated insulin resistance may therefore lead to or worsen eating disorder behaviours. Treatment often includes broad diet recommendations, specific exercises, and medications.

There are two main types of medications used to treat insulin resistance: metformin and glucagon-like peptide 1 receptor agonists (commonly referred to as the GLP-1 class). Metformin works to help correct the “unpairable” insulin, and allow glucose in the blood to enter cells, and it prevents the liver from overproducing unneeded glucose. The GLP-1 class of medications help glucose enter cells, so it is usable by the body, they reduce the rate at which the stomach empties, inhibit glucagon secretion, and increase insulin secretion in response to elevated blood sugar levels. It is rare for anyone to develop low blood sugars from either of these medications, because they work primarily on the insulin; however, in cases of food restriction, low blood sugars could occur. There are some side effects for each medication that should be noted. With metformin, there can be several gastrointestinal side effects, most commonly diarrhea and stomach upset, which often resolves after a few weeks. However, if these effects persist, switching to extended-release metformin can help, as it impacts the gastrointestinal tract less. GLP-1 medications by design induce gastroparesis, meaning food stays longer in the stomach, allowing for efficient carbohydrate absorption in the small intestine. GLP-1 side effects are often related to the gastroparesis – heart burn, and constipation, nausea, and vomiting.  Pancreatitis, while infrequent, can occur with GLP-1 administration, so it is key to assess for these symptoms as well. Diet recommendations include, with the least restrictions possible, balancing carbohydrates with protein, fat, and fibre to delay the rate of absorption, and providing sustained energy. By decreasing the glycemic index of the diet by balancing carbohydrates with protein, fat, and fibre, patients can experience evenly sustained energy versus quick bursts, and drops in energy. Some research suggests that weight training is highly effective at improving insulin sensitivity within the cell.[12] It is important to move one’s body within limits that incorporate recovery, preferences, and physical abilities.

9 Henderson M, Rabasa-Lhoret R, Bastard JP, et al. Measuring insulin sensitivity in youth: How do the different indices compare with the gold-standard method?. *Diabetes Metab*. 2011;37(1):72-78. doi:10.1016/j.diabet.2010.06.008

10 Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985;28(7):412-419. doi:10.1007/BF00280883

11 Salgado AL, Carvalho Ld, Oliveira AC, Santos VN, Vieira JG, Parise ER. Insulin resistance index (HOMA-IR) in the differentiation of patients with non-alcoholic fatty liver disease and healthy individuals. *Arq Gastroenterol*. 2010;47(2):165-169. doi:10.1590/s0004-28032010000200009

12 Shaibi GQ, Cruz ML, Ball GD, et al. Effects of resistance training on insulin sensitivity in overweight Latino adolescent males. *Med Sci Sports Exerc*. 2006;38(7):1208-1215. doi:10.1249/01.mss.0000227304.88406.0f

## CASE STUDY

Andy is a 17-year-old senior in high school, who has always participated in school sports and consumed a normal diet. Last spring when schools closed due to COVID-19, his physical activity level decreased, and he started to binge eat; he gained 30 pounds in 5 months. He attempts to limit his portions at meals, but often ends up eating double portions, and binges on sweets and candy in the evening. He would like some help getting his binge eating under control.

Metabolic profile via indirect calorimetry results show that Andy is hypometabolic and catabolic as a result of insufficient nutritional intake. Andy's protein turnover rate is above 15%, indicating lean body mass breakdown for energy in the absence of adequate nutrition. His body is starving; in turn, his fat substrate utilization, which should be at 85%, slowed down to 16%, as a means of body preservation. Carbohydrate substrate utilization should be 0% in the fasting state and Andy's is 61%, indicating a delay in which his body is using carbohydrates and possible insulin resistance. Note that all patients are fasting for minimum of 4 hours, prior to metabolic testing.

**Lab Values:**

**CBC** – Normal

**Glucose level** – 5.17 mmol/L, *normal*

**ALT** – 56 IU/L, *elevated*

**TSH** – 2.2 IU/L – *normal*

**Vitamin D** – 22.1, *low*

**Hemoglobin A1C** – 4.8, *normal*

**Insulin** – 15.3 mU/L, *normal*

**Cholesterol** – 4.74 mmol/L, *normal*

**Triglycerides** – 1.72 mmol/L, *elevated*

**HDL** – 0.85 mmol/L, *low*

**Non-HDL** – 3.89 mmol/L, *elevated*

**LDL** – 3.11 mmol/L, *elevated*

**Phosphorous** – 1.20 mmol/L, *low*

$$\text{HOMA-IR} : \frac{\text{Fasting Glucose X Fasting Insulin}}{22.5}$$

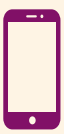
$$= \frac{5.17 \text{ X } 15.3}{22.5}$$

$$= \frac{79.1}{22.5}$$

$$= 3.5$$

**Result:** Andy's triglyceride levels are elevated, and are a direct reflection of the body's inability to process carbohydrates efficiently. In addition, he has an elevated HOMA-IR level at 3.5. Andy is insulin resistant; he is started on metformin and over-the-counter vitamin D3 supplement. His dietitian places him on a 2800-calorie meal plan that meets his energy needs and is balanced with adequate protein, carbohydrate, and fat. His treatment plan also includes incorporating movement he enjoys three times per week. On a two-week follow-up, the binge eating begins to decline. Andy has more energy, feels full and satisfied after meals, and is able to focus on his schoolwork better. The positive changes are sustained over time with this treatment plan. Andy continues to work with a therapist to improve his coping skills, but no longer identifies himself as a binge eater.

In summary, it is important to assess all patients with eating disorders for all possible compounding factors that could have an impact on recovery, as well as provide the patient with a better understanding of how their body is physically functioning and what their body needs to function best.



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