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# Eating disorders and carbohydrate metabolism interrelations

Współzależności zaburzeń żywienia i zaburzeń gospodarki węglowodanowej

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**Abstract**

**Introduction:** Eating disorders are characterised by persistent disturbances in eating behavior, resulting in severe carbohydrate homeostasis changes. The aim of the study is to review mutual correlations between eating disorders, with emphasis on anorexia nervosa, and carbohydrate metabolism, including glycemia and the levels of relevant hormones.

**Material and methods:** A priori general inclusion criteria were established and included patients with eating disorders or with glucose metabolism disorders. A MEDLINE database review was carried out. Relevant articles have been extracted and approved by supervisor.

**Results:** 40 studies got included in the review. Patients with active anorexia nervosa display low levels of fasting and postprandial glucose, decreased lipid metabolism, and decreased pancreatic endocrine activity. Insulin levels remain changed even after patient's re-nourishment – insulin response tends to be delayed and decreased compared to patients without anorexia history. Eating disorders are associated with poorer glycemic control and a higher percentage of diabetic complications in patients with pre-existing diabetes – mostly type I. There are also reports of higher carbohydrate metabolism disturbances among patients with eating disorders.

**Conclusions:** Carbohydrate metabolism disorders and eating disorders are clearly interrelated, although data on the nature of these relationships are still lacking. Treatment of eating disorders is not possible without normalizing eating patterns, thus also carbohydrate metabolism. However, it is not usual to monitor the mental state in terms of eating disorders potential development in patients during the treatment of carbohydrate disorders. According to existing data, this approach should be changed due to the risk of anorexia nervosa and other eating disorders in this group.

*Keywords*: eating disorders, anorexia nervosa, glucose metabolism disorders

**Streszczenie**

**Wstęp:** Zaburzenia odżywiania charakteryzują się uporczywie występującymi nieprawidłowościami zachowań żywieniowych, co skutkuje poważnymi zmianami w gospodarce węglowodanowej. Celem pracy jest przegląd literatury dotyczący wzajemnych zależności między zaburzeniami odżywiania się, ze szczególnym uwzględnieniem jadłowstrętu psychicznego i metabolizmu węglowodanów, w tym glikemii i poziomu hormonów regulujących gospodarkę węglowodanową.

**Materiał i metody:** Ustalono ogólne kryteria włączenia do niniejszego przeglądu, które obejmowały badania z udziałem

pacjentów z zaburzeniami odżywiania bądź zaburzeniami gospodarki węglowodanowej. Dokonano przeglądu literatury bazy MEDLINE. Wyodrębnione adekwatne artykuły zostały zatwierdzone przez osobę nadzorującą merytorycznie realizację przeglądu.

**Wyniki:** W przeglądzie uwzględniono 40 publikacji. Pacjenci z aktywnym jadłowstrętem psychicznym wykazują niskie stężenie glukozy w surowicy na czczo i poposiłkowej, osłabiony metabolizm lipidów i osłabioną aktywność endokrynną trzustki. Poziom insuliny pozostaje zmieniony nawet po ponownym odżywieniu pacjenta – odpowiedź insulinowa jest zwykle opóźniona i zmniejszona w porównaniu z pacjentami bez anoreksji. Zaburzenia odżywiania są związane z gorszą kontrolą glikemii i wyższym odsetkiem powikłań cukrzycowych u pacjentów z wcześniej rozpoznaną cukrzycą – głównie typu I. Istnieją również doniesienia o częstszym występowaniu i większym nasileniu zaburzeń metabolizmu węglowodanów wśród pacjentów z zaburzeniami odżywiania.

**Wnioski:** Zaburzenia metabolizmu węglowodanów i zaburzenia odżywiania są ze sobą wyraźnie powiązane, chociaż nadal brakuje danych na temat charakteru tych zależności. Leczenie zaburzeń odżywiania nie jest możliwe bez normalizacji wzorców żywieniowych, a co za tym idzie również metabolizmu węglowodanów. Nie jest jednak normą monitorowanie stanu psychicznego pod kątem potencjalnego rozwoju zaburzeń odżywiania u pacjentów w trakcie leczenia zaburzeń węglowodanowych. Według istniejących danych podejście to należy zmienić ze względu na ryzyko wystąpienia jadłowstrętu psychicznego i innych zaburzeń odżywiania w tej grupie.

*Słowa kluczowe*: zaburzenia żywienia, jadłowstręt psychiczny, zaburzenia gospodarki węglowodanowej

## Introduction

Eating disorders (ED) are a group of conditions manifested by persistent disturbances in eating behavior that negatively affects physical, psychological and social functions [1]. Despite the growing public awareness, the case number of ED worldwide was 55.5 million, or 7.2 per 1000 people in 2019, the incidence remains on the rise as well [2,3]. Anorexia nervosa (AN), which is one of the most often observed ED, is defined as a restriction of caloric intake in relation to the demand, leading to a significant reduction in body weight in terms of age, gender, developmental stage and physical health, most often caused by an obsessive fear of weight gain, obesity, as well as persistent behavior that prevents weight gain, even with significantly low body weight, and disturbances in the perception of own weight and body image, excessive influence of body weight on self-esteem, or ignorance of the seriousness of the weight deficiency [1]. The short- and long-term effects of ED are clearly reflected in the DALY index (Disability Adjusted Life Years - represents the loss of the equivalent of one year of full health), which for ED is globally 6.6 million (85.9 per 100,000 people) [3]. Psychopathological comorbidities of ED are very common, and include mood disorders, depressive disorders, anxiety disorders, obsessive-compulsive disorders, attention deficit hyperactivity disorder (ADHD), personality disorders, substance addiction, borderline disorders. Positive correlations between anorexia and schizophrenia have been shown as well [4,5]. One of the many somatic consequences of anorexia nervosa, due to the restriction of caloric intake, are disturbances in the carbohydrate metabolism of a mainly deficient nature - hypoglycemia, and hormonal disorders which do not necessarily reverse after the patients are nourished.

## Materials and methods

A priori general inclusion criteria were established and included research and review papers with patients with ED or with glucose metabolism disorders. A MEDLINE database review was carried out. The search query was as follows: "anorexia nervosa[MeSH Terms] AND "glucose metabolism disorders"[MeSH Terms] OR "insulin resistance" OR insulin OR glucagon OR hyperglycaemia OR hypoglycaemia OR hyperinsulinism OR glycosuria. The search resulted in 1041 records found. Relevant articles have been extracted by 4 researchers independently. The questionable cases varying between sets of different researches were examined and approved by the supervisor of the study. Two papers were obtained from different sources. The number of 40 records got included in the final review.

## Carbohydrate homeostasis in active AN

Low glucose levels in chronically malnourished patients with AN determine the production of compensatory mechanisms ensuring a constant supply of energy materials to the body tissues [6]. However, these mechanisms are partially exhausted after some time, which may result in a series of disturbances in carbohydrate metabolism. JT Alderdice et al. in 1985 were one of the first to assess hormones in the active phase of anorexia. After an overnight fast, pancreatic glucose, insulin, and glucagon levels were significantly lower in anorexic patients than in age- and sex-matched healthy

controls. Two hours post-meal blood testing revealed significant impaired glucose tolerance, decreased and delayed insulin response, and decreased release of gastric inhibitory polypeptide in patients [7]. It has been shown - which was later confirmed in other studies - that patients with AN display not only low fasting glucose, but also low postprandial glucose compared to healthy patients [8,9]. These dysregulations may be caused not only by glucose increased use in tissues, but also by significantly reduced lipid metabolism [9], which most likely is an expression of the intensified restoration of the body's energy reserves to compensate for malnutrition. Low postprandial glycemia cannot be explained by insulin levels in anorexic patients, which also remain low both in fasting and nourished patients, due to, among other things, the extremely low body fat in these patients. Low insulin levels and postprandial glycemia result in high insulin sensitivity as measured by the Homeostatic Model Assessment – Insulin Resistance (HOMA-IR) index. Interestingly, insulin sensitivity is quite different in other ED. It is also worth adding that insulin sensitivity in AN is modifiable and is influenced by the value of the BMI index. [10–13]. Unfortunately, there are no studies checking this phenomenon with the more objective method such as glucose clamp technique.

One of the hypotheses of the AN development causes was low constitutive fasting insulin level. Broberg et al. in 1988 compared the insulin secretion efficiency of the brain phase between AN patients and a healthy control group. Fasting patients were presented with a visually attractive meal, then, after 5 minutes, the level of secreted insulin was measured, which turned out to be even higher in the AN group compared to the control group, which disproved the hypothesis [14]. Insulin antagonist glucagon also shows abnormal levels in active anorexia. The level of fasting glucagon is much lower in AN patients compared to the control group, and its release after the supply of energy (in the form of a meal and a glucose load test) is much higher, and after several minutes it shows a significant decrease [13].

**Carbohydrate metabolism in AN patients after renourishment**

Renourishment is a factor that impacts glycemic homeostasis in patients with anorexia. D. R. Counts et al. showed that significantly reduced serum Insulin-like Growth Factor I (IGF-I) and Insulin-like Growth Factor Binding Globulin (IGFBP) 3 levels returned to near-normal levels after refeeding anorexic patients [15]. Fasting Growth Factor (GH) and serum IGFBP-1 and IGFBP-2 levels were significantly increased in low body weight patients with anorexia nervosa and also returned to near-normal levels upon re-nutrition. This indicates that malnutrition alters the GH-IGF axis and that it is reversible with re-nutrition, as also noted by L. Gianotii et al. [16], but not always, as indicated by the study by J. Argente et al. [17]. They tested a group of 50 patients with anorexia before and after re-nourishment in which IGF-1 and IGFBP-3 remained decreased regardless of nutritional status. L. Audi et al. [18] and L. Caregaro et al. [19] also emphasized the positive correlation of IGF-1 with BMI, and suggested its possible use as a nutritional marker in ED. Normal fasting glucose levels were observed by N. W. Brown et al. [20] in both healthy individuals and re-nourished AN patients, while fasting insulin was lower in patients who recovered from anorexia, and the fasting glucose / insulin ratio was higher in the treated group. Since the insulin response to the meal was delayed, this may suggest a permanent change in pancreatic function as a result of anorexia. Similar conclusions were reached by M. Kumai et al. [21], which additionally checked the level of glucagon, which turned out to be reduced after glucose consumption in both the control group and patients after treatment. G. A. Heruc et al. [8] included patients after two weeks of re-nourishment in their study - in this group, postprandial glycemia remained low, and glucagon and GLP-1 high with no differences in baseline glucagon and postprandial insulin compared to healthy individuals. K. P Kinzig et al. [22] noticed that the delay in insulin release and increased levels of the pancreatic polypeptide (PP) do not get corrected after short-term (2 weeks) re-nutrition and therefore may contribute to the high relapse rate and persistence of anorexia nervosa. In the study by T. Nakahara et al. [23] with patients after treatment and improved nutritional status, the glucose and insulin responses were normalized. Women with anorexia nervosa developed visceral obesity related to insulin resistance after partially regaining weight. Y. Kim et al. [24] assessed 24 women with anorexia who had abnormal HOMA-IR after weight restoration, but mean HOMA-IR between healthy and AN group did not differ significantly. The results suggest that the glucose response in women with AN changes due to visceral adipose tissue after regaining full body weight, but they do not develop overt insulin resistance, as also pointed out by A. Prioletta et al. [25]. In the study by D. Modan-Moses et al. [26] the decreased insulin sensitivity was confirmed by a significant increase in HOMA-IR in patients undergoing anorexia treatment. In summary, the increase in body weight and BMI in patients with anorexia nervosa has various consequences in terms of carbohydrate metabolism, and therefore cannot be considered the only goal of anorexia treatment [17,20]. Future research should focus on whether glycemic disturbances in patients with anorexia nervosa are associated with long-term medical risks. Moreover, most of the research conducted so far has

focused primarily on the group of women, which also sets the direction of scientific research in the future.

**Management of previously diagnosed carbohydrate disorders in AN**

ED, including anorexia and bulimia, have been reported in people with type I diabetes. The estimated frequency of occurrence varies depending on the type and methods of research, but in most cases the incidence does not differ from the population, and is estimated to occur in around 15 in 100 000 people [27]. The causes for the development of anorexia or bulimia in people with diabetes are thought not to differ from patients without carbohydrate disturbances. According to Reinehr et al., the development of ED is significantly associated with the later age of diabetes onset and not using the insulin pump. The most common measures to achieve a lower body weight among patients with diabetes are deliberate skipping of insulin doses and episodes of fasting. This tendency is associated with poorer glycemic control and a higher percentage of diabetic complications - ketoacidosis or vascular complications [28–30]. According to Biggs M et al., skipping insulin injections can also have a significant impact on the development of ED – insulin withholders compared with patients which took insulin regularly exhibited higher rate of pathological scores on the Eating Disorder Inventory 2, as well as higher probability to report current or past symptoms of AN or bulimia nervosa [31]. The Scheuing et al. 2014 study proved that after taking into account the age, sex and duration of diabetes, patients with ED present higher levels of glycosylated hemoglobin by about 0.5-1 mmol/l than the control group without ED, a higher percentage of incorrect insulin injection sites by about 20%, and approximately twice as high as ketoacidosis hospitalizations. Moreover, the number of days spent in hospital by AN patients due to complications is also longer compared to control group (11.31 ± 0.21 vs. 4.81 ± 0.01 days per year) [32].

**The development of eating disorders in patients with impaired carbohydrate metabolism**

ED can also present as a consequence of carbohydrate metabolism abnormalities. According to Al Hourani et al., who conducted a study on 497 adolescents from Jordan, the disorders are much more common in patients diagnosed with diseases related to glucose metabolism than in the healthy population – 35.7% vs. 25.0% [33]. However, AN is not the dominant disorder in this group of patients. The ED are most often observed in type 1 diabetes, which is why a common term was created to describe the coexistence of these two disorders. Nevertheless, this is not an accurate wording due to the fact that not all diabetes with ED exhibit symptoms characteristic of bulimia nervosa, contrary to what the proposed name suggests. ED in people suffering from type 1 diabetes can be divided into two groups. The first of them includes diseases diagnosed according to the specific symptoms, such as anorexia nervosa, bulimia nervosa, binge-eating disorder, pica (eating or craving of things that are not food), and rumination (regurgitation of meals after consumption). The second type known as disordered eating symptoms includes dieting for weight loss, binge eating, self-induced vomiting, excessive exercise, and laxative or diuretic use [34]. The most common is binge eating disorder, while AN is quite rare [33,35]. According to the available studies, the problem occurs mainly in young patients, up to about 5 years after the onset of symptoms and diagnosis of type 1 diabetes, but the first symptoms indicating ED can be observed even after 2 years [28]. The most vulnerable group are people between 7 and 18 years of age [22], and according to Larrañaga et al., mainly women (37.9% of women vs. 15.9% of men) [29]. Additionally, it was noted that girls without migration experience had a 2.8 times higher risk of developing disordered eating behavior than those who experienced a change of place of residence [28]. A serious problem among young patients with type 1 diabetes is that they tend to deliberately take inadequate insulin doses to control their own body weight. The reasons for such behavior include: diagnosis of diabetes itself which is stressful for a young person, a sense of lack of control over his own body, bad relationships with caregivers, and above all, numerous restrictions resulting from the treatment of diabetes, which may provoke binge eating and even be the reason for self-harm [35]. The incorrect insulin therapy increases the risk of dehydration, a reduction in the percentage of muscle tissue in the body, and the possibility of developing infections, fatigue, and even kidney failure or blindness due to vascular damage [30,36,37]. For this reason, extensive screening is essential. For diagnosis, the following are used: Diabetes Eating Problem Survey (DEPS-R), the Eating Disorder Inventory 3 (EDI-3) (adapted for Polish patients) [34] which has been modified specifically for patients with type 1 diabetes [38], and the modified SCOFF (mSCOFF) questionnaire [39]. However, according to Markowitz, J T et al., asking the question "Have you ever been overweight?" may prove to be a very effective tool if clinicians do not have time to complete the diagnostic process in detail (83% sensitivity and 94% negative predictive value) [40]. Taking all this into account, the treatment of patients facing these problems should be carried out by an interdisciplinary team and be characterized by a holistic approach - less restrictive glycemic control, adjusting the acceptable blood glucose level to the patient's condition instead of following the optimal norms in a population without disorders and considering individual or family psychotherapy [29,34].

Despite many observational studies, the exact number of people with ED among patients with type 1 diabetes is still unknown. Moreover, there is a lack of epidemiological data on the prevalence of these disorders in people with other types of diabetes, and most of the research conducted focuses primarily on the group of women.

## Conclusion

ED as a mental determinant of the limited supply of nutrients is a factor that disturbs the organism's homeostasis, including carbohydrate homeostasis. Anorexia is not only the cause, but also the effect of carbohydrate disturbances, therefore this relationship should be emphasised. It is impossible to treat anorexia without compensating for carbohydrate disorders, and special attention should also be paid to the possibility of ED development in diabetes. Much is unknown about the metabolic basis of these interactions, and attempts to answer these questions should be one of the directions of future research.

## Conflict of interest

The author has declared no conflict of interest.

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