
**Risk Factors and Comorbid Conditions Associated with Insulin Resistance:
Results from a Cross-sectional Study**

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Abstract

Insulin resistance (IR) contributes to adverse metabolic and cardiovascular conditions, yet population data from Southeastern Europe are limited. We conducted a cross-sectional online survey of adult participants from Bosnia and Herzegovina (n = 115) to assess self-reported IR prevalence and its associations with demographic, lifestyle, dietary, and metabolic/clinical factors, as long-term medication use. Associations were tested using chi-square or Fisher's exact tests. Among 115 participants, 64 (55.7%) self-reported insulin resistance, which was not significantly higher than the expected reference value of 50% (one-sample proportion test, $p = 0.1316$). IR was significantly associated with age ($p = 0.016$), vitamin D deficiency ($p = 0.0013$), reporting at least one diagnosed condition ($p = 0.0043$), and hormonal medication use ($p = 0.0032$). Participants with IR were more likely to report frequent whole-grain intake ($p = 0.0078$) and less likely to report frequent refined-carbohydrate intake ($p = 0.0381$), patterns that may reflect post-diagnosis dietary changes. Symptoms more common in IR included sudden weight gain ($p < 0.0001$), increased hunger ($p = 0.0091$), sleepiness after meals ($p = 0.0491$), and fatigue ($p = 0.0497$). No significant associations were observed for sex or BMI. While low physical activity, chronic stress, and poor sleep were more common in the IR group, none reached statistical significance. In this population-based sample, IR clustered with younger age, vitamin D deficiency, reported comorbidity, specific symptoms, and hormonal medication use. These findings underscore the potential value of early screening and targeted health education and indicate the need for larger, longitudinal studies with detailed exposure measurement to confirm these associations.

Keywords: Insulin resistance, risk factors, cross-sectional study, comorbidities

1. Introduction

The impaired physiological response of target tissues to insulin stimulation is known as insulin resistance (IR). Although IR can develop in any insulin-sensitive tissue, it primarily affects the liver, skeletal muscle, and adipose tissues (Freeman & Pennings, 2018). In insulin-resistant states, these tissues respond inadequately to insulin, leading to elevated blood glucose levels and

compensatory increase in insulin secretion by pancreatic β -cells. While initially compensatory, hyperinsulinemia can worsen IR, disrupting the coordination between β -cell function and insulin action, which contributes to the development of type 2 diabetes (Schinner et al., 2005).

IR development is multifactorial, involving oxidative stress, inflammation, and a combination of genetic, behavioral, environmental, and epigenetic factors (Ndisang et al., 2017). Key demographic and lifestyle risk factors include age, hormonal changes, abdominal obesity, and physical inactivity (Fahed et al., 2020). Aging is consistently associated with decreased insulin sensitivity, independent of body mass index (Oya et al., 2014; Karakelides et al., 2010). Differences in fat distribution and hormonal profiles may explain the variations in IR between men and women (Ciarambino et al., 2023; Geer & Shen, 2009). Lack of sleep and disrupted sleep patterns, chronic psychological stress, and sedentary behavior contribute to IR by disrupting neuroendocrine function and promoting inflammation (Gratas-Delamarche et al., 2014; Yan et al., 2016; Li et al., 2013; Spiegel et al., 2005). Environmental exposure to pollutants and heavy metals has also been implicated in metabolic dysfunction (Hyman, 2010; Kelishadi et al., 2009).

Numerous studies have shown that nutrition significantly affects insulin resistance. Diets high in saturated fats and refined carbohydrates increase IR risk, whereas fibre-rich foods, low glycemic index carbohydrates, vegetables, and low-fat dairy products are linked with lower insulin resistance (Zuo et al., 2013; Martins & Conde, 2022; Gołabek & Regulska-Ilow, 2019). IR may even affect underweight individuals due to inadequate protein intake, leading to impaired β -cell function (Salihefendic, 2023).

In general, IR can manifest as a variety of clinical symptoms, including fatigue, increased hunger, difficulty in losing weight, darkened skin patches (particularly on the neck and armpits), mental health disturbances, and cognitive decline (Mestre Font et al., 2023). Clinically, IR is central to many metabolic and cardiovascular disorders, including metabolic syndrome, polycystic ovary syndrome, non-alcoholic fatty liver disease, and certain cancers (Lebovitz, 2001; Kumar et al., 2023; Zhao et al., 2023). Emerging evidence indicates that the prevalence of insulin resistance in type 1 diabetes is increasing, despite its historical association with type 2 diabetes (Ndisang et al., 2017). As insulin resistance typically develops years before these conditions appear, early detection and intervention are crucial for preventing more serious health problems (Rao, 2001).

Hormonal imbalances, including those involving thyroid hormones, cortisol, and androgens, may reduce insulin sensitivity (Rogowicz-Frontczak et al., 2017; Xu & Qiao, 2022; Gierach et al., 2014; Gür et al., 2015). Deficiencies in vitamin D, magnesium, zinc, and vitamin B12 have also been associated with impaired insulin function (Cigerli et al., 2016; Chutia & Lynrah, 2015; Cruz et al., 2018). Infections and chronic inflammation also contribute to IR pathogenesis through immune and hormonal disruption (Aydemir et al., 2005; Romero-Gómez, 2006; Lazar et al., 2021). Certain medications, including blood pressure drugs, contraceptives, corticosteroids,

antipsychotics, β -blockers, and HIV treatments, can induce IR through various metabolic pathways (Zaman, 2020).

Despite increasing global awareness of insulin resistance, limited data are available on its prevalence and associated factors in Southeastern Europe. This study aimed to assess the self-reported prevalence of IR and its potential associations with demographic, lifestyle, dietary, metabolic, and clinical factors in a Bosnian adult population using a cross-sectional survey.

2. Methods

Sample collection

The study was conducted as a cross-sectional survey in the population of Bosnia and Herzegovina. Data were collected through an anonymous online questionnaire over two months. Participation was voluntary. The inclusion criteria required participants to be aged 18 years or older, capable of providing reliable self-reported health information, and either diagnosed with insulin resistance or with at least one potentially related condition, including diabetes mellitus, cardiovascular disease, hypertension, general or central obesity, polycystic ovary syndrome (PCOS), dyslipidemia, non-alcoholic fatty liver disease (NAFLD), or cancer. The questionnaire included sections on demographic characteristics, lifestyle risk factors, specific dietary patterns, metabolic and clinical risk factors, and long-term medication use.

This study was approved by the Ethics Committee of the Department of Genetics and Bioengineering, International Burch University. Prior to participation, all respondents were provided with information regarding the study's objectives and procedures and were informed that participation was entirely voluntary and anonymous. Informed consent was obtained electronically before the completion of the online questionnaire. No personally identifiable information was collected, in accordance with ethical guidelines to protect the confidentiality of the participants.

Statistical analysis

Initial data preprocessing was performed using Google Sheets. This included the removal of duplicate or incomplete responses and the exclusion of participants who did not meet the predefined inclusion criteria. Body Mass Index (BMI) was calculated using self-reported height and weight using the following formula:

$$\text{BMI} = \text{weight (kg)} / [\text{height (m)}]^2.$$

Based on these values, the participants were categorized into standard BMI groups: underweight, normal weight, overweight, and obese.

The final dataset was imported into RStudio (R version 4.1.2) for statistical analysis. Each questionnaire variable was analyzed separately. Demographic characteristics, lifestyle behaviors,

dietary habits, metabolic factors, clinical conditions, and long-term medication use were examined in relation to the self-reported insulin resistance status.

Descriptive statistics were used to summarize the data, and contingency tables were constructed to examine the distribution and relationships between categorical variables. To facilitate interpretation, selected variables such as BMI and dietary habits were recoded into grouped categories (e.g., frequent vs. infrequent consumption).

Associations between categorical variables and IR status were tested using the Chi-square test. Fisher's exact test was used for variables with small sample sizes in individual cells to ensure statistical accuracy. When statistically significant associations were found, odds ratios (ORs) were calculated to quantify the strength and direction of the relationship between specific factors and IR. In addition, a one-sample proportion test was used to determine whether the observed proportion of participants reporting IR (55.7%) was significantly different from the expected proportion (50 %).

This approach enabled the identification of statistically significant trends and potential risk factors associated with insulin resistance in the study population.

3. Results

After applying the exclusion criteria, 115 participants were included in the study. All participants were adults (≥ 18 years) who reported a diagnosis of insulin resistance and/or at least one related condition listed in the survey. The sample was predominantly female, comprising 98 (85.22%) women and 17 (14.78%) males. Participants were categorized into three age groups: 45 individuals (39.13%) were aged 18–34 years, 48 (41.74%) were aged 35–54 years, and 22 (19.13%) were aged 55 years or older.

A statistically significant difference in the prevalence of insulin resistance was observed across age groups ($p = 0.016$), with the youngest group (18–34 years) showing the highest prevalence of insulin resistance. In contrast, no statistically significant difference in the prevalence of insulin resistance was found between the sexes ($p = 0.299$), indicating no strong evidence of sex-related variation. However, it is important to acknowledge that the sample was predominantly female, which may limit the ability to detect significant sex-based differences and affect the generalizability of the results. Additionally, no statistically significant difference in insulin resistance prevalence was found across BMI categories ($p = 0.368$), suggesting that insulin resistance did not vary significantly by BMI within this sample.

Table 1. Distribution of Insulin Resistance by Demographic Characteristics: Age, Sex, and BMI

Category/Risk Factor	Total Number of Participants	IR Diagnosed	IR not Diagnosed	p-value
Age groups:				
18 - 34	45	31	14	0. 016
35 - 54	48	26	22	
55+	22	7	15	
Sex:				
Female	98	57	41	p = 0.299
Male	17	7	10	
BMI				
Underweight	1	0	1	p = 0.368
Normal	38	20	18	
Overweight	46	24	22	
Obese	30	20	10	

An analysis was conducted to examine the relationship between self-reported lifestyle factors and insulin resistance prevalence using Fisher’s exact test. Although none of the evaluated lifestyle factors showed a statistically significant association with insulin resistance in this sample, both low physical activity and chronic stress demonstrated a trend toward significance ($p = 0.0881$ and $p = 0.0886$, respectively). Also, other factors, including poor sleep quality, exposure to environmental toxins, and alcohol consumption, did not show statistically significant associations.

Table 2. Distribution of Lifestyle Factors by Insulin Resistance Status

Lifestyle risk factor	P-value	With insulin resistance	Without insulin resistance
Low physical activity	0.0881	42	25
Chronic stress	0.0886	41	24
Poor sleep quality	0.2644	32	20
Exposure to toxins	0.3246	19	20
None of the risk factors	0.3803	4	1
Alcohol consumption	1.0000	1	1

To explore the relationship between dietary patterns and insulin resistance, the participants' frequency of food intake was recorded on a four-point scale: 1 = never, 2 = rarely, 3 = often, and 4 = daily. For a clearer analysis, responses were recoded into two categories: "Frequent" (3–4) and "Infrequent" (1–2). Fisher's exact test was used because of the small subgroup sizes.

Ten food groups were analyzed: refined carbohydrates, beverages, whole grains, fibre-rich foods, protein-rich foods, healthy fats, processed foods, desserts, dairy products, and low-fat options. Statistically significant associations were found for whole grain intake ($p = 0.0078$) and refined carbohydrate intake ($p = 0.0381$). Cross-tabulations indicated that participants with insulin resistance were more likely to report frequent consumption of whole grains (44 IR vs. 22 non-IR) and less likely to report a frequent intake of refined carbohydrates (28 IR vs. 33 non-IR). This unexpected direction of association may reflect dietary modifications following diagnosis. Other food groups showed no statistically significant associations with insulin resistance.

Analysis of specific dietary patterns showed a near-significant association ($p = 0.051$), with an odds ratio of 2.60. Among the 29 participants who followed a specific diet, 21 had insulin resistance. Although this result did not reach statistical significance, it may suggest post-diagnosis dietary adjustments among insulin-resistant individuals.

To explore the relationship between self-reported symptoms and insulin resistance, participants were asked to indicate any symptoms they experienced, allowing for multiple responses. Each symptom was analyzed individually using Fisher's exact test. Statistically significant associations were observed between several symptoms. Sudden weight gain, particularly in the abdominal region, was most strongly associated with insulin resistance ($p < 0.0001$), with 46 participants with insulin resistance reporting this symptom compared with 16 without. Increased hunger ($p = 0.0091$), sleepiness after meals ($p = 0.0491$), and fatigue ($p = 0.0497$) were also

significantly more common in insulin-resistant participants. In contrast, other symptoms, such as hypoglycemia episodes, increased thirst, slow wound healing, brain fog, blurred vision, frequent urination, and dark patches on the skin, did not show statistically significant differences between the groups. These findings suggest that certain metabolic symptoms may serve as clinical indicators of insulin resistance, whereas others may be less reliable for identifying affected individuals.

Table 3. Symptoms of insulin resistance reported by insulin-resistant vs. non-insulin-resistant research participants

Symptom	p-value	Insulin resistance	No insulin resistance
Sudden weight gain	<0.0001	46	16
Increased hunger	0.0091	41	20
None of the symptoms	0.0153	0	5
Sleepiness after meals	0.0491	47	28
Fatigue	0.0497	57	38
Hypoglycemia episodes	0.0550	31	15
Increased thirst	0.1076	25	12
Slow wound healing	0.1084	9	2
Frequent urination	0.6837	18	17
Blurred vision	0.6931	23	16
Brain fog	0.7049	40	30
Dark patches on skin	0.7969	11	7

Among the 115 participants included in this study, 64 (55.7%) self-reported a diagnosis of insulin resistance. A one-sample proportion test was conducted to assess whether the observed proportion was significantly higher than the expected reference value of 50%. The results were not statistically significant ($p = 0.1316$). This means that although more than half of the participants reported insulin resistance, the difference was not large enough to be considered significant.

Insulin Resistance Status

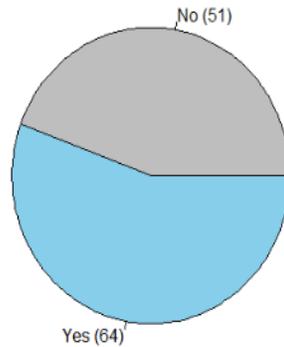


Figure 1. Proportion of insulin-resistant vs. non-insulin-resistant research participants

To examine the association between insulin resistance and specific self-reported clinical conditions, Fisher's exact tests were conducted, and odds ratios were calculated. Among the listed conditions, cardiovascular disease (OR = 0.25, $p = 0.0181$) and hypertension (OR = 0.41, $p = 0.0286$) were significantly more common in participants without insulin resistance. These findings may be due to sampling variations or unmeasured factors. Central obesity showed a trend toward significance ($p = 0.0524$), suggesting a possible association with insulin resistance, although this association did not reach statistical significance. Other conditions, including PCOS and type 2 diabetes, appeared more common in insulin-resistant participants, but the differences were not statistically significant in this sample.

Table 4. Diagnosed diseases reported by insulin-resistant vs. non-insulin-resistant research participants

Disease	p-value	Odds ratio(OR)	IR	No IR
None of the diseases	0.0011	More likely with IR	11	0
Cardiovascular diseases	0.0181	Less likely with IR	5	13
Hypertension	0.0286	Less likely with IR	15	22
Central obesity	0.0524	More likely with IR	12	3
PCOS	0.820	More likely with IR	28	14
Diabetes type 2	0.2948	More likely with IR	7	2
NAFLD	0.5793	Less likely with IR	7	8
Diabetes type 1	0.6539	Less likely with IR	2	3
Obesity	1.0000	Less likely with IR	19	16
Dyslipidemia	1.0000	Less likely with IR	12	10

Fisher’s exact test was used to examine the relationship between the presence of any diagnosed disease and insulin resistance. A statistically significant association was found ($p = 0.0043$), indicating that participants who reported at least one diagnosed condition were significantly more likely to have insulin resistance. Only nine participants with insulin resistance did not report any additional diagnosed diseases. These findings suggest that insulin resistance in this sample was closely linked to the presence of other clinical conditions and highlight the importance of screening for insulin resistance even in individuals without diagnosed conditions.

The relationship between self-reported nutrient deficiencies and insulin resistance was analyzed using Fisher’s exact test. Vitamin D deficiency was significantly associated with insulin resistance ($p = 0.0013$, $OR = 3.58$). In contrast, the absence of deficiencies ($p = 0.0395$, $OR = 0.44$) and vitamin B12 deficiency ($p = 0.0432$, $OR = 0.28$) were more common in participants without insulin resistance. Vitamin K and omega-3 deficiencies were reported only in the non-insulin-resistant group, though the sample size was small. Magnesium, zinc, and chromium showed no significant differences ($p = 1.000$). These results suggest a possible link between vitamin D deficiency and insulin resistance in our sample.

The association between self-reported hormonal imbalance and insulin resistance was examined using Fisher’s exact test. None of the individual conditions reached statistical significance ($p > 0.05$). However, the odds ratios indicated a non-significant trend toward more prevalent insulin

resistance in participants with high androgens/PCOS ($p = 0.146$), hypothyroidism ($p = 0.334$), and hyperthyroidism ($p = 0.628$). These findings suggest possible associations that should be explored in future studies with larger sample sizes.

Fisher’s exact test was used to examine the associations between self-reported infections, inflammatory conditions, and insulin resistance. None of these differences reached statistical significance ($p > 0.05$). Gut microbiota imbalance was reported only in insulin-resistant participants ($p = 0.1281$), while autoimmune diseases ($p = 0.4142$) and periodontal disease ($p = 1.000$) were also more frequent in this group. Although these patterns were not statistically significant, they warrant further investigation.

The association between medication use and insulin resistance was analyzed using Fisher’s exact test. The use of hormonal medications was significantly associated with a higher likelihood of insulin resistance ($p = 0.0032$). Other medications, including beta-blockers, corticosteroids, statins, and antipsychotics, were not significantly associated with insulin resistance. These findings highlight a potential link between hormonal therapy and insulin resistance in this cohort.

Table 5. Associations between clinical factors and insulin resistance

Variable category	Specific variable	p - value	Significant($p < 0.05$) in IR cases
Diagnosed disease	At least one disease diagnosed	0.0043	Yes
Nutrient deficiencies	Vitamin D	0.0013	Yes
	Vitamin B12, Magnesium, Vitamin K, Omega-3 fatty acids, Zinc, Chromium	$p > 0.05$	No
Hormonal imbalances	High androgens, Hypothyroidism, Hyperthyroidism, High cortisol, Acromegaly	$p > 0.05$	No
Infections and inflammations	Disbiosis, Autoimmune diseases, Hepatitis C infection, Helicobacter pylori infection, Periodontal disease	$p > 0.05$	No
Medication use	Hormonal medication	0.0032	Yes
	Beta-blockers, Corticosteroids, Statins, Antipsychotics	$p > 0.05$	No

4. Discussion

This study aimed to explore a range of factors potentially associated with self-reported insulin resistance in a sample of 115 adults. Several statistically significant associations were identified, offering potential insights into the factors linked to IR and highlighting areas for further investigation.

Our finding that the youngest adult group (18–34 years) had the highest prevalence of insulin resistance aligns with the NHANES data, which showed that approximately 40–45% of U.S. adults aged 18–44 years have insulin resistance (Parcha et al., 2022). No significant sex differences were found; however, the predominantly female sample (85%) limited the ability to detect sex-specific trends. This imbalance may have influenced associations related to conditions more prevalent in women, such as PCOS, hormonal imbalances, and the use of hormonal treatments, as well as potential postdiagnosis dietary changes. Similarly, BMI did not show a statistically significant association with IR. This may reflect the limitations of BMI as an indicator of metabolic risk, especially in populations in which body composition or fat distribution may differ significantly.

Although none of the lifestyle factors reached statistical significance, low physical activity and chronic stress showed trends toward an association with IR. This aligns with evidence indicating that physical inactivity contributes to metabolic dysfunction, including reduced insulin sensitivity, whereas regular exercise enhances glucose metabolism and insulin sensitivity (Małkowska, 2024). Moreover, chronic or repeated psychological stress has been implicated in IR development (Gianotti et al., 2021). Given that many non-IR participants in our sample also reported these lifestyle risk factors, such patterns may indicate a heightened risk of developing insulin resistance in this population. These trends, although not statistically significant in our study, underscore the value of further investigation in larger longitudinal studies.

In terms of dietary patterns, the unexpected finding that participants with IR reported more frequent consumption of whole grains and less frequent intake of refined carbohydrates likely reflects post-diagnosis dietary changes. A randomized controlled trial found that whole-grain diets can improve insulin sensitivity and glucose metabolism in adults at risk of type 2 diabetes (Malin et al., 2018). However, longitudinal studies are needed to determine whether such dietary habits precede or follow IR diagnosis. Additionally, a non-significant trend was observed among participants who reported following specific diets, possibly indicating behavioral modifications following an IR diagnosis.

Among the self-reported symptoms, several were significantly associated with insulin resistance. Notably, abdominal weight gain, increased hunger, fatigue, and post-meal sleepiness were more common among participants with IR. These findings are consistent with previous research showing that weight gain from early adulthood to middle age strongly increases the risk of insulin resistance syndrome (Everson et al., 1998). In addition, experimental studies in animal models have revealed that repeated hunger can induce visceral obesity and metabolic

dysfunction(Han et al., 2014). These symptoms may reflect the metabolic consequences of insulin dysregulation and could be useful clinical screening indicators for early IR detection.

In this study, cardiovascular diseases and high blood pressure were more prevalent in non-insulin-resistant participants, contrasting to prior reports linking these conditions to insulin resistance (Wang et al., 2022). This may reflect sampling variability or the specific characteristics of our cohort. Central obesity showed a nonsignificant trend toward a higher prevalence in insulin-resistant individuals ($p = 0.0524$), consistent with its established role in metabolic dysfunction. Other conditions, including PCOS, type 2 diabetes, and NAFLD, were more frequent in insulin-resistant participants but did not reach statistical significance, likely due to the limited sample size. Participants with at least one diagnosed condition were significantly more likely to have insulin resistance, underscoring the broader association between insulin resistance and metabolic comorbidities.

In terms of nutrient deficiencies, vitamin D deficiency was significantly more common among insulin-resistant participants, consistent with a meta-analysis that reported an inverse relationship between vitamin D status and insulin resistance in both healthy individuals and those with type 2 diabetes, concluding that hypovitaminosis D is associated with increased insulin resistance worldwide(Rafiq & Jeppesen, 2021). In contrast, vitamin B12 deficiency and the absence of any deficiency were more common in the non-IR participants. Other deficiencies, including those of magnesium, zinc, chromium, omega-3 fatty acids, and vitamin K, showed no statistically significant associations, although some were reported only in the non-IR group.

Although no statistically significant associations were found between hormonal imbalances and IR, non-significant trends suggested possible links between insulin resistance and conditions such as high androgen levels/PCOS, hypothyroidism, and hyperthyroidism. Similarly, although not statistically significant, gut dysbiosis, autoimmune conditions, and periodontal disease were more frequently reported in insulin-resistant participants. The potential roles of chronic inflammation and microbial imbalance in IR development remain important areas for future research.

A statistically significant association was found between hormonal medication use and insulin resistance, suggesting that certain therapies may contribute to impaired glucose metabolism or reflect the treatment of underlying endocrine disorders. Other medication categories, including beta-blockers, corticosteroids, statins, and antipsychotics, were not significantly associated with IR in this study sample.

Collectively, these findings underscore important public health implications. Early recognition of symptom clusters such as abdominal weight gain, fatigue, and post-meal sleepiness, combined with routine screening for vitamin D deficiency and careful monitoring of hormonal medication use, could help identify individuals at elevated risk of insulin resistance before progression to more severe metabolic disease. Implementing targeted interventions that integrate lifestyle

modification, nutritional monitoring, and clinical follow-up may therefore represent a valuable strategy to reduce the burden of insulin resistance in the population.

Strengths and Limitations

A major strength of this study was the inclusion of a broad range of lifestyle, dietary, and clinical variables, providing a comprehensive picture of potential associations with insulin resistance. The inclusion of symptom-specific data, such as abdominal weight gain, increased hunger, fatigue, and post-meal sleepiness, provides clinically relevant insights that could support early detection strategies. Additionally, the focus on the adult population in Bosnia and Herzegovina addresses a gap in the regional literature, offering novel data from a Southeastern European context.

The unequal sex distribution, with a predominance of female participants, and the relatively modest sample size may have reduced the statistical power to detect some associations. Despite these constraints, this study offers valuable baseline evidence for future, larger-scale, and longitudinal research.

5. Conclusion

This study identified several statistically significant associations between insulin resistance and age, symptoms, vitamin D deficiency, health conditions, and hormonal medication use. While many findings require confirmation in larger longitudinal studies, they underscore the importance of considering both metabolic and non-metabolic factors in assessing insulin resistance risk. Future research should aim to validate these observations in more diverse and representative populations and explore the mechanisms underlying these associations. Collectively, these findings underscore the importance of early diagnosis, regular clinical screening, and implementation of targeted metabolic health strategies that address diverse risk factors.

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References

- Freeman, A. M., & Pennings, N. (2018). Insulin resistance.
- Schinner, S., Scherbaum, W. A., Bornstein, S. R., & Barthel, A. (2005). Molecular mechanisms of insulin resistance. *Diabetic Medicine*, 22(6), 674-682.
- Ndisang, J. F., Vannacci, A., & Rastogi, S. (2017). Insulin resistance, type 1 and type 2 diabetes, and related complications, 2017. *Journal of diabetes research*, 2017, 1478294.
- Fahed, M., Abou Jaoudeh, M. G., Merhi, S., Mosleh, J. M. B., Ghadieh, R., Al Hayek, S., & El Hayek Fares, J. E. (2020). Evaluation of risk factors for insulin resistance: a cross-sectional study among employees at a private university in Lebanon. *BMC Endocrine Disorders*, 20, 1-14.
- Oya, J., Nakagami, T., Yamamoto, Y., Fukushima, S., Takeda, M., Endo, Y., & Uchigata, Y. (2014). Effects of age on insulin resistance and secretion in subjects without diabetes. *Internal medicine*, 53(9), 941-947.
- Karakelides, H., Irving, B. A., Short, K. R., O'Brien, P., & Nair, K. S. (2010). Age, obesity, and sex effects on insulin sensitivity and skeletal muscle mitochondrial function. *Diabetes*, 59(1), 89-97.
- Ciarambino, T., Crispino, P., Guarisco, G., & Giordano, M. (2023). Gender differences in insulin resistance: new knowledge and perspectives. *Current Issues in Molecular Biology*, 45(10), 7845-7861.
- Geer, E. B., & Shen, W. (2009). Gender differences in insulin resistance, body composition, and energy balance. *Gender medicine*, 6, 60-75.
- Gratas-Delamarche, A., Derbré, F., Vincent, S., & Cillard, J. (2014). Physical inactivity, insulin resistance, and the oxidative-inflammatory loop. *Free radical research*, 48(1), 93-108.
- Yan, Y. X., Xiao, H. B., Wang, S. S., Zhao, J., He, Y., Wang, W., & Dong, J. (2016). Investigation of the relationship between chronic stress and insulin resistance in a Chinese population. *Journal of epidemiology*, 26(7), 355-360.
- Li, L., Li, X., Zhou, W., & Messina, J. L. (2013). Acute psychological stress results in the rapid development of insulin resistance. *The Journal of Endocrinology*, 217(2), 175.
- Spiegel, K., Knutson, K., Leproult, R., Tasali, E., & Van Cauter, E. (2005). Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. *Journal of Applied Physiology*.
- Hyman, M. A. (2010). Environmental toxins, obesity, and diabetes: an emerging risk factor. *Alternative Therapies in Health & Medicine*, 16(2), 56.
- Kelishadi, R., Mirghaffari, N., Poursafa, P., & Gidding, S. S. (2009). Lifestyle and environmental factors associated with inflammation, oxidative stress and insulin resistance in children. *Atherosclerosis*, 203(1), 311-319.
- Zuo, H., Shi, Z., Yuan, B., Dai, Y., Pan, X., Wu, G., & Hussain, A. (2013). Dietary patterns are associated with insulin resistance in Chinese adults without known diabetes. *British journal of nutrition*, 109(9), 1662-1669.
- Martins, F. O., & Conde, S. V. (2022). Impact of diet composition on insulin resistance. *Nutrients*, 14(18), 3716.
- Gołabek, K. D., & Regulska-Ilow, B. (2019). Dietary support in insulin resistance: An overview of current scientific reports. *Adv Clin Exp Med*, 28(11), 1577-1585.

- Salihefendic, D. (2023). Eating and Lifestyle Habits in Underweight Patients with Insulin Resistance. *Materia Socio-medica*, 35(1), 18.
- Mestre Font, M., Busquets-Cortés, C., Ramírez-Manent, J. I., Tomás-Gil, P., Paublíni, H., & López-González, Á. A. (2023). Influence of sociodemographic variables and healthy habits on the values of insulin resistance indicators in 386,924 Spanish workers. *Nutrients*, 15(24), 5122.
- Lebovitz, H. E. (2001). Insulin resistance: definition and consequences. *Experimental and clinical endocrinology & diabetes*, 109(Suppl 2), S135-S148.
- Kumar, S., Senapati, S., Bhattacharya, N., Bhattacharya, A., Maurya, S. K., Husain, H., ... & Pandey, A. K. (2023). Mechanism and recent updates on insulin-related disorders. *World Journal of Clinical Cases*, 11(25), 5840.
- Zhao, X., An, X., Yang, C., Sun, W., Ji, H., & Lian, F. (2023). The crucial role and mechanism of insulin resistance in metabolic disease. *Frontiers in endocrinology*, 14, 1149239.
- Rao, G. (2001). Insulin resistance syndrome. *American Family Physician*, 63(6), 1159-1164.
- Rogowicz-Frontczak, A., Majchrzak, A., & Zozulińska-Ziółkiewicz, D. (2017). Insulin resistance in endocrine disorders—treatment options. *Endokrynologia Polska*, 68(3), 334-351.
- Xu, Y., & Qiao, J. (2022). Association of insulin resistance and elevated androgen levels with polycystic ovarian syndrome (PCOS): a review of literature. *Journal of healthcare engineering*, 2022(1), 9240569.
- Gierach, M., Gierach, J., & Junik, R. (2014). Insulin resistance and thyroid disorders. *Endokrynologia Polska*, 65(1), 70-76.
- Gür, C., Boz, M., Müderrisoğlu, C., & Polat, H. (2015). The Relationship between Insulin Resistance and Cortisol Levels. *Istanbul Med J*, 16, 73-76.
- Cigerli, O., Parildar, H., Unal, A. D., Tarcin, O., Kut, A., Eroglu, H., & Guvener, N. (2016). Vitamin deficiency and insulin resistance in nondiabetic obese patients. *Acta Endocrinologica (Bucharest)*, 12(3), 319.
- Chutia, H., & Lynrah, K. G. (2015). Association of serum magnesium deficiency with insulin resistance in type 2 diabetes mellitus. *Journal of Laboratory Physicians*, 7(02), 075-078.
- Cruz, K. J. C., de Oliveira, A. R. S., Morais, J. B. S., Severo, J. S., Mendes, P. M. V., de Sousa Melo, S. R., ... & Marreiro, D. D. N. (2018). Zinc and insulin resistance: biochemical and molecular aspects. *Biological trace element research*, 186, 407-412.
- Aydemir, S., Bayraktaroglu, T., Sert, M., Sokmen, C., Atmaca, H., Mungan, G., ... & Ustundag, Y. (2005). The effect of Helicobacter pylori on insulin resistance. *Digestive diseases and sciences*, 50, 2090-2093.
- Romero-Gómez, M. (2006). Insulin resistance and hepatitis C. *World journal of gastroenterology: WJG*, 12(44), 7075.
- Lazar, E., Sherzai, A., Adeghate, J., & Sherzai, D. (2021). Gut dysbiosis, insulin resistance and Alzheimer's disease: review of a novel approach to neurodegeneration. *Frontiers in Bioscience-Scholar*, 13(1), 17-29.
- Zaman, G. S. (2020). Pathogenesis of insulin resistance. In *Cellular metabolism and related disorders*. London, UK: IntechOpen.

- Parcha, V., Heindl, B., Kalra, R., Li, P., Gower, B., Arora, G., & Arora, P. (2022). Insulin resistance and cardiometabolic risk profile among nondiabetic American young adults: insights from NHANES. *The Journal of Clinical Endocrinology & Metabolism*, 107(1), e25-e37.
- Małkowska, P. (2024). Positive effects of physical activity on insulin signalling. *Current Issues in Molecular Biology*, 46(6), 5467-5487.
- Gianotti, L., Belcastro, S., D'Agnano, S., & Tassone, F. (2021). The stress axis in obesity and diabetes mellitus: an update. *Endocrines*, 2(3), 334-347.
- Malin, S. K., Kullman, E. L., Scelsi, A. R., Haus, J. M., Filion, J., Pagadala, M. R., ... & Kirwan, J. P. (2018). A whole-grain diet reduces peripheral insulin resistance and improves glucose kinetics in obese adults: A randomised controlled trial. *Metabolism*, 82, 111-117.
- Everson, S., Glodberg, D. E., Helmrich, S. P., Lakka, T. A., Lynch, J. W., Kaplan, G. A., & Salonen, J. T. (1998). Weight gain and the risk of developing insulin resistance syndrome. *Diabetes care*, 21(10), 1637-1643.
- Han, J. M., Kim, H. G., Lee, J. S., Choi, M. K., Kim, Y. A., & Son, C. G. (2014). Repeated sense of hunger leads to the development of visceral obesity and metabolic syndrome in a mouse model. *PloS one*, 9(5), e98276.
- Wang, T., Li, M., Zeng, T., Hu, R., Xu, Y., Xu, M., ... & Lu, J. (2022). Association between insulin resistance and cardiovascular disease risk varies according to glucose tolerance status: a nationwide prospective cohort study. *Diabetes Care*, 45(8), 1863-1872.
- Rafiq, S., & Jeppesen, P. B. (2021). Insulin resistance is inversely associated with the status of vitamin D in both diabetic and non-diabetic populations. *Nutrients*, 13(6), 1742.