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Are Nesfatin-1, Apelin, Chemerin, Omentin-1, and Resistin Hormone Levels Comparable in Individuals with Opioid Use Disorder and Obesity?

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ABSTRACT

Objective: The primary aim of this research was to assess whether individuals with obesity or opioid use disorder (OUD) exhibit altered levels of specific adipokines compared with healthy subjects.

Materials and Methods: A total of 90 participants were included (obesity group: 30, OUD group: 30, control group: 30).

Results: Participants in both the obesity and OUD groups exhibited significantly increased concentrations of resistin, chemerin, and omentin-1 ($p<0.05$). Conversely, apelin levels were substantially reduced in both groups compared to healthy controls ($p=0.001$). Notably, nesfatin-1 levels were significantly lower only in the OUD group relative to controls ($p=0.005$). No statistically significant difference in body mass index (BMI) was found between individuals with OUD and healthy subjects ($p=0.619$).

Conclusion: These findings suggest that obesity may share pathophysiological similarities with addiction. Therefore, obesity management should include psychological support. Additionally, considering the physiological roles of adipokine-related hormones, patients should be closely monitored for blood glucose levels, insulin resistance, diabetes mellitus, lipolysis, inflammation, and clinical complications such as atherosclerosis.

Keywords: Adipokines, apelin, chemerin, nesfatin, obesity.

INTRODUCTION

Adipose tissue is regarded in the current literature as an endocrine organ. Beyond storing fat, it is involved in the synthesis and secretion of various endocrinologically active compounds called adipokines.[1] Apelin, chemerin, omentin-1, resistin, and nesfatin-1 are among these adipokines. They have multiple complex functions, including the regulation of body weight, appetite, glucose balance, and blood pressure.[1] Elevated levels of resistin, chemerin, and

apelin have been associated with endocrinological diseases and obesity.[1,2] In contrast, nesfatin-1 and omentin levels have been reported to be decreased in obese individuals.[1,3] Recent studies have also shown that adipokines are involved in neuroendocrine and immune system interactions,[4] and may serve as novel markers in diseases related to these systems.[5]

Recent literature has proposed that obesity may be conceptualized within an addiction framework, highlighting the roles of neuroendocrine and immune system interactions.[6] However, defining obesity solely as a form of food addiction may be an oversimplification.[7] Neurobehavioral studies have identified notable parallels between obesity and substance use disorders, particularly involving the brain's dopaminergic pathways.[8,9] One proposed mechanism suggests that intermittent access to sugar, similar to addictive substances, may trigger compulsive consumption behaviors.[9,10]

Additionally, findings from questionnaire-based assessments of compulsive eating behavior indicate that individuals with higher scores tend to show increased neural activation in the amygdala, anterior cingulate cortex, and orbitofrontal cortex.[11] Based on these findings, mental health experts classify obesity within the addiction model, as stressful life events can trigger uncontrollable eating episodes and certain foods possess addictive properties. It has been suggested that obesity may represent a form of eating addiction, akin to alcohol and substance dependence, and its etiology should therefore be considered within the framework of addiction.[12] As highlighted in the literature, the interaction of genetic, environmental, and individual factors plays a shared role in the development of both addiction and obesity.[6,12] In light of this, our study aims to determine whether adiponectin-group hormones, which are involved in obesity and insulin resistance, exhibit a similar profile in both addiction and obesity. Additionally, the roles of adiponectins in addiction and obesity will be comparatively analyzed.

MATERIALS AND METHODS

The Ethics Committee of Gaziosmanpaşa University granted approval for the study (date: 04.10.2021, number: 83116987 - 720). All procedures were conducted in accordance with the ethical standards outlined in the Declaration of Helsinki.

Power analysis was performed to determine the sample size using the G*Power program (Foul, Erdfelder, Lang, and Buchner, 2007). An F Test (analysis of variance, ANOVA) for repeated measurements indicated that the minimum sample size required was 66 for an effect size of 0.40, a 5% margin of error, and a 95% confidence interval across three groups.

KEY MESSAGES

- Obese and addicted patient groups showed similar results, at least in terms of some adiponectins.
- It was concluded that there may be a relationship between the etiology of obesity and the mechanisms underlying addiction, and psychotherapeutic interventions may be beneficial in obesity treatment.
- It was also suggested that certain adiponectins may also be useful in the treatment of patients with both obesity and opioid use disorder.

The study comprised 90 participants, divided into three groups: obese individuals (BMI >30) who presented to the Endocrinology outpatient clinic; patients diagnosed with opioid use disorder undergoing inpatient treatment at the Alcohol and Substance Addiction Treatment and Education Center (AMATEM) unit; and healthy controls with BMI between 18 and 24, matched for demographic characteristics with the other groups. The obesity group included individuals classified as obese according to the World Health Organization (WHO) criteria (BMI ≥30). Individuals with chronic illnesses, those taking weight-reducing medication, and participants receiving ongoing psychiatric therapy were excluded.

Patients in the addiction group were diagnosed with OUD according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria[13] and were receiving inpatient treatment at the AMATEM clinic. Only patients who voluntarily consented to participate were included. Participants in this group had a BMI between 18 and 24 and did not present with comorbid conditions such as diabetes mellitus or hypertension that could affect laboratory parameters or require chronic treatment. Clinical interviews confirmed that these participants did not have additional psychiatric disorders requiring treatment according to DSM-5 criteria.

Participants in both the healthy control and OUD groups were selected based on a BMI range of 18 to 24. Additionally, all participants across the three groups were between 18 and 45 years of age.

Demographic Data Form

The researcher-designed demographic data form included variables such as age, marital status, and education level. It also contained clinical assessment questions regarding whether the participants or their families had ever received psychiatric treatment and whether they had a history of alcohol or substance use.

Table 1. Distribution of demographic characteristics of the groups

	Obesity group (n=30)	Opioid use disorder group (n=30)	Control group (n=30)	p
Age	34.75±8.98	36.57±9.14	33.93±9.43	0.541
Marital status	20/7/3	13/13/4	14/16/0	0.047
(married/single/divorced)	66.6/23.3/10	43.3/43.3/13.3	46.6/53.3/0	
Educational level*				
8 years	19 (46.3%)	9 (22.0%)	13 (31.7%)	
8–12 years	6 (20.0%)	15 (50.0%)	9 (30.0%)	
>12 years	4 (25.0%)	4 (25.0%)	8 (50.0%)	
Employment status*				
Regular employment	22 (34.9%)	18 (28.6%)	23 (36.5%)	
Irregular employment/never employed	7 (29.2%)	10 (41.7%)	7 (29.2%)	

Values are presented as mean±standard deviation for age; other data are given as n (%). *: Fisher Exact test (Fisher–Freeman–Halton test) and Chi-square test were used in the computations.

Biochemical Evaluation

Venous blood samples were collected between 8:00 and 10:00 a.m. following at least 8 hours of overnight fasting. Samples were centrifuged at 2520 g for 10 minutes and stored at -20°C. Apelin and nesfatin-1 levels were analyzed using a competitive inhibition enzyme immunoassay technique (ELK Biotechnology, ELK8309; ELK2828), while omentin, chemerin, and resistin were measured using a sandwich enzyme immunoassay (ELK Biotechnology, ELK2003; ELK1953; 1225). Microplate readings were performed with an Organon Teknika enzyme-linked immunosorbent assay (ELISA) reader.

Statistical Analysis

Statistical analyses were conducted using SPSS for Windows, version 22 (Statistical Package for the Social Sciences, SPSS Inc., Chicago, IL).[14] The Shapiro-Wilk test was used to assess normality. Among the demographic and laboratory variables, age and nesfatin demonstrated a normal distribution, whereas resistin, chemerin, omentin, apelin, and BMI were non-normally distributed. The Fisher Exact test (Fisher–Freeman–Halton test) and Chi-square test were used for demographic characteristics. Continuous variables with a normal distribution are presented as mean±standard deviation, while those without a normal distribution are presented as median (Q1–Q3). Categorical data are presented as counts and percentages. One-way ANOVA was used for normally distributed numerical data, followed by Tukey's post hoc test for multiple comparisons. The Kruskal-Wallis test was applied to non-normally distributed variables, with the Dunn-Bonferroni post hoc test used for subsequent pairwise comparisons. Spearman's correlation analysis was also performed. Statistical significance was determined based on a threshold of $p<0.05$.

RESULTS

Using an F test (ANOVA) for repeated measurements, the minimum sample size was determined to be 66 participants, based on an effect size of 0.40, a 5% margin of error, a 95% confidence interval, and three study groups.

The mean age was 34.75±8.98 years for the obese group, 36.57±9.14 years for the OUD group, and 33.93±9.43 years for the control group (p=0.541). The majority of participants were married: 66.6% in the obese group, 43.3% in the OUD group, and 46.6% in the control group (p=0.047). Additionally, most participants had completed primary education and were employed in regular income-generating jobs. The percentage of participants with a primary school education was 66.6% in the obese group, 33.3% in the OUD group, and 43.3% in the control group (Table 1).

ANOVA Test

Analysis of variance was conducted to evaluate nesfatin-1 levels, as they followed a normal distribution. Tukey's honestly significant difference (HSD) post hoc test showed that nesfatin-1 levels were decreased in the OUD group (p=0.005), while the comparison between the obese and control groups did not yield a significant difference (p=0.485) (Table 2).

Dunn-Bonferroni

The Dunn-Bonferroni post-hoc test was performed to analyze resistin, chemerin, omentin, and apelin levels, as these variables were non-normally distributed. Apelin levels were lower in both the obesity and OUD groups compared to controls (p=0.001), with no difference observed between the obese and OUD groups (p=1.0).

Table 2. Distribution of quantitative variables among the groups

	Obesity group (n=30)	Opioid use disorder group (n=30)	Control group (n=30)	p
Nesfatin*	40830.71[ab]	33630.65[a]	44774.01[b]	**0.007**
Resistin	43.81[ab]	57.45[b]	31.63[a]	**0.001**
Chemerin	49.48[a]	63.39[a]	20.60[b]	**0.000**
Omentin	43.36a[b]	55.05[b]	34.30[a]	**0.007**
Apelin	38.55[a]	34.73[a]	57.92[b]	**0.001**
BMI	70.28[a]	33.46[b]	28.43[b]	**0.000**

BMI: Body Mass Index; *: ANOVA test was employed for normally distributed variables above the dividing line, with Tukey's HSD test used to determine the source of the difference. The Kruskal-Wallis test was employed for the analysis of variables that did not follow a normal distribution, and the Dunn-Bonferroni test was used to determine the source of the differences. (Resistin: Obesity-Control p=0.192; Obesity-OUD p=0.125; Control-OUD p=0.000) (Chemerin: Obesity-Control p=0.000; Obesity-OUD p=0.123; Control-OUD p=0.000) (Omentin: Obesity-Control p=0.505; Obesity-OUD p=0.242; Control-OUD p=0.005) (Apelin: Obesity-Control p=0.007; Obesity-OUD p=1; Control-OUD p=0.001). Values in the table are presented as mean rank values. Different superscript letters (a, b, c) indicate statistical significance.

Table 3. Median (Q1–Q3) values for parameters that do not fit the normal distribution

	Resistin	Chemerin	Omentin	Apelin	BMI
Mean	0.69±0.75	0.20±0.26	531.29±536.31	636.84±293.19	27.84±6.58
Median (Q1–Q3)	0.48 (0.40–0.59)	0.16 (0.07–0.23)	347.61 (224.78–512.14)	640.05 (528.12–762.82)	25.26 (22.86–22.86)

BMI: Body Mass Index.

Resistin, chemerin, and omentin levels were elevated in the OUD group compared to healthy controls (p<0.001 for resistin and chemerin; p=0.005 for omentin). In the obesity group, resistin levels did not differ significantly from either the control or OUD groups (OUD vs. obese: p=0.125; obese vs. control: p=0.192) (Table 2, 3).

Analysis of Spearman Correlation

A statistically significant positive association was found between BMI and chemerin (r=0.229, p=0.033), while a significant inverse correlation was observed between BMI and apelin (r=-0.271, p=0.011) (Table 4).

DISCUSSION

The principal goal of this research is to evaluate differences in adipokine concentrations among obese, OUD, and healthy individuals. The levels of resistin, chemerin, omentin-1, apelin, and nesfatin-1 were analyzed. Chemerin levels were significantly elevated in both patient groups, with the highest concentrations observed in individuals with OUD. Conversely, apelin and nesfatin-1 levels were reduced in the patient groups, with the lowest values observed in the OUD group. Although resistin and omentin-1 levels did not differ, both markers were higher in OUD patients.

Previous studies have shown that resistin and chemerin levels are increased in obesity.[1] Resistin is implicated in insulin resistance and is predominantly expressed in immune cells, where it contributes to inflammation, lipolysis, angiogenesis, and the development of atherosclerosis.[15] Similarly, chemerin plays a key role in the immune system, modulating inflammatory responses and being linked to glucose tolerance and insulin sensitivity.[16] The elevated levels of these adipokines in obese individuals are in agreement with previous research.[1,17] However, studies examining their levels in patients with substance use disorders are limited, often focusing on a single adipokine and a specific substance.[18,19] For example, a study on alcohol-dependent patients reported higher serum resistin levels compared to controls but found no correlation between resistin levels and alcohol cravings.[18] Research on serum chemerin levels in substance users is also scarce.[19] One study found increased chemerin levels in patients with chronic alcohol use, which was similarly observed in experimental models where ethanol was administered to rats.[19] Consistent with these findings, our study demonstrated elevated resistin and chemerin levels in OUD patients compared to controls. Since both adipokines are synthesized in visceral adipose tissue,[20] and chemerin acts as a pro-inflammatory cytokine,[21] their elevated levels in both OUD and obese patients highlight potential shared mechanisms. Independent of BMI, these patients should be monitored for metabolic risks such as insulin resistance, diabetes mellitus, and atherosclerosis. These findings also suggest a possible overlap in the pathophysiology of obesity and addiction.

Table 4. Results of Spearman correlation analysis

	Resistin	Chemerin	Omentin	Apelin	Nesfatin	BMI
Resistin	r=1	r=0.318*	r=0.290*	r=0.-414*	r=-285*	r=-0.015
		p=0.003	p=0.006	p=0.000	p=0.008	p=0.895
Chemerin	r=0.318*	1	r=0.313*	r=-0.336*	r=-0.231	r=0.229*
	p=0.003		p=0.003	p=0.001	p=0.031	p=0.033
Omentin	r=0.290*	r=0.313*	1	r=-0.054	r=-0.222*	r=0.138
	p=0.006	p=0.003		p=0.620	p=0.039	p=0.203
Apelin	r=0.-414*	r=-0.336*	r=-0.054	1	r=0.240*	r=-0.271*
	p=0.000	p=0.001	p=0.620		p=0.025	p=0.011
Nesfatin	r=-285*	r=-0.231	r=-0.222*	r=0.240*	1	r=0.071
	p=0.008	p=0.031	p=0.039	p=0.025		p=0.511
BMI	r=-0.015	r=0.229*	r=0.138	r=-0.271*	r=0.071	1
	p=0.895	p=0.033	p=0.203	p=0.011	p=0.511	

BMI: Body Mass Index. The figures presented in the table are r values. *: p<0.05.

Omentin-1, another adipokine, was found to be elevated only in OUD patients. Previous studies have reported an inverse relationship between omentin levels and BMI, noting reduced serum omentin concentrations in individuals with obesity and diabetes mellitus.[1,22] To date, no studies have investigated omentin levels in patients with substance use disorders. Our findings suggest that elevated omentin-1 in OUD may indicate a potential role for this adipokine in opioid addiction, warranting further research.

Unlike the three adipokines mentioned above, apelin and nesfatin-1 were lower in both obese and OUD groups. Apelin, a peptide hormone acting through a G protein-coupled receptor, is expressed in various peripheral tissues and brain regions.[23] Studies on apelin levels in obesity have yielded conflicting results. Some research has reported lower apelin levels in young obese individuals, associating this with insulin resistance,[24] while other studies found elevated apelin levels contributing to impaired insulin sensitivity.[25] These discrepancies have been attributed to tissue-specific variations in apelin expression.[26] In a previous study on OUD patients, no significant difference in apelin levels was observed.[27] Our findings, however, showed decreased apelin levels in both patient groups. This may result from differences in tissue expression or receptor-level changes, highlighting the need for further research. Given apelin's role in glucose uptake and insulin sensitivity, clinicians should closely monitor blood glucose levels in these patients.

Lastly, while nesfatin-1 levels were reduced in both obese and OUD patients, a statistically significant decrease was observed only in the OUD group. Nesfatin-1 is a relatively new anorexigenic hormone.[28] One study found a positive correlation between nesfatin-1 levels and eating disorder scores in obese women, but not in men.[29] Additionally, *in vitro* research demonstrated that nesfatin-1 could counteract methamphetamine-induced neurotoxicity.[30] Given the significantly reduced nesfatin-1 levels in OUD patients, future studies should explore the potential therapeutic role of nesfatin-1 in opioid addiction.

When evaluated through correlation analyses, previous studies, primarily conducted in obese populations, have generally reported interrelationships among these hormones.[18,19,30] In a study involving patients with alcohol use disorder, hormone levels were found to be associated with alcohol craving; however, the relationships among the hormones themselves were not examined.[18] In our findings, omentin, resistin, and chemerin were positively correlated with each other, whereas apelin and nesfatin showed negative correlations. To the best of our knowledge, our study is the first in the literature to compare three distinct groups while simultaneously investigating hormones of different classes. Although our results are of considerable value, they warrant further confirmation through future research.

CONCLUSION

This study compared adipokine levels in OUD patients, obese individuals, and a healthy control group, revealing similar patterns in both patient groups. Nesfatin-1 levels were lower in both groups, but a significant difference was observed only in OUD patients. These findings suggest a potential link between the pathophysiology of obesity and addiction, indicating that psychotherapeutic interventions could be beneficial in obesity treatment. Additionally, certain adipokines may hold promise

for the treatment of both obesity and OUD. Considering the metabolic roles of these adipokines, clinicians should closely monitor glucose metabolism, insulin resistance, inflammation, and cardiovascular risks in these patients.

This study represents a novel contribution to the literature; however, certain limitations should be noted. First, the inclusion of only male participants restricts the generalizability of the findings. Therefore, further studies involving larger and more diverse populations are warranted to confirm and extend these results.

Ethics Committee Approval: The Gaziosmanpaşa University Clinical Research Ethics Committee granted approval for this study (date: 04.10.20221, number: 83116987 - 720).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Conflict of Interest: The authors have no conflict of interest to declare.

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REFERENCES

1. Recinella L, Orlando G, Ferrante C, Chiavaroli A, Brunetti L, Leone S. Adipokines: new potential therapeutic target for obesity and metabolic, rheumatic, and cardiovascular diseases. Front Physiol 2020;11:578966. [CrossRef]
2. Ernst MC, Mulrow CJ. Chemerin: at the crossroads of inflammation and obesity. Trends Endocrinol Metab. 2010;21:660-7. [CrossRef]
3. de Souza Batista CM, Guess RZ, Lee MJ, et al. Omentin plasma levels and gene expression are decreased in obesity. Diabetes 2007;56(6):1655-61. [CrossRef]
4. Xie C, Chen Q. Adipokines: a new therapeutic target for osteoarthritis? Curr Rheumatol Rep 2019; 21:71. [CrossRef]
5. Collins KH, Lenz KL, Pollitt EN, et al. Adipose tissue is a critical regulator of osteoarthritis. Proc Natl Acad Sci 2021;118(1):e2021096118. [CrossRef]
6. Murray S, Tulloch A, Gold MS, Avena NM. Hormonal and neural mechanisms of food reward, eating behavior and obesity. Nature Reviews Endocrinol 2014;10(9):540-52. [CrossRef]
7. Ziauddeen H, Fletcher PC. Is food addiction a valid and useful concept? Obes Rev 2013;14:19-28. [CrossRef]
8. Volkow ND, Wise RA, Baler R. The dopamine motive system: implications for drug and food addiction. Nature Rev Neurosci 2017;18(12):741-52. [CrossRef]
9. García-García I, Horstmann A, Jurado MA, et al. Reward processing in obesity, substance addiction and non-substance addiction. Obesity Rev 2014;15(11):853-69. [CrossRef]
10. Malik VS, Hu FB. The role of sugar-sweetened beverages in the global epidemics of obesity and chronic diseases. Nature Rev Endocrinol 2022;18(4):205-18. [CrossRef]
11. Filbey FM, Myers US, Dewitt S. Reward circuit function in high BMI individuals with compulsive overeating: similarities with addiction. Neuroimage 2012;63:1800-6. [CrossRef]
12. Gupta A, Osadchiy V, Mayer EA. Brain-gut-microbiome interactions in obesity and food addiction. Nat Rev Gastroenterol Hepatol 2020;17(11):655-72. [CrossRef]
13. APA. American Psychiatry Assosiation. Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM 5). Washington, DC, American Psychiatric Association, 2013.
14. Gerber SB, Finn KV. Using SPSS for Windows: Data analysis and graphics. Springer, 2013.
15. Jamaluddin MS, Weakley Q, Yao Q, Chen C. Resistin: functional roles and therapeutic considerations for cardiovascular disease. Br J Pharmacol 2012;165:622-32. [CrossRef]
16. De Henau OGN, Degroot V, Imbault V, et al. Signaling properties of chemerin receptors CMKLR1, GPR1 and CCRL2. PLoS One 2016;11:e0164179. [CrossRef]
17. Funcke JB, Scherer PE. Beyond adiponectin and leptin: adipose tissue-derived mediators of inter-organ communication. Journal of Lipid Res 2019;60(10):1648-97. [CrossRef]
18. Akkişi-Kumsar N, Dilbaz N. Relationship between craving and ghrelin, adiponectin, and resistin levels in patients with alcoholism. Alcoholism: Clin Exp Res 2015;39(4):702-9. [CrossRef]
19. Ren RZ, Zhang X, Xu J, et al. Chronic ethanol consumption increases the levels of chemerin in the serum and adipose tissue of humans and rats. Acta Pharmacol Sinica 2012;33(5):652-9. [CrossRef]
20. Takahashi M, Okimura Y, Iguchi G, et al. Chemerin regulates beta-cell function in mice. Sci Rep 2011;1:123. [CrossRef]
21. Lee THY, Cheng KKY, Hoo RLC, Siu PMF, Yau SY. The novel perspectives of adipokines on brain health. Int J Molecular Sci 2019;20(22):5638. [CrossRef]

22. El-Mesallamy HO, El-Derany MO, Hamdy NM. Serum omentin-1 and chemerin levels are interrelated in patients with Type 2 diabetes mellitus with or without ischaemic heart disease. Diabet Med 2011;28(10):1194-200. [CrossRef]
23. Castan-Laurell L, Dray C, Attané C, Duparc T, Knauf C, Valet P. Apelin, diabetes, and obesity. Endocrine 2011;40:1-9. [CrossRef]
24. Kotanidou EP, Kalinderi K, Kyrgios I, et al. Apelin and G212A apelin receptor gene polymorphism in obese and diabetes youth. Pediatr Obes 2015;10:213-9. [CrossRef]
25. Krist J, Wieder K, Klöting N, et al. Effects of weight loss and exercise on apelin serum concentrations and adipose tissue expression in human obesity. Obes Facts 2013;6:57-69. [CrossRef]
26. Wysocka MB, Pietraszek-Gremplewicz K, Nowak D. The role of apelin in cardiovascular diseases, obesity and cancer. Front Physiol 2018;9:557. [CrossRef]
27. Tavanai A, Asadikaram G, Masoumi M. Opium Addiction is Associated with Increased Damage to Cardiomyocytes: Protective Roles Played by Apelins. Iranian Heart J 2020;21(3):6-14.
28. Stengel A, Taché Y. Nesfatin-1-role as possible new potent regulator of food intake. Regulatory Pept 2010;163(1-3):18-23. [CrossRef]
29. Weibert E, Hofmann T, Elbelt U, Rose M, Stengel A. NUCB2/nesfatin-1 is associated with severity of eating disorder symptoms in female patients with obesity. Psychoneuroendocrinol 2022;143:105842. [CrossRef]
30. Abbasi Z, Khaksari M, Shayannia A, Jafarisani M, Abbaszadeh-Goudarzi G, Nazarnezhad S, Rahmati M. Protection of the PC12 Cells by Nesfatin-1 Against Methamphetamine-Induced Neurotoxicity. Int J Peptide Res Ther 2022;28(4):10. [CrossRef]