PERINATAL JOURNAL

Original Article

Perinatal Journal 2025;33(1):5-10

Assessment of Xenopsin Related Peptide-1 levels in pregnant women with gestational diabetes mellitus

Emre Ağdemir[1], Melda Kuyucu[2], Mehtap Yücedağ[3], Kamile Kübra Ağdemir[1]

[1]Karasu State Hospital, Department of Obstetrics and Gynecology, Sakarya, Türkiye
[2]Bezmialem Vakif University, Medical Faculty Department of Obstetrics and Gynecology,İstanbul, Türkiye
[3]Umraniye Training and Research Hospital, Department of Obstetrics and Gynecology, İstanbul, Türkiye

Abstract

Objective: To compare serum Xenopsin-Related Peptide-1 (XP-1) levels in pregnant women diagnosed with gestational diabetes mellitus (GDM) versus healthy pregnant controls.

Methods: This prospective cohort study included pregnant women attending the Gynecology and Obstetrics outpatient clinic at Health Sciences University (SBU) Ümraniye Training and Research Hospital between April 1, 2023, and October 1, 2023. Pregnacies were between 24–28 weeks of gestation, aged 18–45 years, with singleton pregnancies, and without chronic systemic diseases. Following a 75 g oral glucose tolerance test (OGTT), 44 women diagnosed with GDM, while 44 women without GDM classified as controls. Serum XP-1 levels were assessed and compared between the groups. Blood samples were collected into anticoagulant-free tubes, centrifuged (2000–3000 rpm, 10 minutes), and serum samples were stored at -80°C until analysis. On the day of measurement, thawed samples were evaluated using the ELISA method with a commercial kit.

Results: Pregnancies in the GDM group were significantly older than those in the control group ($p = 0.001$). However, no statistically significant differences were observed between the groups regarding BMI at blood collection, pre-pregnancy BMI, weight gain, molecular XP-1 levels, or history of GDM ($p > 0.05$). Serum XP-1 levels ranged from 0.2 to 8.63 ng/mL, with a mean of 2.52 ± 1.81 ng/mL. XP-1 levels showed no significant association with treatment agents, BMI, pre-pregnancy BMI, weight gain, abdominal circumference (AC), or estimated fetal weight (EFW) at the time of blood collection ($p > 0.05$).

Conclusion: We found no significant association between serum XP-1 levels and GDM. Older maternal age was more prevalent in the GDM group. Further multidisciplinary, prospective studies are recommended to explore the potential role of XP-1 in pregnancy and GDM.

Keywords: Gestational diabetes mellitus, OGTT, Xenopsin Related Peptide-1

Introduction

Gestational diabetes mellitus (GDM) is characterized as glucose intolerance with an onset or first recognition during pregnancy.[1] Insulin resistance typically develops in mid-pregnancy and persisits into the third trimester.[2] Key contributors to insulin resistance during pregnancy include placental hormones, such as tumor necrosis factor-α (TNF- α), human placental lactogen (hPL), human placental growth hormone (hPGH), and adipokines. Additionally, elevated levels of estrogen, progesterone, and cortisol exacerbate the glucose-insulin balance.[3] Insulin secretion from the pancreas increases to compensate for peripheral insulin resistance. However, GDM arises when the pancreas fails to produce sufficient insulin to overcome the metabolic stress by insulin resistance.[4]

GDM has significant implications for both maternal and fetal health. Women with GDM are at high risk for the development of preeclampsia and the necessity for cesarean delivery.[5,6] Screening for GDM during pregnancy typically involves one-step or two-step processes. The International Association of Diabetes and Pregnancy Study Groups (IADPSG), based on findings from the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study, recommends a one-step 75 g oral glucose tolerance test. This test is performed after an 8–12 hour fasting period, with fasting, 1-hour, and 2-hour plasma glucose levels measured to confirm diagnosis.[7]

Xenopsin related peptide-1 (XP-1) is an octapeptide

Correspondence: Emre Ağdemir, Karasu State Hospital, Department of Obstetrics and Gynecology, Sakarya, Türkiye, **e-mail:** emreagdemir118@gmail.com, **Received:** December 13, 2024 **Accepted:** January 10, 2025

How to cite this article: Ağdemir E, Kuyucu M, Yücedağ M, Ağdemir KK. Assessment of Xenopsin Related Peptide-1 levels in pregnant women with gestational diabetes mellitus. Perinatal Journal 2025;33(1):5-10 DOI: 10.59215/prn.25.0331002

ORCID ID: E Ağdemir 0009-0000-2872-6186; M Kuyucu 0000-0002-4126-0541; M Yücedağ 000-0002-4382-3192; KK Ağdemir 0009-0004-2282-3275

CETUS Publishing

with common structural and biological similarities to the neurotensin (NT)/xenopsin/xenin family and associated with predator defense mechanisms. It has been identified in mammalian gastric mucosa and human gastric fluid, particularly in patients with duodenal ulcers. Studies administering synthetic XP-1 to dogs demonstrated hyperglycemia and stimulated rapid secretion of glucagon and cortisol.[8] There are studies showing that XP-1 is associated with glucose intolerance and insulin resistance. The receptors for the xenin molecule, which is in the same family as xenopsin, together with neurotensin receptor 1, have been shown to act indirectly on pancreatic beta cells in humans, causing the conversion from normal glucose tolerance to T2DM.[9] Additionally, Temur et al. found XP-1 as a potential biomarker of insulin resistance in patients with polycystic ovary syndrome (PCOS), revealing significantly higher XP-1 levels in PCOS patients compared to healthy controls.[10]

We aimed to investigate the association between XP-1 levels and GDM in pregnant women, a condition primarily characterized by insulin resistance and glucose intolerance.

Methods

This prospective cohort study was conducted between April 2023 and October 2023 at the Gynecology and Obstetrics Clinic of Ümraniye Training and Research Hospital, Türkiye. This study was conducted following the Helsinki Declaration Ethical Standards. The ethics committee approval for this study was obtained from the Clinical Research and Ethics Committee of Ümraniye Training and Research Hospital on 21/03/2023 with barcode number B.10.1.THK.4.34.H.GP.0.01/106. All participants were informed and written informed voluntary consent was obtained.

Our inclusion criteria were pregnant women with singleton pregnancies between 24 and 28 weeks of gestation without any systemic diseases.

Pregnant women with multiple pregnancies, morbid obesity (BMI> 35 kg/m²), conceived through assisted reproductive treatments (ART) and presence of known diseases (i.e., chronic hypertension, pregestational diabetes) were excluded from the study.

A 75 g oral glucose tolerance test (OGTT) was performed in pregnant women between 24 and 28 weeks of gestation. Xenopsin-Related Peptide-1 levels were compared between pregnant women with and without GDM. GDM was diagnosed by performing a single-step 2-hour 75-gram OGTT test and was established when any threshold value is met or exceeded (fasting value, 92 mg/dL; 1-hour value, 180 mg/dL; or 2-hour value, 153 mg/dL) and were included in the GDM group.[11] Pregnant women with all three normal values were considered to have normal glucose tolerance and were included in the non-GDM group.

In order to investigate Xenopsin Related Peptide-1 levels, peripheral venous blood samples were obtained from the participants included in the study after 8 hours of fasting. Centrifuged at 2000-3000 rpm for 10 minutes. After centrifugation, the upper serum portion of the samples was transferred to eppendorf tubes with the help of an automatic pipette. Each eppendorf tube was numbered and labeled and stored in the biochemistry laboratory of our hospital at -80°C until the study day. These serum samples were sent to Farmasina Tıbbi ve Kimyevi Ürünler San. ve Dış Tic. Ltd. Şti., Ataşehir, İstanbul/TURKEY, according to the Xenopsin Related Peptide-1 ELISA KIT instructions.

Maternal characteristics included maternal age, nulliparity, a history of gestational diabetes mellitus (GDM), pre-pregnancy Body Mass Index (BMI), and weight gain during pregnancy. BMI was calculated using the formula weight (kilogram) / height (meter) squared.

Sonographic outcomes encompassed fetal growth parameters, including persantiles of abdominal circumference (AC) and estimateted fetal birth weight (EFW) and amniotic fluid status, measured as the amniotic fluid index (AFI). Neonatal outcomes were assessed by gestational age (GA) at birth, mode of delivery (categorized as vaginal delivery or cesarean section), birth weight, the need for neonatal intensive care unit (NICU) admission, and 1st and 5th APGAR scores.

Power analysis was performed with the G*Power (v3.1.9.2) program [12] to determine the sample size. The statistical power of a study is usually expressed as 1-β (β = probability of Type II error) and it is recommended that a study should ideally have 80% power. In this case, based on Cohen's effect size coefficients, it was assumed that the analysis between two independent groups would have a medium-sized effect (d = 0.5). Based on this prediction, it was calculated that there should be at least 40 individuals in each group at a significance level of β= 0.05. However, taking into account potential data losses during the study process, 44 participants were selected for the control group and 44 participants for the study group.

SPSS 26 (Statistical Package for the Social Sciences) program was used for statistical analyses. Quantitative variables were represented by mean, standard deviation,

median, min and max values and qualitative variables were represented by descriptive statistical methods such as frequency and percentage. Shapiro Wilks test and Box Plot graphs were used to evaluate the suitability of the data for normal distribution. Student t-test was used for two-group evaluations of quantitative variables with normal distribution; Repeated Measures was used for intragroup evaluations. Mann Whitney-U test [13] was used for two-group evaluations of variables that did not show normal distribution; Kruskal Wallis test [14] was used for comparisons of three groups or more. Pearson test [15] was used in the evaluation of the relationships between variables. Chi-Square test [16], Fisher's Exact test [17] and Fisher Freeman Halton test were used in the comparison of qualitative data. The results were evaluated at 95% confidence interval and significance at $p<0.05$ level.

Results

In total, 88 women were included in our study and divided into two groups as GDM (n: 44) and non-GDM (n: 44). Among the maternal characteristics, there was a statistically significant difference only between maternal age and it was higher in the GDM group [32 (23-45) versus 27 (18-40) p= 0.001*]

Table 1. Comparison of maternal characteristics and laboratory values by groups

	Total (n:88)	Control Group (n=44)	GDM Group (n=44)	p value
Maternal age ¶	30 (18-45)	27 (18-40)	32(23-45)	0.001*
Nullipar, n (%)	39 (44.3)	24 (54.5)	15 (34.1)	0.053 **
History of GDM, n (%)	8 (9.1)	3 (6.8)	5 (11.4)	0.713 ***
Prepregnancy BMI (kg/m2) ¶	26 (17-36)	25 (17-36)	28 (20-34)	0.352*
Weight gain during pregnancy (kg)¶	5 (10-15)	5 (10-15)	5 (5-13)	0.451 *
GA of blood collection¶ weeks	26 (24-28)	25(24-27)	26 (24-27)	0.948 *
XP-1 level (ng/ml) ¶	2 (0-8)	2 (0-8)	2 (1-8)	0.665 *

Data are given as median¶ (min-max), or n (%). GDM, Gestational Diabetes Mellitus; BMI, Body mass index; GA, Gestational age; XP-1, Xenopsin-Related Peptide-1
*Student-t Test **Pearson chi-square *** Fisher's Exact Test

There was no significant difference between the two groups for other sonographic features and neonatal outcomes (Table2).

Table 2. Comparison of sonographic features and neonatal outcomes by groups

	Total (n=88)	Control Group (n=44)	GDM Group (n=44)	p value
AC (persantil)¶	56 (6-99)	44 (9-98)	60.5 (6-99)	0.133 *
EFW (persantil)¶	52 (1-99)	37 (10-99)	72 (1-99)	0.018*
AFI Status, n (%)				
Normal	75 (85.2)	38 (86.4)	37 (84.1)	1.000****
Polihydramnios	10 (11.4)	5 (11.4)	5 (11.4)	
Oligohydramnios	3 (3.4)	1 (2.3)	2 (4.5)	
GA at birth¶	39 (31-41)	39 (31-41)	38.6 (35-41)	0.126*
Mode of delivery, n (%)				
Vaginal delivery	48 (54.5)	24 (54.5)	24 (54.5)	1.000 **
Cesarean section	40 (45.5)	20 (45.5)	20 (45.5)	
Birth weight¶ (grams)	3380 (1251-4230)	3390 (1251-4210)	3350 (1965-4230)	0.988 *
Need of NICU, n (%)	21 (23.9)	8 (18.2)	8 (18.2)	0.211**
APGAR 1 min¶	8 (5-9)	8 (6-9)	7 (5-9)	0.040***
APGAR 5 min¶	9 (6-10)	9 (7-10)	8.5 (6-10)	0.071***

Data are given as median¶ (min-max), or n (%). AC, Abdominal Circumference; EFW, Estimated fetal weight; AFI; Amniotic Fluid Index; GA, Gestational age; NICU, Neonatal Intensive Care Unit
*Student-t Test **Pearson chi-square ***Mann Whitney-U Test ****Fisher Freeman Halton Test

In the GDM group, 39.8% (n = 35) had diet-regulated blood sugars, while 10.2% (n = 9) needed insulin. No statistically significant difference was found between the XP-1 levels of pregnant women according to treatment agents (p > 0.05), and to AFI status groups (p > 0.05) (Table 3).

Table 3. Comparison of XP-1 Levels according to groups and AFI status

	XP-1 level (ng/ml)	p value
Groups		
Control Group (n=44)	30.16±5.26 30 (18-45)	0.614 *
Diet-regulated GDM (n:35)	2.65±1.73 1.9 (1.1-6.9)	
Insulin-regulated GDM (n:9)	2.41±2.23 1.5 (1.2-8.3)	
AFI Status		
Normal (n=75)	2.57±1.85 1.8 (1.1-8.6)	0.619 **
Poli (n=10)	2.24±1.85 1.8 (0-6.8)	
Oligo (n=3)	2.26±0.72 2 (1.7-3.1)	

* Kruskal Wallis Test

** Mann Whitney-U Test

Table 4 shows the association between XP-1 levels and pre-pregnancy BMI, weight gain, AC measurements and EFW measurements during blood collection, fasting, 1st hour and 2nd hour OGTT measurements. There was no statistically significant relationship between XP-1 levels and these components (p > 0.05).

Table 4. Association of XP-1 Levels with Clinical Characteristics and OGTT Results

	XP-1 level (ng/ml)	
	r	p
Prepregnancy BMI (kg/m2)	-0.155	0.148
Weight gain during pregnancy (kg)	-0.040	0.714
AC (persantil)	-0.026	0.813
EFW (persantil)	0.042	0.696
OGTT fasting	0.007	0.948
OGTT 1. hour	-0.094	0.381
OGTT 2. hour	0.184	0.086

BMI, Body mass index; AC, Abdominal Circumference; EFW, Estimated fetal weight; OGTT, Oral Glucose Tolerance Test

r=Pearson's Correlation Test

Discussion

In this study, we compared maternal serum XP-1 levels between 24 and 28 weeks of gestation in pregnant women diagnosed with GDM and pregnant women without GDM. There was no difference between the GDM group and the control group in terms of serum XP-1 levels. Similarly, there was no difference between the GDM subgroups of diet-regulated and insulin-treated groups and the control group in terms of serum XP-1 levels.

Scientific studies continue to explore the pathogenesis of GDM, which is primarily attributed to chronic insulin resistance and β-cell dysfunction. Hormonal changes during pregnancy, such as increased estrogen, progesterone, leptin, cortisol, placental lactogen, and growth hormone, contribute to hyperinsulinemia and insulin resistance. While OGTT is widely accepted as the gold standard for diagnosing GDM, the search for new biomarkers for early diagnosis persists due to the test's limitations, including patient compliance and procedural complexity.[18]

Previous studies have highlighted insulin resistance in GDM. For example, Kautzky-Miller et al. reported significant insulin resistance in non-overweight GDM patients compared to normal pregnancies, which persisted postpartum.[19] In our study, prepregnancy BMIs were similar between the two groups. On average, both groups were classified as overweight. However, no significant difference was found between the BMI status and XP-1 level between the groups.

The role of XP-1 in insulin resistance and secretion has been explored in other contexts. Araki et al. initially discovered xenopsin in 1973, noting its effects on smooth muscle contraction.[20] Xenopsin related peptide-1 (XP-1) is an octapeptide that shares some structural and biological properties with the neurotensin (NT)/xenopsin/xenin family. In one study, it was detected in gastric fluids of patients with duodenal ulcer.[21] Feurle showed that XP-1, discovered in amphibians, is also found in mammalian gastric mucosal endocrine cells. He suggested that it may have various functions in signaling in the gastrointestinal tract.[22] Studies have shown that synthetic xenopsin causes hyperglycemia and Kawanishi et al. administered synthetic xenopsin to anesthetized dogs. As a result, they obtained a hyperglycemic response and a rapid increase in hormone release from the pancreas.[23] Feurle et al. observed that xenopsin induced hyperplasia of the pancreas in rats injected with xenopsin.[24] In a study on mammals, xenopsin precursors, which yield xenopsin-related peptide when digested with pepsin-related proteases, were detected in every organ, with the highest concentration in the

liver-stomach-intestine. In the study, precursors detected in blood, spinal fluid and intestinal lumen suggested that xenopsin related peptides may be present in endocrine and exocrine secretions.[25]

In 2017, Temur et al. included 40 women with polycystic ovary syndrome and 38 healthy women, totaling 78 women. Pregnant women, women with thyroid disease, women with type 1 or type 2 diabetes and smokers were excluded. XP-1 levels were significantly higher in women with PCOS. Insulin resistance was also found to be high in women with PCOS.[10] Based on this study, we planned to detect elevated XP-1 levels in pregnant women with GDM in which insulin resistance also plays a role in the mechanism. In our study, no significant difference was found between XP-1 levels between the two groups. There are not many studies on XP-1 in the literature. There is no study investigating serum Xenopsin Related Peptide-1 levels in pregnant women with GDM.

Clinical ImplicationsThis study is the first to investigate the relationship between Xenopsin Related Peptide-1 (XP-1) and GDM. Although our results did not demonstrate a significant difference in XP-1 levels between GDM and non-GDM groups, they provide a foundation for future research. Given XP-1's potential role in insulin secretion and resistance, it remains a candidate for further exploration as a biomarker for GDM. Larger, multicenter studies with more diverse populations and longitudinal designs are needed to validate or refute these findings.

LimitationsThe main limitations of this study were the small number of participants limited the statistical power to detect subtle differences in XP-1 levels and single-center, potentially limiting the generalizability of the findings. Additionally, serum XP-1 levels were assessed only during the diagnostic window (24-28 weeks of gestation). The potential changes in XP-1 levels following insulin or diet treatment were not examined, and the paucity of studies on XP-1 in pregnancy and GDM constrained our ability to contextualize the findings fully.

Conclusion

In our study, no significant difference was observed between serum XP-1 levels in the GDM population and the healthy pregnant population. We believe that more comprehensive and multidisciplinary prospective clinical studies on this subject are needed.

References

1. Sweeting A, Wong J, Murphy HR, Ross GP. A Clinical Update on Gestational Diabetes Mellitus. Endocr Rev [Internet]. 2022 Oct 1 [cited 2024 Aug 14];43(5):763–93. [PubMed][CrossRef]
2. Catalano PM, Tyzbir ED, Roman NM, Amini SB, Sims EAH. Longitudinal changes in insulin release and insulin resistance in nonobese pregnant women. Am J Obstet Gynecol [Internet]. 1991 [cited 2024 Aug 14];165(6 Pt 1):1667–72. [PubMed][CrossRef]
3. Barbour LA, McCurdy CE, Hernandez TL, Kirwan JP, Catalano PM, Friedman JE. Cellular Mechanisms for Insulin Resistance in Normal Pregnancy and Gestational Diabetes. Diabetes Care [Internet]. 2007 Jul 1 [cited 2024 Aug 14];30(Supplement_2):S112–9. [PubMed][CrossRef]
4. Alfadhli EM. Gestational diabetes mellitus. Saudi Med J [Internet]. 2015 [cited 2024 Aug 18];36(4):399–406. [PubMed][CrossRef]
5. Yogev Y, Xenakis EMJ, Langer O. The association between preeclampsia and the severity of gestational diabetes: The impact of glycemic control. Am J Obstet Gynecol [Internet]. 2004 Nov [cited 2024 Aug 14];191(5):1655–60. [PubMed][CrossRef]
6. Be M, Lp L, Ar D, Er T, U C, Dr C, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med [Internet]. 2008 May 8 [cited 2024 Aug 14];358(19):1991–2002. [PubMed][CrossRef]
7. Lende M, Rijhsinghani A. Gestational Diabetes: Overview with Emphasis on Medical Management. Int J Environ Res Public Health [Internet]. 2020 Dec 2 [cited 2024 Aug 14];17(24):1–12. [PubMed][CrossRef]
8. Kruszewska J, Laudy-Wiaderny H, Kunicki M. Review of Novel Potential Insulin Resistance Biomarkers in PCOS Patients-The Debate Is Still Open. Int J Environ Res Public Health [Internet]. 2022 Feb 1 [cited 2024 Aug 14];19(4). [PubMed][CrossRef]
9. Chowdhury S, Wang S, Patterson BW, Reeds DN, Wice BM. The combination of GIP plus xenin-25 indirectly increases pancreatic polypeptide release in humans with and without type 2 diabetes mellitus. Regul Pept. 2013 Nov 10;187:42–50. [PubMed][CrossRef]
10. Temur M, Özün Özbay P, Aksun S, Yilmaz Ö, Çift T, Üstünel S, et al. Elevated circulating levels of xenopsin-related peptide-1 are associated with polycystic ovary syndrome. Arch Gynecol Obstet [Internet]. 2017 Oct 1 [cited 2024 Aug 14];296(4):841–6. [PubMed][CrossRef]
11. Association AD. Standards of Medical Care in Diabetes—2015 Abridged for Primary Care Providers. Clin Diabetes [Internet]. 2015 [cited 2024 Aug 14];33(2):97. [PubMed][CrossRef]
12. Kang H. Sample size determination and power analysis using the G*Power software. J Educ Eval Health Prof [Internet]. 2021 [cited 2025 Jan 4];18. [PubMed][CrossRef]

13. McNemar And Mann-Whitney U Tests - PubMed [Internet]. [cited 2025 Jan 4].

14. Guo S, Zhong S, Zhang A. Privacy-preserving Kruskal-Wallis test. Comput Methods Programs Biomed [Internet]. 2013 Oct [cited 2025 Jan 4];112(1):135–45. [PubMed] [CrossRef]

15. F.r.s. Kp. X. On the criterion that a given system of deviations from the probable in the case of a correlated system of variables is such that it can be reasonably supposed to have arisen from random sampling. Philosophical Magazine Series 1. 2009 Jul;50(302):157–75. [CrossRef]

16. McHugh ML. The chi-square test of independence. Biochem Med (Zagreb) [Internet]. 2013 [cited 2025 Jan 4];23(2):143–9. [PubMed][CrossRef]

17. Jung SH. Stratified Fisher's exact test and its sample size calculation. Biom J [Internet]. 2014 [cited 2025 Jan 4];56(1):129–40. [PubMed][CrossRef]

18. Sharma AK, Singh S, Singh H, Mahajan D, Kolli P, Mandadapu G, et al. Deep Insight of the Pathophysiology of Gestational Diabetes Mellitus. Cells [Internet]. 2022 Sep 1 [cited 2024 Aug 14];11(17). [PubMed][CrossRef]

19. Kautzky-Willer A, Prager R, Waldhäusl W, Pacini G, Thomaseth K, Wagner OF, et al. Pronounced insulin resistance and inadequate beta-cell secretion characterize lean gestational diabetes during and after pregnancy. Diabetes Care [Internet]. 1997 [cited 2024 Aug 14];20(11):1717–23. [PubMed][CrossRef]

20. Araki K, Tachibana S, Uchiyama M, Nakajima T, Yasuhara T. Isolation and structure of a new active peptide "Xenopsin" on the smooth muscle, especially on a strip of fundus from a rat stomach, from the skin of Xenopus laevis. Chem Pharm Bull (Tokyo) [Internet]. 1973 [cited 2024 Aug 18];21(12):2801–4. [PubMed][CrossRef]

21. Shaw C, Stöckmann F, Conlon JM. Xenopsin- and neurotensin-like peptides in gastric juice from patients with duodenal ulcers. Eur J Clin Invest [Internet]. 1987 [cited 2024 Aug 18];17(4):306–12. [PubMed][CrossRef]

22. Feurle GE, Carraway RE, Rix E, Knauf W. Evidence for the presence of xenopsin-related peptide(s) in the gastric mucosa of mammals. J Clin Invest [Internet]. 1985 [cited 2024 Aug 18];76(1):156–62. [PubMed][CrossRef]

23. Kawanishi K, Goto A, Ishida T, Kawamura K, Nishina Y, Machida S, et al. The effects of xenopsin of endocrine pancreas and gastric antrum in dogs. Horm Metab Res [Internet]. 1978 [cited 2024 Aug 14];10(4):283–6. [PubMed] [CrossRef]

24. Feurle GE, Ohnheiser G, Löser C. Dissimilar trophic effects of cerulein and xenopsin on the rat pancreas. International Journal of Pancreatology. 1990 Mar;6(2):129–37. [PubMed][CrossRef]

25. Carraway RE, Mitra SP, Cochrane DE. Pro-xenopsin(s) in vesicles of mammalian brain, liver, stomach and intestine is apparently released into blood and cerebral spinal fluid. Regul Pept [Internet]. 2000 Nov 24 [cited 2024 Aug 14];95(1–3):115–24. [PubMed][CrossRef]