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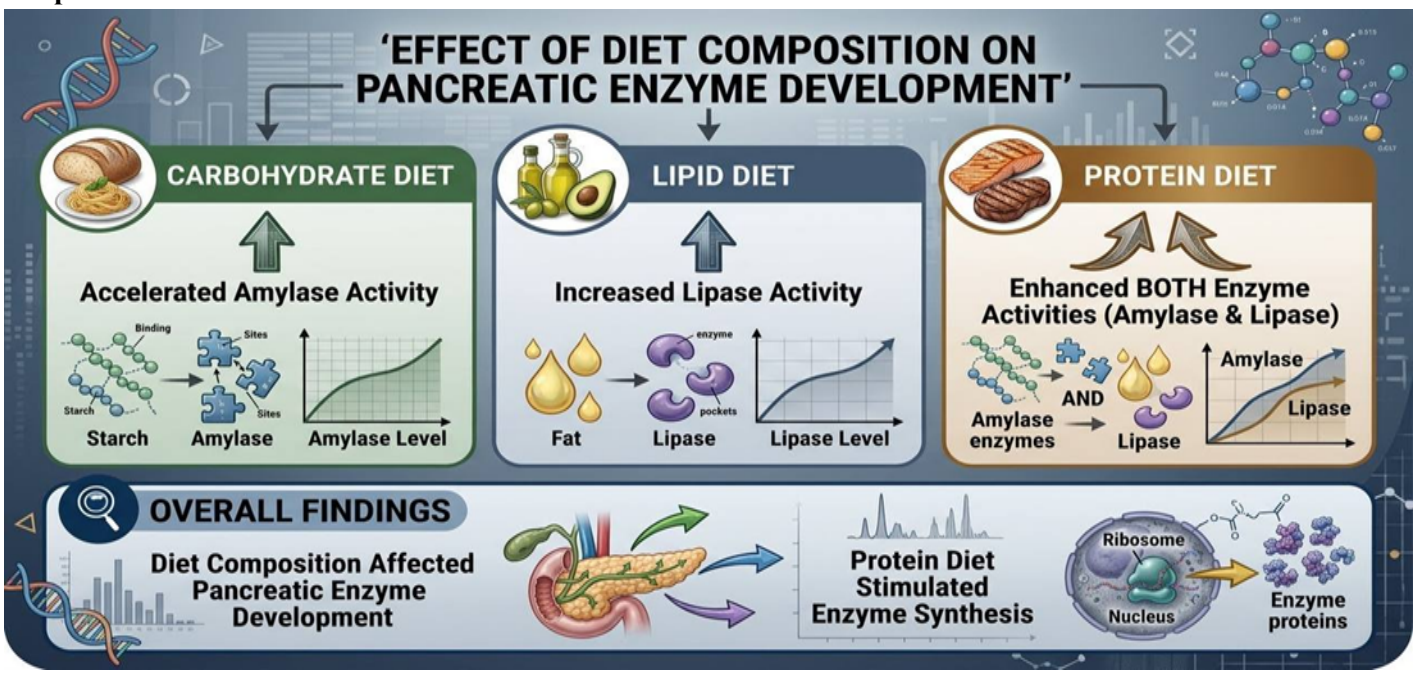
Research Article

The influence of nutrition on the formation of amylase and lipase secretion and homeostasis in the pancreas of rats of various ages during postnatal ontogenesis

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Graphical Abstract



Article Info

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Highlights

- Carbohydrate diet accelerated amylase activity during postnatal development.
- Lipid diet increased lipase activity from 2 months of age onward.
- Protein diet enhanced both enzyme activities.
- Diet composition affected pancreatic enzyme development.
- Protein diet stimulated enzyme synthesis.

Abstract

The present study investigated the influence of dietary macronutrient composition on the postnatal development of pancreatic amylase and lipase activities in male outbred rats. Animals were maintained after weaning on one of four diets—control mixed feed plus carrots, carbohydrate-enriched (combined

feed and bread), lipid-enriched (combined feed, bread and butter), or protein-enriched (combined feed and cooked egg white)—and enzyme activities were measured in pancreatic homogenate and serum at six ages (15 days, 1 month, 1.5 months, 2 months, 3 months and 4 months).
In the control group both enzyme activities increased with age, with amylase rising more steeply than lipase and reaching maximal values at 4 months. The carbohydrate-enriched diet accelerated the rise in pancreatic and serum amylase activity from one month of age (relative increases of 13.6–20.8 %). The lipid-enriched diet selectively accelerated lipase activity from two months of age (relative increases of 9–28.5 %). The protein-enriched diet elevated both amylase and lipase activities at most ages examined, consistent with a broader stimulatory effect on acinar-cell protein synthesis.
Strong positive correlations between tissue and serum activities (r = 0.41–0.99 for amylase; r = 0.57–0.94 for lipase) supported coordinated enzyme release into the circulation. These findings demonstrate that dietary composition modulates the age-dependent formation of amylolytic and lipolytic activity homeostasis in the pancreas and blood during postnatal ontogenesis, with protein-rich feeding exerting the most general effect on enzyme development.

Keywords: amylase, lipase, postnatal ontogenesis, homeostasis, pancreas, diet adaptation

1. Introduction

Digestion is a complex physiological and biochemical process in which ingested nutrients undergo physicochemical and chemical changes within the digestive system. Nutrients are mechanically broken down, swell, dissolve, and, under the influence of hydrolytic enzymes in the digestive juices, polymers are hydrolyzed into oligo-, di-, and monomers, which are then absorbed into the blood and lymph. The nutrients that reach the tissues are utilized for the body's plastic and energetic needs. The pancreas plays a very significant role in the digestive process. The juice of this gland contains enzymes that hydrolyze all nutrients. In a newborn, the functions of all organs and tissues, including the digestive glands, are not yet fully formed; they develop during the growth process out of the necessity to meet the body's demands. Ninety percent of pancreatic juice protein is produced in acinar cells, and these are enzyme proteins. If calculated as dry matter, 20 mg of enzymes, or 10^7 enzyme molecules, are synthesized in acinar cells per minute [1–3]. According to Rothman et al. [4], the synthesis of enzyme proteins in the pancreas at such a rate is likely unrealistic. By their calculations, a large portion of the enzymes that pass from the pancreas into the intestine is reabsorbed into the blood and then re-secreted as part of the pancreatic juice; in other words, an enteropancreatic circulation of enzymes occurs.

Classic studies have established that the exocrine pancreas adapts its enzyme profile to the macronutrient composition of the diet. Carbohydrate-rich diets preferentially increase amylase synthesis and secretion, lipid-rich diets increase lipase, and protein-rich diets tend to elevate overall proteolytic and, to a lesser extent, other enzyme activities [5–10]. These adaptive responses are mediated by both luminal nutrient sensing and hormonal signals, particularly cholecystokinin and secretin, and involve changes in gene transcription, mRNA stability, and translational efficiency within acinar cells [11–14].

During postnatal ontogenesis the digestive system undergoes rapid maturation. The transition from milk to solid food coincides with major shifts in pancreatic enzyme expression. However, the precise interaction between age-dependent maturation and dietary composition has not been fully quantified in outbred rat models across the first four months of life. Understanding this interaction is relevant both for basic physiology and for interpreting developmental aspects of nutritional programming of the exocrine pancreas. The present study was therefore designed to examine how four different dietary regimens influence the age-related formation of amylase and lipase activity homeostasis in pancreatic tissue and blood serum of male rats from 15 days to 4 months of age.

2. Materials and methods

2.1 Animals and experimental design

A long-term study was conducted on male white outbred rats of various ages (15-day, 1-month, 1.5-month, 2-month, 3-month, and 4-month-old). Body weights ranged from 20 to 205 g. The rats were divided into four groups, each receiving a diet of different composition. Water was continuously available. Until weaning at approximately 21 days of age, all animals remained with their dams and received maternal milk; thereafter they were

maintained exclusively on the assigned solid diet until the planned age of sacrifice.

The first (control) group was fed a diet of combined feed and carrots. One hundred grams of the combined feed contained 14.9 g of protein, 4.4 g of fat, 47 g of carbohydrates, and 11.7 g of dietary fiber.

The second group received equal (50/50) amounts of combined feed and loaf bread (carbohydrate-enriched). The third group was fed a mixture of combined feed (1/3), loaf bread (1/3), and butter (1/3) (lipid-enriched). The fourth group received equal proportions of combined feed (1/2) and cooked egg white (1/2) (protein-enriched).

2.2 Tissue collection and enzyme assays

Animals were decapitated under anesthesia at the designated ages. The pancreas was isolated, and blood released during decapitation was collected. A homogenate was prepared by mixing physiological saline with the gland at a 1:10 weight ratio. The homogenate was filtered, and amylase and lipase activities were determined in the filtrate and in serum. The activity of α-amylase was determined using the Mindray BA-88A semi-automatic biochemical analyzer with the "Amylase novo" reagent (kinetic method with CNP-G3 substrate). Lipase activity was determined using the Novo B-8343 lipase reagent on the same analyzer. Activities are expressed in U/mL.

2.3 Statistical analysis and ethics

Data are presented as mean ± SEM. Differences between groups were evaluated by one-way analysis of variance followed by Tukey's post-hoc test where appropriate. Pearson correlation coefficients were calculated between pancreatic and serum enzyme activities. Statistical significance was set at $p < 0.05$. The study was approved by the institutional animal ethics committee of Andijan State Medical Institute and conducted in accordance with national guidelines for the care and use of laboratory animals.

3. Results

In control (mixed-diet) rats, amylolytic activity of the pancreatic homogenate increased with age, reaching maximal values at maturity (Figure 1). At 15 days the activity was 143.3 ± 12.8 U/mL, approximately 10 % of the mature (4-month) value. Lipolytic activity was consistently lower than amylolytic activity at every age examined (Table 1).

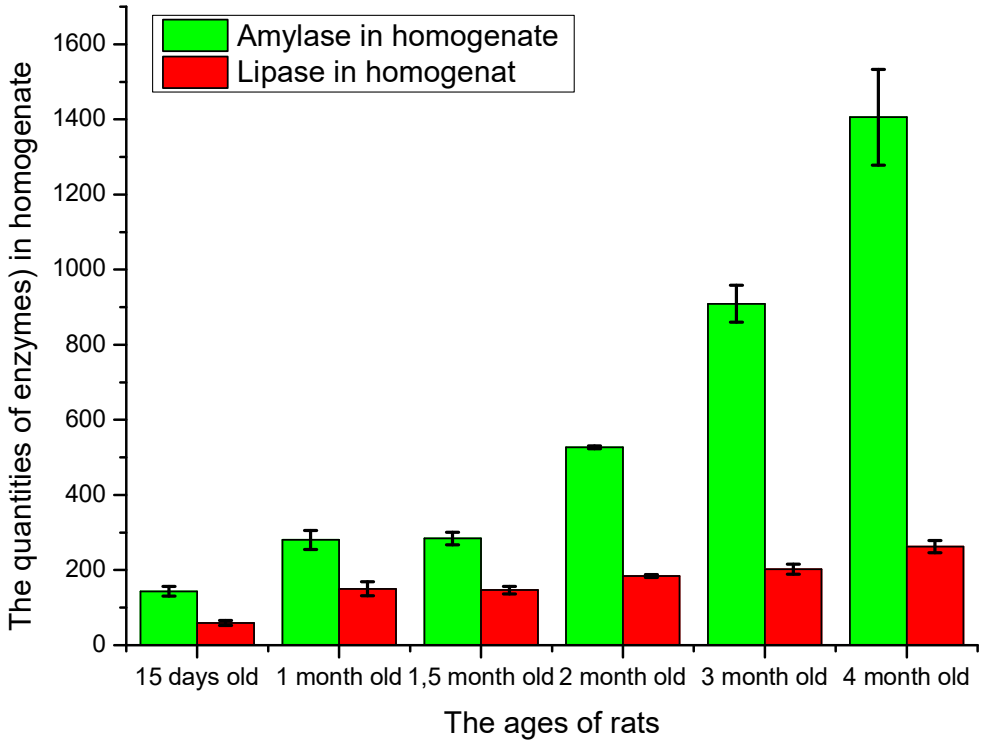


Figure 1. Hydrolytic enzymes in the pancreatic homogenate of mixed-fed rats of different ages (M±m). * indicates a statistically significant difference from the indicators of 15-day-old rats.

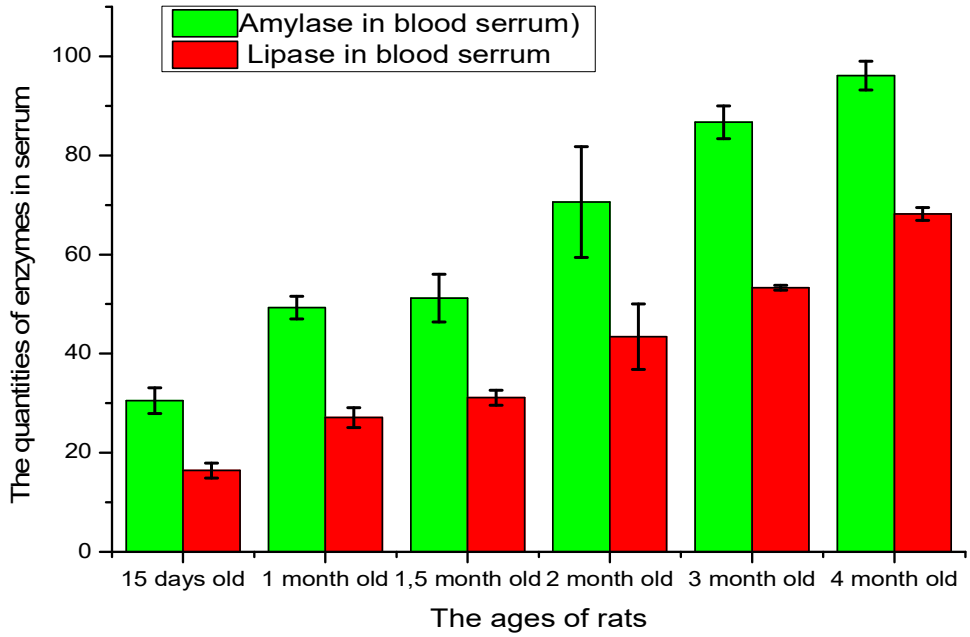


Figure 2. Hydrolytic enzymes in the blood serum of mixed-fed rats of different ages (M±m). * indicates a statistically significant difference from the indicators of 15-day-old rats.

Table 1. Pancreatic homogenate enzyme activities in control (mixed-diet) rats

Age	Amylase (U/mL)	Lipase (U/mL)	Amylase/Lipase ratio
15 days	143 ± 13	60 ± 6	2.4
1 month	280 ± 18	145 ± 12	1.9
1.5 months	285 ± 16	150 ± 11	1.9
2 months	520 ± 25	185 ± 14	2.8
3 months	910 ± 40	200 ± 15	4.6
4 months	1405 ± 65	263 ± 16	5.4

In the carbohydrate-enriched group, pancreatic amylase activity was elevated relative to age-matched controls from 1 month of age (relative increases of 13.6–20.8 %). Serum amylase was similarly increased. Lipase activity remained close to control values. In the lipid-enriched group, lipase activity in both pancreas and serum rose above control levels from 2 months of age (relative increases 9–28 %). In the protein-enriched group both enzymes were elevated at most ages examined (Table 2).

Table 2. Percentage deviation of enzyme activities from age-matched control group

Diet / Age	Pancreatic amylase	Pancreatic lipase	Serum amylase	Serum lipase
Carbohydrate 1 mo	+13.6%	≈ control	+17%	≈ control
Carbohydrate 2 mo	+20.8%	≈ control	+13%	≈ control
Lipid 2 mo	≈ control	+17%	≈ control	+17%
Lipid 3 mo	≈ control	+28%	≈ control	+12%
Protein 1 mo	+10%	+35%	+5 to +40%	+87%
Protein 2 mo	+19%	+18%	+40%	+18%

Correlation coefficients between pancreatic homogenate and serum activities were positive and high across most age groups (r = 0.41–0.99 for amylase; r = 0.57–0.94 for lipase), indicating coordinated tissue synthesis and release into the circulation.

4. Discussion

The present results demonstrate that dietary macronutrient composition modulates the postnatal maturation of pancreatic amylase and lipase activities. In control animals both enzymes increased with age, but amylase activity rose more steeply, consistent with the predominant use of carbohydrate as an energy source during growth

[15–17]. The carbohydrate-enriched diet further accelerated amylase development, while the lipid-enriched diet selectively accelerated lipase development from two months of age. These directional adaptations recapitulate the classic substrate-specific responses described in adult rats [5–8, 16, 18].

The protein-enriched diet elevated both amylase and lipase activities. This broader effect suggests that high protein intake may stimulate overall acinar-cell protein synthetic capacity rather than acting solely through substrate-specific transcriptional pathways. Similar trophic effects of protein-rich diets on pancreatic enzyme content have been reported previously [19–21, 24–27]. The strong positive correlations between tissue and serum activities support the concept of enteropancreatic circulation of enzymes originally proposed by Rothman and colleagues [4, 22]. Additional support for coordinated acinar-cell regulation comes from studies of stimulus-secretion coupling and hormonal control [11, 20, 28–30].

Age-related changes in the tissue-to-serum activity ratio indicate that the efficiency of enzyme release into the blood also matures during the first months of life. The relatively greater effect of the experimental diets after one month of age is consistent with the completion of weaning and the full establishment of solid-food intake.

Limitations of the study include the modest number of animals per age–diet combination, the absence of precise laboratory analysis of the macronutrient composition of the experimental diets, and the lack of molecular (mRNA or protein) confirmation of the enzyme changes. Future work incorporating larger samples, controlled purified diets, and gene-expression endpoints would allow stronger mechanistic conclusions.

5. Conclusions

1. In rats fed a carbohydrate-rich diet, the formation of amylolytic activity homeostasis in the pancreatic tissue and blood serum was accelerated during postnatal ontogenesis.
2. In rats fed a lipid-rich diet, the formation of lipolytic activity homeostasis in the pancreatic tissue accelerated during postnatal ontogenesis, starting from 2 months of age.
3. In experimental rats fed a protein-rich diet, the developmental formation of both studied enzymes (amylase and lipase) in the pancreatic tissue and blood accelerated during postnatal ontogenesis.

Declarations

Ethics approval

The study was approved by the institutional animal ethics committee of Andijan State Medical Institute.

Animal ethics approval: Confirm that formal approval was obtained before the animal experiments were conducted and all procedures were performed in accordance with national guidelines for the care and use of laboratory animals.

Availability of data and materials

The datasets generated during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

MNM and HSX conceived the study, performed the experiments, analyzed the data, and wrote the manuscript. Both authors read and approved the final version.

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Declaration of Use of Artificial Intelligence

During the preparation of this manuscript, the authors used artificial intelligence (AI)-assisted tools for language refinement, grammar correction, and improvement of readability. The AI tools were not used to generate research data, perform statistical analyses, interpret results, or draw scientific conclusions. The authors take full responsibility for the accuracy, originality, and integrity of the manuscript and have reviewed and approved the final version.

References

1. Korotko, G. F. (2005). Pancreatic secretion (2nd ed.). KSMU Publishing House.

2. Korotko, G. F. (2022). Recirculation and adaptation of digestive gland secretion enzymes. Experimental and Clinical Gastroenterology, 200(4), 138–152.
3. Shlygin, G. K. (2001). The role of the digestive system in metabolism. Sinergiya.
4. Rothman, S., Liebow, C., & Isenman, L. (2002). Conservation of digestive enzymes. Physiological Reviews, 82(1), 1–18.
5. Deschodt-Lanckman, M., Robberecht, P., Camus, J., & Christophe, J. (1971). Short-term adaptation of pancreatic hydrolases to nutritional and physiological stimuli in adult rats. Biochimie, 53(6), 789–796.
6. Grossman, M. I., Greengard, H., & Ivy, A. C. (1943). The effect of dietary composition on pancreatic enzymes. American Journal of Physiology, 138(4), 676–682.
7. Reboud, J. P., Marchis-Mouren, G., Paséro, L., Cozzone, A., & Desnuelle, P. (1966). Adaptation of the rate of biosynthesis of pancreatic amylase and chymotrypsinogen to starch-rich or protein-rich diets. Biochimica et Biophysica Acta, 117(2), 351–367.
8. Ben Abdeljlil, A., Visani, A. M., & Desnuelle, P. (1963). Adaptation of the exocrine secretion of rat pancreas to the composition of the diet. Biochemical and Biophysical Research Communications, 10(2), 112–116.
9. Scheele, G. A. (1975). Two-dimensional gel analysis of soluble proteins. Characterization of guinea pig exocrine pancreatic proteins. Journal of Biological Chemistry, 250(14), 5375–5385.
10. Case, R. M. (1978). Synthesis, intracellular transport and discharge of exportable proteins in the pancreatic acinar cell and other cells. Biological Reviews, 53(2), 211–354.
11. Williams, J. A. (2006). Regulation of pancreatic acinar cell function. Current Opinion in Gastroenterology, 22(5), 498–504.
12. Logsdon, C. D., & Ji, B. (2013). The role of protein synthesis and digestive enzymes in acinar cell injury. Nature Reviews Gastroenterology & Hepatology, 10(6), 362–370.
13. Petersen, O. H. (2014). Calcium signalling and secretory epithelia. Cell Calcium, 55(6), 282–289.
14. Yule, D. I. (2010). Pancreatic acinar cells: Molecular insight from studies of signal-transduction using transgenic animals. International Journal of Biochemistry & Cell Biology, 42(11), 1757–1761.
15. Giorgi, D., Bernard, J. P., Lapointe, R., & Dagorn, J. C. (1984). Regulation of amylase messenger RNA concentration in rat pancreas by food content. EMBO Journal, 3(7), 1521–1524.
16. Brannon, P. M. (1990). Adaptation of the exocrine pancreas to diet. Annual Review of Nutrition, 10, 85–105.
17. Duan, R. D., & Erlanson-Albertsson, C. (1989). Pancreatic lipase and colipase activity increase in pancreatic acinar tissue of diabetic rats. Pancreas, 4(3), 329–334.
18. Sabbatini, M. E., Gorelick, F., & Glaser, S. (2014). Adenylyl cyclases in the digestive system. Cellular Signalling, 26(6), 1173–1181.
19. Liddle, R. A. (2012). Regulation of pancreatic secretion. In L. R. Johnson (Ed.), Physiology of the gastrointestinal tract (5th ed., pp. 1425–1460). Academic Press.
20. Chandra, R., & Liddle, R. A. (2009). Neural and hormonal regulation of pancreatic secretion. Current Opinion in Gastroenterology, 25(5), 441–446.
21. Wang, B. J., & Cui, Z. J. (2007). How does cholecystokinin stimulate exocrine pancreatic secretion? From birds, rodents, to humans. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 292(2), R666–R678.
22. Isenman, L. D., & Rothman, S. S. (1977). Transport of alpha-amylase across the basolateral membrane of the pancreatic acinar cell. Proceedings of the National Academy of Sciences, 74(9), 4068–4072.
23. Bulgakova, V. A. (1999). Salivodiagnostics of the level of endogenous and exogenous blood components [Doctoral dissertation]. Krasnodar.
24. Pandol, S. J. (2010). The exocrine pancreas. Morgan & Claypool Life Sciences.
25. Whitcomb, D. C., & Lowe, M. E. (2007). Human pancreatic digestive enzymes. Digestive Diseases and Sciences, 52(1), 1–17.
26. Layer, P., & Keller, J. (1999). Pancreatic enzymes: Secretion and luminal nutrient

digestion in health and disease. Journal of Clinical Gastroenterology, 28(1), 3–10.
27. Keller, J., & Layer, P. (2005). Human pancreatic exocrine response to nutrients in health and disease. Gut, 54(Suppl. 6), vi1–vi28.
28. Czakó, L., Hegyi, P., Rakonczay, Z., Jr., Wittmann, T., & Otsuki, M. (2009). Interactions between the endocrine and exocrine pancreas and their clinical relevance. Pancreatology, 9(4), 351–359.
29. Hegyi, P., & Petersen, O. H. (2013). The exocrine pancreas: The acinar-ductal tango in physiology and pathophysiology. Reviews of Physiology, Biochemistry and Pharmacology, 165, 1–30.
30. Williams, J. A., & Yule, D. I. (2012). Stimulus-secretion coupling in pancreatic acinar cells. In L. R. Johnson (Ed.), Physiology of the gastrointestinal tract (5th ed., pp. 1361–1398). Academic Press.