Technical Note

Proposal for a New Surgical Technique: Long Biliopancreatic Limb and Complete Duodenal Exclusion with Preservation of Pyloric Sphincter Function

Kyung Yul Hur *

Posted Date: 31 March 2026

doi: 10.20944/preprints202603.2347.v1

Keywords: metabolic surgery; duodenal exclusion; pyloric preservation; epithelial-mesenchymal crosstalk; enteroendocrine reprogramming

 doi:10.20944/preprints202603.2347.v1

Technical Note

Proposal for a New Surgical Technique: Long Biliopancreatic Limb and Complete Duodenal Exclusion with Preservation of Pyloric Sphincter Function

Kyung Yul Hur

Department of Surgery, Chung Hospital, Seoul, Republic of Korea, Emeritus Professor, Department of Surgery, Soonchunhyang University College of Medicine, Seoul, Republic of Korea; hurusa@hanmail.net;
Tel.: +82-10-8936-3304

Abstract

Background: In non-obese patients with type 2 diabetes mellitus (T2DM), metabolic surgery is often limited by the unexpected inconsistent outcomes. Although pylorus-preserving gastric bypass procedures have been widely adopted, incomplete foregut exclusion frequently results in unsatisfactory glycemic control or relapse. To address this limitation, we propose Single Anastomosis Pyloro-Enterostomy (SAPE), designed to achieve complete duodenal exclusion while maintaining pyloric sphincter function. **Methods:** Based on long-term clinical observation and analysis of incretin dynamics, SAPE was developed as a loop-type single-anastomosis configuration incorporating a sufficiently long biliopancreatic (BP) limb. The duodenal tissue attached to the pyloric ring is completely removed, and the small intestine is anastomosed directly to the pylorus using interrupted sutures to preserve sphincteric motility. Anatomical design was guided by evidence from enteroendocrine physiology, epithelial-mesenchymal crosstalk, and reprogramming of regional intestinal identity after anastomosis. **Results:** Compared with pylorus-preserving duodenal-jejunal bypass (DJB) and other incomplete foregut-excluding procedures, SAPE theoretically enables more profound and durable suppression of a key diabetogenic signal originating from the proximal small intestine. The combination of complete duodenal exclusion and an adequately long BP limb minimizes the re-expansion of proximal epithelial identity and maintains long-term glycemic improvement without compromising digestive continuity or nutritional status. **Conclusion:** SAPE may provide a physiologically optimized surgical framework for the treatment of non-obese T2DM by integrating anatomical precision with metabolic efficacy. This technique ensures complete foregut exclusion, preserves pyloric function, and potentially prevents enteroendocrine reprogramming associated with late glycemic relapse. Further clinical evaluation is warranted to confirm its metabolic and functional outcomes.

Keywords: metabolic surgery; duodenal exclusion; pyloric preservation; epithelial-mesenchymal crosstalk; enteroendocrine reprogramming

1. Metabolic Surgery for Non-Obese Type 2 Diabetes in Korea

In Korea, a characteristic feature of type 2 diabetes is the high proportion of non-obese patients compared to other countries.[1] Therefore, metabolic surgery can be applied to a significant number of non-obese diabetic patients. The causes of surgically treatable diabetes are two independent factors—obesity and the imbalance of incretin secretion from the small intestine—both of which result in hyperglycemia, making it nearly impossible to distinguish their respective contributions when both are present. From this perspective, Korea provides a unique clinical environment to evaluate the incretin-related effects of metabolic surgery independent of obesity.

 doi:10.20944/preprints202603.2347.v1

Metabolic surgery for non-obese type 2 diabetes began in Korea in 2009. The author, as one of the early surgeons to perform such procedures, applied institution-specific modifications, but the outcomes varied markedly across centers—some satisfactory, others disappointing. Most hospitals performed pylorus-preserving duodenal-jejunal bypass (DJB), which often resulted in recurrence of hyperglycemia and unsatisfactory glycemic control.[2,3] In contrast, the author performed one-anastomosis gastric bypass (OAGB) and observed a durable and stable improvement in glycemic control without relapse.[4,5] Since patient BMI and diabetes severity were comparable across centers, the author attributed this difference to the surgical technique itself. Continuous observation and analysis led to the development of a novel procedure, Single Anastomosis Pyloro-Enterostomy (SAPE), which maximizes glycemic improvement while preserving normal gastrointestinal function.

This manuscript is presented as a conceptual and narrative review incorporating a hypothesis-driven surgical proposal, rather than a systematic review or report of prospective clinical outcomes.

Additionally, according to the use of artificial intelligence, the author confirms that this manuscript complies with the TITAN 2025 Guidelines. No generative AI tools were employed for data analysis, statistical interpretation, or drafting of the scientific content. Only minor editorial assistance for language polishing under full human supervision.[6]

2. GIP as a Parameter for Foregut Exclusion

Enteroendocrine K-cells that secrete glucose-dependent insulinotropic polypeptide (GIP) are most densely distributed in the first portion of the duodenum, gradually decreasing distally. GIP levels are elevated in type 2 diabetes and considered a strong diabetogenic factor, although its precise role remains controversial. The recent advent of dual glucagon-like peptide-1 (GLP-1)/GIP agonists demonstrating potent antidiabetic effects will likely further intensify this debate.

However, regardless of whether GIP is a causal factor in diabetes, the change in serum GIP concentration before and after surgery serves as a useful biomarker for the extent of foregut exclusion. If the surgical goal for non-obese metabolic patients is appropriate foregut exclusion, GIP measurement provides a valuable indicator of surgical efficacy. Notably, even in classic biliopancreatic diversion (BPD)—the procedure associated with the most profound and durable glucose-lowering effects—significant postoperative reductions in serum GIP levels have been documented.[7]

3. Epithelial-Mesenchymal Crosstalk and Reprogramming by Anastomosis

The small intestine possesses distinct regional identities along its length to facilitate sequential food processing and nutrient absorption. These regional characteristics are regulated by epithelial-mesenchymal crosstalk.[8] Subepithelial mesenchymal cells, characterized by gradients of Wnt and Shh signaling factors, stimulate epithelial growth and influence epithelial gene expression, shaping regional villus architecture along the anterior-posterior (AP) axis.[9]

Thus, when intestinal resection or bypass surgery connects disparate intestinal segments, new crosstalk between epithelial and mesenchymal tissues emerges. This interaction may induce reprogramming of epithelial identity along the AP axis. Supporting this concept, a study has reported that distal intestinal epithelium can acquire proximal identity following surgical resection and anastomosis.[10]

Such reprogramming offers a plausible explanation for the transient glycemic improvement observed soon after surgery followed by gradual recurrence of hyperglycemia months later. The underlying mechanism is likely enteroendocrine reprogramming due to altered epithelial-mesenchymal crosstalk, wherein proximal intestinal identity migrates distally and proliferates across the anastomosis, re-establishing a diabetogenic phenotype.[11]

4. Metabolic Outcomes Depend on Bypassed Foregut Length and Proximal Intestinal Identity at Anastomosis

 doi:10.20944/preprints202603.2347.v1

Maximizing glycemic improvement through metabolic surgery depends on achieving adequate foregut exclusion, which requires both sufficient bypass length and careful preparation of the proximal intestinal identity at the anastomosis.

The critical importance of biliopancreatic (BP) limb length has been well-documented.[12] Moreover, the nature of the proximal segment at anastomosis—whether the alimentary limb is connected to the stomach or duodenum—also affects glycemic outcomes. In non-obese type 2 diabetic patients, results of various operations can be roughly divided into disappointing groups (pylorus-preserving DJB and similar forms)[2,3,13–16] and acceptable groups (procedures ensuring adequate foregut exclusion and longer BP limb length).[4,5,17–21]

Even in Roux-en-Y gastric bypass (RYGB) with complete duodenal exclusion, despite separating the area of highest K-cell density from nutrient flow, a short BP limb can lead to postoperative GIP levels that are elevated compared to preoperative values.[22,23] This may result from the migration and proliferation of K-cells into the alimentary or common limb from the distal end of the BP limb—that is, from the proximal intestine near the anastomosis where K-cell density remains high—thereby reestablishing proximal identity and increasing serum GIP levels.[11]

5. Technical Aspects of Metabolic Surgery

Non-obese patients with type 2 diabetes often present with normal or even subnormal body weight. Therefore, in optimizing glycemic control, it is crucial to preserve adequate nutrient absorption and maintain normal digestive function to prevent unintended weight loss. This requires preservation of the pyloric sphincter and sufficient intestinal length to ensure proper nutrient assimilation and physiological continuity of digestion.

In RYGB, the alimentary limb carries food that does not mix with biliopancreatic secretions. While this can be beneficial for weight loss in morbidly obese patients, it may stimulate incretin secretion due to undiluted nutrient passage and is therefore not ideal for foregut exclusion in non-obese patients. Hence, a one-anastomosis loop configuration is more suitable for non-obese type 2 diabetic patients. N. Scopinaro also recommended extending the common channel when applying classic BPD to non-obese individuals, effectively creating a loop-like configuration as OAGB (Figure 1).[24] To illustrate the anatomical and mechanistic differences among pylorus-preserving DJB, RYGB, and SAPE, a comparative schematic is shown in Figure 2.

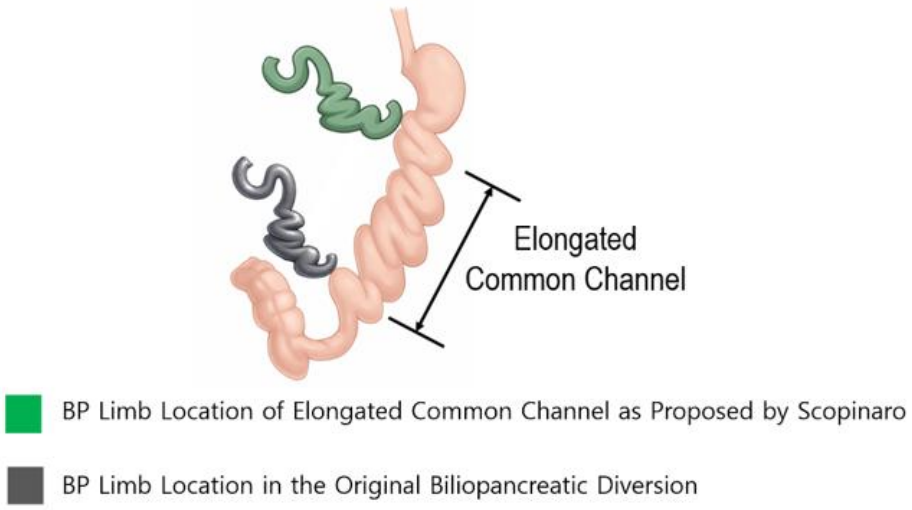

Figure 1. In classic BPD, elongation of the common channel is equivalent to OAGB. N. Scopinaro et al. suggested that a new BPD model with a longer common limb in the simply overweighted T2DM patients is now giving encouraging results.[24] Interestingly, in classic BPD, elongation of the common channel is equivalent to OAGB.

 doi:10.20944/preprints202603.2347.v1

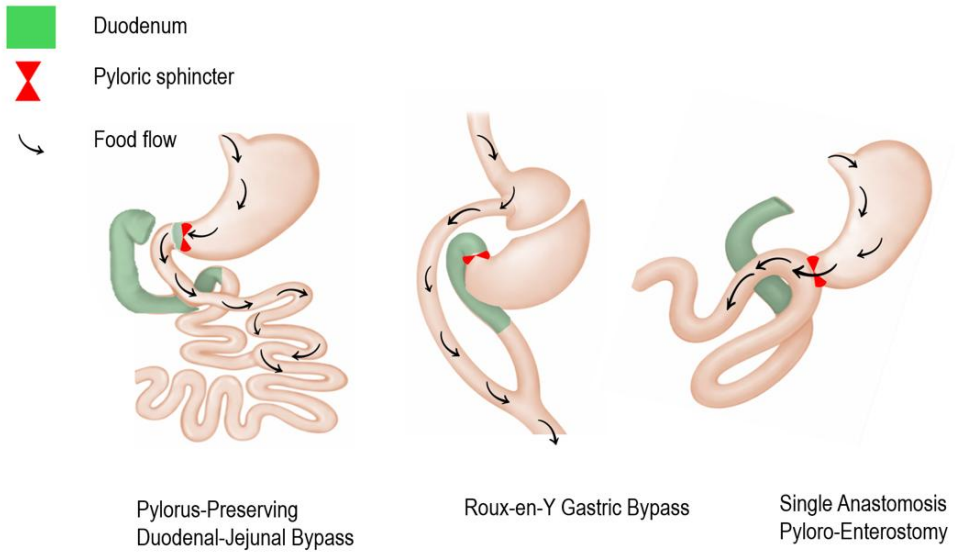

Figure 2. Comparative Schematic of Pylorus-Preserving Duodenal–Jejunal Bypass (DJB), Roux-en-Y Gastric Bypass (RYGB), and Single Anastomosis Pyloro-Enterostomy (SAPE). Green segments indicate the duodenum, red markers indicate the pyloric sphincter, and arrows represent nutrient flow. In pylorus-preserving DJB, nutrients continue to contact the duodenum, allowing exposure of enteroendocrine K-cells. In RYGB and SAPE, nutrient flow bypasses the duodenum, preventing direct contact with proximal K-cell–rich mucosa. SAPE uniquely preserves the pyloric sphincter while achieving complete duodenal exclusion.

To achieve complete duodenal exclusion while preserving pyloric function, the duodenal tissue connected to the pyloric sphincter must be completely removed, followed by anastomosis between the pyloric ring and the small intestine. The pyloric sphincter, embryologically derived from hypertrophied inner circular muscle of the stomach, is covered by gastric mucosa and is macroscopically distinguishable from the duodenum.[25] Importantly, interrupted sutures should be used to maintain sphincteric motility (Figure 3). In conclusion, for type 2 diabetic patients who do not require significant weight loss, the recommended procedure is SAPE, a loop-type configuration with an adequately long BP limb (usually 150~200 cm from the ligament of Treitz), complete duodenal exclusion, and interrupted anastomosis between the pylorus and the small intestine.

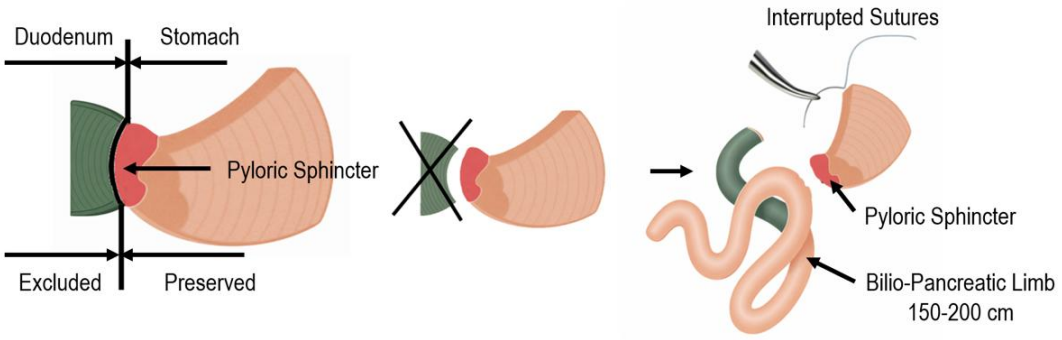

Figure 3. Schematic illustration of Single Anastomosis Pyloro-Enterostomy (SAPE). To achieve complete exclusion of the duodenum while preserving the function of the pyloric sphincter, it is recommended to completely remove the duodenal tissue connected to the pyloric sphincter and then anastomose the remaining segment to the small intestine. However, to maintain sphincteric function, the use of interrupted sutures is advisable.

 doi:10.20944/preprints202603.2347.v1

6. Technical Feasibility and Safety Considerations of SAPE

From a technical standpoint, SAPE requires meticulous identification of the pyloric ring and complete excision of duodenal mucosa attached to the pylorus. Potential procedure-specific risks include anastomotic stricture, delayed gastric emptying, bile reflux, and functional impairment of the pyloric sphincter.

However, these risks may be mitigated by several technical principles: use of interrupted sutures to preserve sphincteric motility, avoidance of excessive tension at the anastomosis, and careful calibration of the anastomotic diameter.

As with other advanced metabolic procedures, SAPE is expected to have a learning curve, and standardized training and procedural refinement will be necessary before widespread adoption.

7. Supporting Evidence from Animal and Human Studies

Many studies exploring the mechanisms of metabolic surgery reveal unexplained postoperative phenomena that can be clarified by applying the concept of epithelial reprogramming, where distal intestinal epithelium acquires proximal identity.[10,11]

A recent study highlighted the critical role of the BP limb: in diabetic rats with improved glycemia following DJB, excision of the BP limb abolished the metabolic benefits, suggesting that the BP limb has functions beyond mere nutrient diversion.[26] This loss of benefit likely results from migration and proliferation of epithelial lineage from proximal small intestine (similar to the phenomenon observed in Roux-en-Y gastric bypass with a very short BP limb) into the alimentary common channel.

Interestingly, gastrostomy feeding into the bypassed segment typically reverses the benefits of RYGB. However, duodenal-jejunal transit after RYGB did not impede glucose tolerance, which has long been puzzling.[27] This paradox can be resolved by recognizing that epithelial lineage propagation from proximal to distal intestine alters the functional identity of the anastomosed segments—such that duodenal characteristics are lost once replaced by jejunal epithelium.

Notably, the defining features of classic BPD are complete duodenal exclusion and a long BP limb.[7] Comparisons between modified and classic BPD procedures consistently emphasize the necessity of total duodenal exclusion and sufficient BP limb length. In pylorus-preserving BPDs with duodenal switch, outcomes were inferior despite identical BP limb length, likely due to residual duodenal mucosa at the anastomosis site.[28]

8. Limitations

This study is primarily a hypothesis-driven conceptual proposal based on existing physiological and experimental evidence. The absence of prospective randomized clinical outcome data represents a major limitation. Therefore, the metabolic and functional advantages of SAPE should be interpreted as theoretical expectations that require validation through well-designed clinical trials.

Although SAPE was developed based on clinical experience in Korean patients with a high prevalence of non-obese type 2 diabetes, the underlying physiological rationale is not population-specific, and the technique may be applicable to diverse ethnic groups with appropriate patient selection.

9. Conclusion

Single Anastomosis Pyloro-Enterostomy represents an evolution in metabolic surgery design, achieving complete duodenal exclusion with preservation of pyloric function incorporating a sufficiently long BP limb. By integrating anatomical precision, incretin physiology, and epithelial biology, SAPE may provide a durable and physiologically balanced approach for the treatment of non-obese T2DM. Further prospective trials are warranted to validate its long-term metabolic and functional outcomes.

Research Registration: Not applicable, this study is a conceptual proposal describing a surgical technique and does not involve clinical trial.

 doi:10.20944/preprints202603.2347.v1

Conflicts of Interest: The author declares no conflicts of interest related to this study.

Ethics Statement: The present study is based on a conceptual and technical proposal without involving human or animal experiments. Therefore, Institutional Review Board (IRB) approval was not required.

Patient Consent: Not applicable. This study does not involve human participants or patient data.

Funding Statement: No financial support or sponsorship was received for this work.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: The author conceptualized the SAPE surgical design, performed literature review, wrote the manuscript, and approved the final version.

Acknowledgments: The author expresses appreciation to the surgical team at our Hospital for their clinical collaboration and technical discussion that contributed to the conceptualization of the SAPE procedure.

References

1. Park SE, Ko SH, Kim JY, Kim K, Moon JH, Kim NH, Han KD, Choi SH, Cha BS. *Diabetes fact sheet in Korea 2024.* Diabetes Metab J. 2025;49:24–33. https://doi.org/10.4093/dmj.2025.0001
2. Lee HC, Kim MK, Kwon HS, Kim E, Song KH. Early changes in incretin secretion after laparoscopic duodenal-jejunal bypass surgery in type 2 diabetic patients. Obes Surg. 2010;20:1530–1535. https://doi.org/10.1007/s11695-010-0174-1
3. Heo Y, Ahn JH, Shin SH, Lee YJ. The effect of duodenojejunal bypass for type 2 diabetes mellitus patients below BMI 25 kg/m²: one-year follow-up. J Korean Surg Soc. 2013;85:109–115. https://doi.org/10.4174/jkss.2013.85.3.109
4. Kim MJ, Park HK, Byun DW, Suh KI, Hur KY. Incretin levels 1 month after laparoscopic single anastomosis gastric bypass in non-morbid obese type 2 diabetes patients. Asian J Surg. 2014;37:130–137. https://doi.org/10.1016/j.asjsur.2013.08.008
5. Kim MJ, Hur KY. Short-term outcomes of laparoscopic single anastomosis gastric bypass for the treatment of type 2 diabetes in lower BMI (<30 kg/m²) patients. Obes Surg. 2014;24(7):1044–1051. https://doi.org/10.1007/s11695-014-1207-9
6. Agha RA, Mathew G, Rashid R, Kerwan A, Al-Jabir A, Sohrabi C, Franchi T, Nicola M, Agha M; TITAN Group. Transparency in the reporting of artificial intelligence: the TITAN guideline. *Premier Journal of Science.* 2025;10:100082. doi:10.70389/PJS.100082.
7. Guidone C, Manco M, Valera-Mora E, Iaconelli A, Gniuli D, Mari A, et al. *Mechanisms of recovery from type 2 diabetes after malabsorptive bariatric surgery.* Diabetes. 2006;55:2025–2031. https://doi.org/10.2337/db05-1896
8. Crosnier C, Stamataki D, Lewis J. *Organizing cell renewal in the intestine: stem cells, signals and combinatorial control.* Nat Rev Genet. 2006;7:349–359. https://doi.org/10.1038/nrg1840
9. Maimets M, Pedersen MT, Guiu J, Dreier J, Thodberg M, Antoku Y, et al. *Mesenchymal-epithelial crosstalk shapes intestinal regionalisation via Wnt and Shh signalling.* Nat Commun. 2022;13:715. https://doi.org/10.1038/s41467-022-28397-0
10. Seiler KM, Waye SE, Kong W, Kamimoto K, Bajinting A, Goo WH, et al. *Single-cell analysis reveals regional reprogramming during adaptation to massive small bowel resection in mice.* Cell Mol Gastroenterol Hepatol. 2019;8:407–426. https://doi.org/10.1016/j.jcmgh.2019.05.008
11. Hur KY. Enteroendocrine reprogramming by altered epithelial–mesenchymal crosstalk in metabolic surgery. J Metab Bariatr Surg. 2024;13(1):1–7. https://doi.org/10.17476/jmbs.2024.13.1.1
12. Bruinsma FF, Nienhuijs SW, Liem RS, Greve JWM, van der Mheen PJ. The impact of longer biliopancreatic limb length on weight loss and comorbidity improvement at 5 years after primary Roux-en-Y gastric bypass surgery. Obes Surg. 2024;34:3236–3245. https://doi.org/10.1007/s11695-024-07009-4
13. Kindel TL, Martins PJ, Yoder SM, Jandacek RJ, Seeley RJ, D'Alessio DA, et al. *Bypassing the duodenum does not improve insulin resistance associated with diet-induced obesity in rodents.* Obesity (Silver Spring). 2011;19:380–387. https://doi.org/10.1038/oby.2010.173

 doi:10.20944/preprints202603.2347.v1

14. Petry TZ, Fabbrini E, Otoch JP, Carmona MA, Caravatto PP, Salles JE, et al. *Effect of duodenal-jejunal bypass surgery on glycemic control in type 2 diabetes: a randomized controlled trial.* Obesity (Silver Spring). 2015;23:1973–1979. https://doi.org/10.1002/oby.21187
15. Klein S, Fabbrini E, Patterson BW, Polonsky KS, Schiavon CA, Correa JL, et al. *Moderate effect of duodenal-jejunal bypass surgery on glucose homeostasis in patients with type 2 diabetes.* Obesity (Silver Spring). 2012;20:1266–1272. https://doi.org/10.1038/oby.2011.375
16. Grong E, Arbo IB, Thu OK, Kuhry E, Kulseng B, Mårvik R. The effect of duodenojejunostomy and sleeve gastrectomy on type 2 diabetes mellitus and gastrin secretion in Goto-Kakizaki rats. Surg Endosc. 2015;29:723–733. https://doi.org/10.1007/s00464-014-3704-4
17. Hu C, Zhang G, Sun D, Han H, Hu S. Duodenal-jejunal bypass improves glucose metabolism and adipokine expression independently of weight loss in a diabetic rat model. Obes Surg. 2013;23:1436–1444. https://doi.org/10.1007/s11695-013-0888-4
18. Sun D, Wang K, Yan Z, Zhang G, Liu S, Liu F, et al. Duodenal-jejunal bypass surgery up-regulates hepatic insulin signaling proteins and intestinal gluconeogenesis in diabetic Goto-Kakizaki rats. Obes Surg. 2013;23:1734–1742. https://doi.org/10.1007/s11695-013-0971-x
19. Speck M, Cho YM, Asadi A, Rubino F, Kieffer TJ. Duodenal-jejunal bypass protects GK rats from β-cell loss and aggravation of hyperglycemia and increases enteroendocrine cells coexpressing GIP and GLP-1. Am J Physiol Endocrinol Metab. 2011;300:E923–E932. https://doi.org/10.1152/ajpendo.00624.2010
20. Geloneze B, Geloneze SR, Chaim E, Hirsch FF, Felici AC, Lambert G, et al. Metabolic surgery for non-obese type 2 diabetes: incretins, adipocytokines, and insulin resistance changes in a 1-year controlled study. Ann Surg. 2012;256:72–78. https://doi.org/10.1097/SLA.0b013e3182536a92
21. Geloneze B, Geloneze SR, Fiori C, Stabe C, Tambascia MA, Chaim EA, et al. *Surgery for non-obese type 2 diabetic patients: an interventional study with duodenal-jejunal exclusion.* Obes Surg. 2009;19:1077–1083. https://doi.org/10.1007/s11695-009-9838-3
22. Laferrère B, Teixeira J, McGinty J, Tran H, Egger JR, Colarusso A, et al. *Effect of weight loss by gastric bypass versus hypocaloric diet on glucose and incretin levels in type 2 diabetes.* J Clin Endocrinol Metab. 2008;93:2479–2485. https://doi.org/10.1210/jc.2007-2851
23. Laferrère B, Heshka S, Wang K, Khan Y, McGinty J, Teixeira J, et al. *Incretin levels and effect are markedly enhanced 1 month after Roux-en-Y gastric bypass in obese patients with type 2 diabetes.* Diabetes Care. 2007;30:1709–1716. https://doi.org/10.2337/dc06-2314
24. Scopinaro N, Adami GF, Papadia FS, Camerini G, Carlini F, Briatore L, et al. *The effects of biliopancreatic diversion on type 2 diabetes mellitus in mild obesity and overweight.* Obes Surg. 2011;21(7):880–888. https://doi.org/10.1007/s11695-011-0402-7
25. Clement PB, Young RH. *The stomach and duodenum.* In: Mills SE, ed. *The Practice of Surgical Pathology.* 2nd ed. New York: Springer; 2009. p. 217–233. [Book chapter – no DOI]
26. Miyachi T, Nagao M, Shibata C, Kitahara Y, Tanaka N, Watanabe K, et al. *Biliopancreatic limb plays an important role in metabolic improvement after duodenal-jejunal bypass in a rat model of diabetes.* Surgery. 2016;159:1360–1371. https://doi.org/10.1016/j.surg.2015.11.022
27. Dolo PR, Yao L, Li C, Zhu X, Shi L, Widjaja J. Preserving duodenal-jejunal (foregut) transit does not impair glucose tolerance following gastric bypass in type 2 diabetes rats. Obes Surg. 2018;28:1313–1320. https://doi.org/10.1007/s11695-017-3012-7
28. Cossu ML, Noya G, Tonolo GC, Profili S, Meloni GB, Ruggiu M, et al. *Duodenal switch without gastric resection: results and observations after 6 years.* Obes Surg. 2004;14:1354–1359. https://doi.org/10.1381/0960892042583903