



# High levels of triglycerides, apolipoprotein B, and the number of colorectal polyps are risk factors for colorectal polyp recurrence after endoscopic resection: a retrospective study

Jia-Yu Du<sup>1#</sup>, Gui-Ying Huang<sup>2#</sup>, Yong-Chun Xie<sup>2</sup>, Nan-Xi Li<sup>2</sup>, Zhi-Wei Lin<sup>2</sup>, Li Zhang<sup>3</sup>

<sup>1</sup>School of Clinical Medicine, Chengdu Medical College, Chengdu, China; <sup>2</sup>School of Medicine, University of Electronic Science and Technology of China, Chengdu, China; <sup>3</sup>Department of Elderly Digestive, Sichuan Academy of Medical Sciences & Sichuan Provincial People's Hospital, Chengdu, China

**Contributions:** (I) Conception and design: JY Du, GY Huang; (II) Administrative support: L Zhang; (III) Provision of study materials or patients: JY Du, GY Huang, L Zhang; (IV) Collection and assembly of data: YC Xie, NX Li, ZW Lin; (V) Data analysis and interpretation: JY Du, GY Huang; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

<sup>#</sup>These authors contributed equally to this work and should be considered as co-first authors.

**Correspondence to:** Li Zhang. Department of Elderly Digestive, Sichuan Academy of Medical Sciences & Sichuan Provincial People's Hospital, 32 West Section 2, Yihuan Road, Chengdu 610072, China. Email: zhangligbyl@med.uestc.edu.cn.

**Background:** The recurrence of polyps after endoscopic treatment is a difficult problem and there may be an association between blood lipid levels and colorectal polyps, but this is controversial and the aim of this study is to explore the risk factors for colorectal polyp recurrence.

**Methods:** A total of 357 patients who underwent intestinal polypectomy from January 1, 2019 to June 1, 2020 in Sichuan Provincial People's Hospital were included in this retrospective study to analyze the potential association between blood indices and recurrence risk. Polyp recurrence was defined as the detection of 1 or more polyps at any time after polypectomy, regardless of site. Follow-up was performed through the electronic medical record system. Patients' age, gender, tobacco and alcohol liking, duration of follow-up, body mass index (BMI), polyp size, number, type of pathology, and lipid profiles (triglycerides, cholesterol, apolipoprotein B, and apolipoprotein A) were collected.

**Results:** Triglycerides ( $1.54 \pm 0.95$  vs.  $1.25 \pm 1.01$ ,  $P=0.036$ ) and apolipoprotein B ( $0.87 \pm 0.26$  vs.  $0.79 \pm 0.16$  mL,  $P=0.001$ ) were significantly different in both the recurrence and non-recurrence groups. Binary logistic regression identified 3 independent risk factors for recurrence: triglycerides [odds ratio (OR): 1.763, 95% confidence interval (CI): 1.003 to 3.098,  $P=0.049$ ], apolipoprotein B (OR: 5.438, 95% CI: 1.411 to 20.961,  $P=0.014$ ), and the number of polyps (OR: 2.540, 95% CI: 1.649 to 3.911,  $P<0.001$ ).

**Conclusions:** High levels of triglycerides, apolipoprotein B, and the number of colorectal polyps are risk factors for colorectal polyp recurrence after endoscopic resection. Therefore, for patients at high risk of polyp recurrence, we recommend aggressive control of triglyceride and apolipoprotein B levels.

**Keywords:** Colorectal polyps; hyperlipidemia; hypertriglyceridemia; dyslipidemia; apolipoprotein B (apoB)

Submitted May 10, 2022. Accepted for publication Jul 12, 2022.

doi: 10.21037/jgo-22-491

View this article at: <https://dx.doi.org/10.21037/jgo-22-491>

## Introduction

Colorectal cancer (CRC) is a common cancer, it is currently ranked third for incidence and first for mortality (1).

Nearly 900,000 people die of CRC every year (2). Early diagnosis and treatment of CRC can substantially improve 5-year survival rates (3). The National Polyp Study demonstrated the incidence of CRC was reduced by

76–90% after the colon was cleared via colonoscopy (4). Prevention and screening remain priorities for countries with high CRC incidence and mortality (5). Ideally, colorectal polyps are detected and removed early, which can reduce the risk of CRC (6). However, polyps can recur after endoscopic removal in 22–69% of patients (7–10). The risk of polyp recurrence has been associated with several factors, including gender, age, obesity index, use of tobacco and alcohol, polyp characteristics, and diet (11). For example, recurrence risk is higher among patients who are obese (12), who have been smoking for longer than 35 years [odds ratio (OR): 2.88, 95% confidence interval (CI): 2.06 to 4.01,  $P=0.001$ ] (13), or who drink alcohol (14).

Identifying non-invasive markers of polyp recurrence risk could help identify at-risk patients. Elevated triglyceride (TG) levels in serum are an independent risk factor for polyp recurrence in patients with advanced adenomas (15). Long-term lipid stimulation suppresses anti-tumor immune responses, facilitating colorectal tumorigenesis and distant metastasis, and suppresses immune infiltrating cell function in the tumor microenvironment, thereby accelerating tumor progression (16). On the other hand, several studies have suggested no association or a negative correlation between lipid levels in blood and risk of colorectal polyps or CRC (17–22). This may be a secondary outcome of changes in patient metabolism or nutrition, or there is detection bias and confounding bias. Therefore, more high-quality studies are needed to determine the association of blood lipids with colorectal polyp recurrence. This study had strict inclusion and exclusion criteria, the diagnosis was clear, all blood samples were tested by the same automatic analyzer in the Department of Laboratory Medicine of our hospital.

To help clarify these discrepancies, we retrospectively analyzed data from 357 patients at our hospital who underwent endoscopic resection of colorectal polyps in order to identify independent risk factors for colorectal polyp recurrence, including lipid levels in the blood. We present the following article in accordance with the STROBE reporting checklist (available at <https://jgo.amegroups.com/article/view/10.21037/jgo-22-491/rc>).

## Methods

### *Study design*

We used a cohort study to elaborate on risk factors for

postoperative recurrence of colorectal polyposis, in which we screened all 2,738 patients who underwent intestinal polypectomy for the first time between 1 January 2019 and 1 June 2020 in the Gastroenterology Department of Sichuan Provincial People's Hospital. To be enrolled, patients had to be 65 years or younger, and had to undergo at least one colonoscopic follow-up after polypectomy. Patients were then divided into recurrence versus nonrelapse groups, and clinical imaging and laboratory data were collected. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the ethics committee of Sichuan Provincial People's Hospital (No. 237 of 2022). The ethics committee of Sichuan Provincial People's Hospital waived the requirement for written informed consent since this was a retrospective study, it could not cause any adverse effects on included patients.

### *Patients*

Patients were excluded if they were diagnosed with high-grade intraepithelial neoplasia or carcinoma based on histopathology; if they had hypertension, diabetes, thyroid disease, or severe cardiac or pulmonary dysfunction; or if they had a history of familial adenomatous polyposis, inflammatory bowel disease, or a malignant tumor at any site. They were also excluded if they took hypolipidemic drugs before or during follow-up.

### *Surgical procedure*

All patients were placed on a liquid diet 24 hours before surgery, and bowel washing medication was taken to clean the bowel. The patients underwent general anesthesia enteroscopy surgery after fasting for 4 hours. The procedures were performed by physicians with experience in digestive endoscopy centers. Surgical modalities collectively included high-frequency electrocoagulation, argon plasma coagulation (APC), endoscopic mucosal resection (EMR), and endoscopic submucosal dissection (ESD). Finally, biopsy specimens were analyzed under the microscope by experienced pathologists.

### *Polyp classification and definition of polyp recurrence*

Pathology type reports were obtained from the hospital pathology department. According to the World Health Organization (WHO) classification, polyps can be classified

into 4 types: adenomatous polyps, inflammatory polyps, hyperplastic polyps, and hamartomatous polyps (23). High-grade intraepithelial neoplasia and carcinoma were excluded. Polyp recurrence was defined as the detection of 1 or more polyps at any time after polypectomy, irrespective of site (24).

### Blood lipid analysis

Fasting peripheral venous blood was analyzed within 24 hours after admission for TG, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), apolipoprotein A1 (apoA1), and apolipoprotein B (apoB). Patients were diagnosed with dyslipidemia if their TC level was  $\geq 5.2$  mmol/L; TG  $\geq 1.7$  mmol/L; HDL-C  $< 1.03$  mmol/L; LDL-C  $\geq 3.3$  mmol/L; apoA1  $< 1.05$  g/L; and apoB  $\geq 1.4$  g/L.

### Follow-up and data collection

Patients had at least one follow-up colonoscopy postoperatively. The following baseline data were collected for all patients: gender, age, body mass index (BMI), history of smoking and alcohol consumption, as well as polyp size, number, and histopathological type. Continuous or cumulative smoking for 6 months or more in a lifetime was defined as a smoker. Drinkers were defined as greater than 20 g/day for men and 10 g/day for women. The data were collected through an electronic medical record system.

### Bias

Patients with other underlying conditions were excluded according to the patient exclusion criteria, and other factors affecting the recurrence of intestinal polyps were avoided. In addition, to avoid selection bias, the researcher in charge of collecting the data was not aware of the grouping.

### Study size

The minimum sample size was established based on previous reports that hypertriglyceridemia predicts polyp recurrence (15), and we defined 0.3 mmol/L as the minimum clinical value for polyp recurrence caused by hypertriglyceridemia. These considerations, combined with a medium effect size of 0.5, power of 0.8, and alpha error of 0.2, meant that at least 76 patients had to be enrolled in the study.

### Statistical analysis

All analyses were performed using SPSS 26.0 (IBM Corp., Armonk, NY, USA). Normally distributed data were expressed as mean (standard deviation), otherwise, as median (interquartile range). Inter-group differences in continuous data (such as age, follow-up time, BMI, and blood lipid levels) were assessed for significance using the Student's *t*-test, analysis of variance (ANOVA), or the Kruskal-Wallis test, as appropriate. Differences in categorical data were assessed using the chi-squared test. The risk of polyp recurrence is associated with several factors, including gender, age, obesity index, tobacco and alcohol use, polyp characteristics, and diet. Binary logistic regression was used to identify independent risk factors for polyp recurrence. Differences of  $P < 0.05$  were considered statistically significant and  $P < 0.05$  was two-sided. For random missing data were handled by direct deletion.

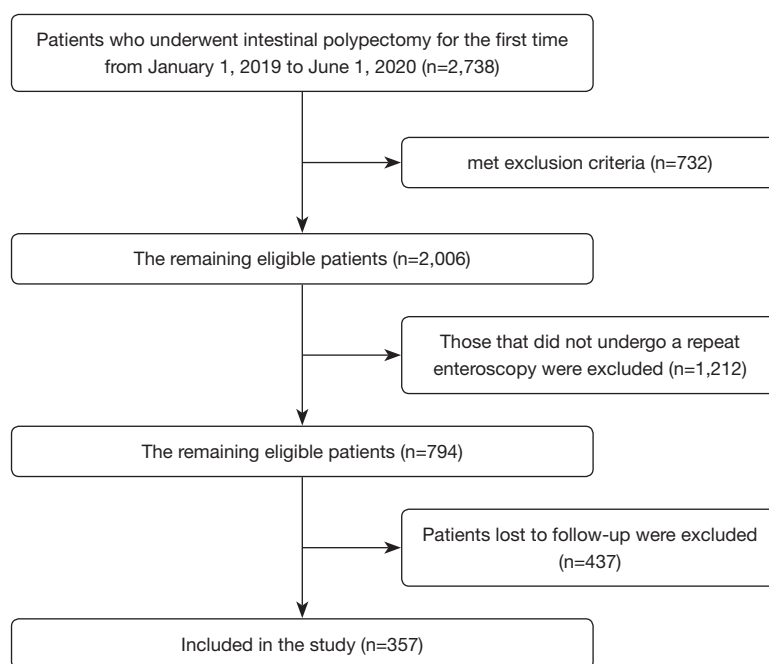
### Results

Of the 2,738 patients screened, 1,212 were excluded because no follow-up enteroscopy data were available, 437 were lost to follow-up, and 732 met the exclusion criteria. Finally, 357 patients were included in the study, of whom 225 (63%) were men and 161 (45%) experienced recurrence during follow-up, which lasted a mean of 12 months (*Figure 1*). Relatively small percentages of patients reported smoking (22%) or drinking (21%) (*Table 1*).

Among the 357 included cases, the follow-up time was  $12.0 \pm 3.0$  months in the recurrence group and  $12.0 \pm 0.75$  months in the non-recurrence group. Patients who experienced recurrence or not were similar in gender distribution, mean age, mean BMI, and smoking or alcohol consumption (*Table 2*). Similarly, the 2 groups of patients did not differ significantly in the size or histopathological type of polyps (*Table 3*). In contrast, the number of polyps was statistically significantly different between the two groups.

In addition, TGs ( $1.54 \pm 0.95$  vs.  $1.25 \pm 1.01$  mmol/L,  $P = 0.036$ ) and apoB ( $0.87 \pm 0.26$  vs.  $0.79 \pm 0.16$  g/L,  $P = 0.001$ ) were significantly different in both the recurrence and non-recurrence groups. (*Table 4*). The 2 groups of patients did not differ significantly in levels of TC ( $4.97 \pm 0.96$  vs.  $4.60 \pm 1.00$  mmol/L,  $P = 0.895$ ), HDL-C ( $1.27 \pm 0.42$  vs.  $1.29 \pm 0.44$  mmol/L,  $P = 0.762$ ) or LDL-C ( $2.30 \pm 0.97$  vs.  $2.17 \pm 0.78$  mmol/L,  $P = 0.146$ ), or apoA1 ( $1.33 \pm 0.35$  vs.  $1.32 \pm 0.32$  g/L,  $P = 0.741$ ).

Binary logistic regression identified the following independent risk factors for polyp recurrence (*Table 5*): high



**Figure 1** Inclusion and exclusion process of study participants. n, number.

**Table 1** Data loss

Reason	N
No follow-up enteroscopy data	1,212
Lost to follow-up	437
Met exclusion criteria	732

N, number.

**Table 2** Characteristics of patients with colorectal polyps, stratified by whether they experienced polyp recurrence during follow-up

Characteristic	Recurrence (n=161)	No recurrence (n=196)	P value*
Gender			0.097
Male	109	116	
Female	52	80	
Age, years	50.2 (12.0)	48.8 (12.0)	0.121
Follow-up, months	12.0 (3.0)	12.0 (0.75)	0.411
History of smoking	42	37	0.103
History of drinking	37	39	0.479
BMI, kg/m <sup>2</sup>	23.1 (4.0)	23.0 (3.9)	0.946

Values are n or mean (standard deviation). \*, based on Student's *t*-test or the Mann-Whitney test (continuous variables) or Fisher's exact test (categorical variables). n, number; BMI, body mass index.

**Table 3** Comparison of polyp characteristics between patients who experienced polyp recurrence or not

Characteristics	Recurrence (n=161)	No recurrence (n=196)	P value*
Polyp type, n			0.081
Hyperplastic	85	96	
Inflammatory	10	10	
Tubular adenoma	20	12	
Hamartomatous	1	4	
N/A	45	74	
Polyp size, n			0.793
<10 mm	126	157	
10–19 mm	24	24	
≥20 mm	8	9	
N/A	3	6	
Polyp number, n			<0.001
1	33	75	
2	30	45	
≥3	96	72	
N/A	2	4	

\*, based on Student's *t*-test or the Mann-Whitney test (continuous variables) or Fisher's exact test (categorical variables). n, number; N/A, unclear.

**Table 4** Lipid levels in blood of patients who experienced polyp recurrence or not

Lipid species	Recurrence (n=161)	No recurrence (n=196)	P value*
TG, mmol/L	1.54 (0.95)	1.25 (1.01)	0.036
TC, mmol/L	4.97 (0.96)	4.60 (1.00)	0.895
HDL-C, mmol/L	1.27 (0.42)	1.29 (0.44)	0.762
LDL-C, mmol/L	2.30 (0.97)	2.17 (0.78)	0.146
Apolipoprotein A1, g/L	1.33 (0.35)	1.32 (0.32)	0.741
Apolipoprotein B, g/L	0.87 (0.26)	0.79 (0.16)	0.001

Values are mean (standard deviation). \*, based on Student's *t*-test or the Mann-Whitney test (continuous variables) or Fisher's exact test (categorical variables). n, number; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

**Table 5** Multivariate analysis of risk factors associated with polyp recurrence (n=357 patients)\*

Parameters	OR (95% CI)	P value
Gender	0.724 (0.446–1.175)	0.191
Age	1.017 (0.994–1.042)	0.152
Follow-up time	0.978 (0.925–1.033)	0.420
Smoking	0.669 (0.329–1.363)	0.268
Drinking	1.308 (0.642–2.664)	0.459
BMI $\geq 25$ kg/m <sup>2</sup>	1.002 (0.631–1.59)	0.995
Polyp size $\geq 20$ mm	1.073 (0.404–2.848)	0.888
Polyp number $\geq 3$	2.540 (1.649–3.911)	<0.001
TG ( $\geq 1.7$ mmol/L)	1.763 (1.003–3.098)	0.049
TC ( $\geq 5.2$ mmol/L)	1.639 (0.923–2.908)	0.092
HDL-C	1.309 (0.577–2.971)	0.519
LDL-C	0.980 (0.926–1.037)	0.485
Apolipoprotein A1	1.300 (0.382–4.432)	0.675
Apolipoprotein B	5.438 (1.411–20.961)	0.014

\*, based on binary regression analysis. OR, odds ratio; CI, confidence interval; BMI, body mass index; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

TG (OR: 1.763, 95% CI: 1.003 to 3.098, P=0.049), high apoB (OR: 5.438, 95% CI: 1.411 to 20.961, P=0.014), and the presence of at least 3 polyps (OR: 2.540, 95% CI: 1.649 to 3.911, P<0.001).

## Discussion

Here, we provide evidence that hypertriglyceridemia,

high apoB, and the number of polyps are independent risk factors for polyp recurrence. To our knowledge, this is the first report linking high apoB to polyp recurrence. A recent meta-analysis confirmed that elevated TG is associated with the development of colorectal polyps, which is consistent with our study (25). Our findings are consistent with studies suggesting that hypertriglyceridemia promotes the formation of reactive oxygen species that damage DNA and thereby initiate CRC (26), and that this and other abnormalities of lipid metabolism suppress anti-tumor immune responses (16).

Early polyp detection and removal are effective for preventing CRC. Adenomatous polyps can progress to malignancy via the adenoma-carcinoma pathway (27,28), while hyperplastic polyps can progress to malignancy via the serrated or microsatellite instability pathway (29). A hamartoma adenoma-carcinoma sequence has also been suggested (30). Therefore, identifying patient characteristics associated with the risk of polyp recurrence may help reduce the risk of CRC.

Studies have explored potential correlations between blood lipid indices and the risk of CRC, but they have been unable to separate potential associations from other dietary or metabolic confounders (17,18,31). Similarly, studies have failed to unambiguously identify associations between dyslipidemia and the risk of colorectal polyps (32,33). Our study provides strong evidence linking hypertriglyceridemia to polyp recurrence. At least 3 mechanisms may underlie this association. One is that hypertriglyceridemia causes hyperinsulinemia and insulin resistance, which drives the proliferation of normal and cancer cells in the large intestine (34). Another mechanism is that dyslipidemia creates a tumorigenic environment by increasing levels of interleukin-6 (IL-6, tumor necrosis

factor- $\alpha$  (TNF- $\alpha$ ) and other inflammatory cytokines (35). Finally, Liu *et al.* suggested that TG may affect polyp recurrence by increasing the proliferative capacity of cancer stem cells or stimulating signaling pathways that promote cell invasion (15).

Our data also link high levels of apoB to polyp recurrence. ApoB is a structural component of chylomicrons as well as lipoproteins of various densities (36), so it plays an important role in atherogenesis (36,37). Given one study linking elevated LDL-C to the formation of colorectal polyps (38), we suggest that apoB may influence polyp recurrence by affecting the metabolism of LDL-C. Elevated levels of LDL-C have been linked to oxidative stress and inflammatory responses in the intestine (35,39). By reducing levels of LDL-C in serum, statins may inhibit polyp formation (40-42), which may help explain their ability to slow the growth of several types of colorectal tumors (43-46). Future studies should explore whether other hypolipidemic agents such as fibrates can inhibit polyp formation and CRC.

Our findings should be interpreted with caution given that our patient sample was relatively small and came from a single center. The numbers of polyps differed between cases that experienced recurrence or not, which may have confounded our analyses. Despite these limitations, our data suggest that administering hypolipidemic drugs to individuals with hyperlipidemia may help reduce the risk of polyp recurrence and thereby CRC.

## Conclusions

High levels of TGs, apoB, and the number of colorectal polyps are risk factors for colorectal polyp recurrence after endoscopic resection. Therefore, for patients at high risk of polyp recurrence, we recommend aggressive control of TG and apoB levels.

## Acknowledgments

*Funding:* This work was supported by Key Research and Development Projects of Sichuan Science and Technology Department (grant Nos. 22ZDYF1691 and 2018FZ0062).

## Footnote

*Reporting Checklist:* The authors have completed the STROBE reporting checklist. Available at <https://jgo.amegroups.com/article/view/10.21037/jgo-22-491/rc>

*Data Sharing Statement:* Available at <https://jgo.amegroups.com/article/view/10.21037/jgo-22-491/dss>

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <https://jgo.amegroups.com/article/view/10.21037/jgo-22-491/coif>). All authors report that this work was supported by Key Research and Development Projects of Sichuan Science and Technology Department (grant Nos. 22ZDYF1691 and 2018FZ0062). The authors have no other conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the ethics committee of Sichuan Provincial People's Hospital (No. 237 of 2022). The ethics committee of Sichuan Provincial People's Hospital waived the requirement for written informed consent since this was a retrospective study, it could not cause any adverse effects on included patients.

*Open Access Statement:* This is an Open Access article distributed in accordance with the Creative Commons Attribution-NonCommercial-NoDerivs 4.0 International License (CC BY-NC-ND 4.0), which permits the non-commercial replication and distribution of the article with the strict proviso that no changes or edits are made and the original work is properly cited (including links to both the formal publication through the relevant DOI and the license). See: <https://creativecommons.org/licenses/by-nc-nd/4.0/>.

## References

1. Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. *CA Cancer J Clin* 2021;71:209-49.
2. Brody H. Colorectal cancer. *Nature* 2015;521:S1.
3. Nelson RS, Thorson AG. Colorectal cancer screening. *Curr Oncol Rep* 2009;11:482-9.
4. Winawer SJ, Zauber AG, Ho MN, et al. Prevention of colorectal cancer by colonoscopic polypectomy. The National Polyp Study Workgroup. *N Engl J Med* 1993;329:1977-81.
5. Kanth P, Inadomi JM. Screening and prevention of colorectal cancer. *BMJ* 2021;374:n1855.

6. Holme Ø, Bretthauer M, Fretheim A, et al. Flexible sigmoidoscopy versus faecal occult blood testing for colorectal cancer screening in asymptomatic individuals. *Cochrane Database Syst Rev* 2013;(9):CD009259.
7. Christie JP. Colonoscopic excision of large sessile polyps. *Am J Gastroenterol* 1977;67:430-8.
8. Bedogni G, Bertoni G, Ricci E, et al. Colonoscopic excision of large and giant colorectal polyps. Technical implications and results over eight years. *Dis Colon Rectum* 1986;29:831-5.
9. Arebi N, Swain D, Suzuki N, et al. Endoscopic mucosal resection of 161 cases of large sessile or flat colorectal polyps. *Scand J Gastroenterol* 2007;42:859-66.
10. Boix J, Lorenzo-Zúñiga V, Moreno de Vega V, et al. Endoscopic removal of large sessile colorectal adenomas: is it safe and effective? *Dig Dis Sci* 2007;52:840-4.
11. Hao Y, Wang Y, Qi M, et al. Risk Factors for Recurrent Colorectal Polyps. *Gut Liver* 2020;14:399-411.
12. Schatzkin A, Lanza E, Corle D, et al. Lack of effect of a low-fat, high-fiber diet on the recurrence of colorectal adenomas. Polyp Prevention Trial Study Group. *N Engl J Med* 2000;342:1149-55.
13. Reid ME, Marshall JR, Roe D, et al. Smoking exposure as a risk factor for prevalent and recurrent colorectal adenomas. *Cancer Epidemiol Biomarkers Prev* 2003;12:1006-11.
14. Tiemersma EW, Wark PA, Ocké MC, et al. Alcohol consumption, alcohol dehydrogenase 3 polymorphism, and colorectal adenomas. *Cancer Epidemiol Biomarkers Prev* 2003;12:419-25.
15. Liu B, Wen P, Gu X, et al. Elevated serum triglyceride predicts recurrence of colorectal polyps in patients with advanced adenomas. *Lipids Health Dis* 2020;19:211.
16. Chen K, Guo J, Zhang T, et al. The Role of Dyslipidemia in Colitis-Associated Colorectal Cancer. *J Oncol* 2021;2021:6640384.
17. Chung YW, Han DS, Park YK, et al. Association of obesity, serum glucose and lipids with the risk of advanced colorectal adenoma and cancer: a case-control study in Korea. *Dig Liver Dis* 2006;38:668-72.
18. Kono S, Ikeda N, Yanai F, et al. Serum lipids and colorectal adenoma among male self-defence officials in northern Kyushu, Japan. *Int J Epidemiol* 1990;19:274-8.
19. Saydah SH, Platz EA, Rifai N, et al. Association of markers of insulin and glucose control with subsequent colorectal cancer risk. *Cancer Epidemiol Biomarkers Prev* 2003;12:412-8.
20. Tsushima M, Nomura AM, Lee J, et al. Prospective study of the association of serum triglyceride and glucose with colorectal cancer. *Dig Dis Sci* 2005;50:499-505.
21. Dayton S, Pearce ML, Hashimoto S, et al. A controlled clinical trial of a diet high in unsaturated fat in preventing complications of atherosclerosis. *Circulation* 1969;40:II-1-II-63.
22. WHO cooperative trial on primary prevention of ischaemic heart disease with clofibrate to lower serum cholesterol: final mortality follow-up. Report of the Committee of Principal Investigators. *Lancet* 1984;2:600-4.
23. Atkin WS, Valori R, Kuipers EJ, et al. European guidelines for quality assurance in colorectal cancer screening and diagnosis. First Edition--Colonoscopic surveillance following adenoma removal. *Endoscopy* 2012;44 Suppl 3:SE151-63.
24. Jacobs ET, Martínez ME, Alberts DS, et al. Association between body size and colorectal adenoma recurrence. *Clin Gastroenterol Hepatol* 2007;5:982-90.
25. Zhang R, Yin J, Huo C, et al. The Relationship Between Colorectal Polyps and Serum Lipid Levels: A Systematic Review and Meta-analysis. *J Clin Gastroenterol* 2022;56:654-67.
26. Bruenderman EH, Martin RC 2nd. High-risk population in sporadic pancreatic adenocarcinoma: guidelines for screening. *J Surg Res* 2015;194:212-9.
27. Morson B. Polyp-cancer sequence in large bowel. *Proc R Soc Med* 1974;67:451-7.
28. Kronborg O, Fenger C. Clinical evidence for the adenoma-carcinoma sequence. *Eur J Cancer Prev* 1999;8 Suppl 1:S73-86.
29. Hyman NH, Anderson P, Blasyk H. Hyperplastic polyposis and the risk of colorectal cancer. *Dis Colon Rectum* 2004;47:2101-4.
30. Bosman FT. The hamartoma-adenoma-carcinoma sequence. *J Pathol* 1999;188:1-2.
31. Rose G, Blackburn H, Keys A, et al. Colon cancer and blood-cholesterol. *Lancet* 1974;1:181-3.
32. Pan J, Cen L, Xu L, et al. Prevalence and risk factors for colorectal polyps in a Chinese population: a retrospective study. *Sci Rep* 2020;10:6974.
33. Kim YJ, Lee KJ, Park SY, et al. Association between Dyslipidemia and the Prevalence of Colon Polyps Based on a Health Evaluation of Subjects at a Hospital. *Korean J Fam Med* 2014;35:143-51.
34. Burchfiel CM, Abbott RD, Curb JD, et al. Association of insulin levels with lipids and lipoproteins in elderly Japanese-American men. *Ann Epidemiol* 1998;8:92-8.

35. Esteve E, Ricart W, Fernández-Real JM. Dyslipidemia and inflammation: an evolutionary conserved mechanism. *Clin Nutr* 2005;24:16-31.
36. Sparks JD, Sparks CE. Apolipoprotein B and lipoprotein metabolism. *Adv Lipid Res* 1985;21:1-46.
37. Olofsson SO, Borèn J. Apolipoprotein B: a clinically important apolipoprotein which assembles atherogenic lipoproteins and promotes the development of atherosclerosis. *J Intern Med* 2005;258:395-410.
38. Xie C, Wen P, Su J, et al. Elevated serum triglyceride and low-density lipoprotein cholesterol promotes the formation of colorectal polyps. *BMC Gastroenterol* 2019;19:195.
39. Katzke VA, Sookthai D, Johnson T, et al. Blood lipids and lipoproteins in relation to incidence and mortality risks for CVD and cancer in the prospective EPIC-Heidelberg cohort. *BMC Med* 2017;15:218.
40. Siddiqui A, Nazario HE, Patel M, et al. Reduction in low-density lipoprotein cholesterol levels during statin therapy is associated with a reduced incidence of advanced colon polyps. *Am J Med Sci* 2009;338:378-81.
41. Swamy MV, Patlolla JM, Steele VE, et al. Chemoprevention of familial adenomatous polyposis by low doses of atorvastatin and celecoxib given individually and in combination to APCMin mice. *Cancer Res* 2006;66:7370-7.
42. Teraoka N, Mutoh M, Takasu S, et al. Inhibition of intestinal polyp formation by pitavastatin, a HMG-CoA reductase inhibitor. *Cancer Prev Res (Phila)* 2011;4:445-53.
43. Huang EH, Johnson LA, Eaton K, et al. Atorvastatin induces apoptosis in vitro and slows growth of tumor xenografts but not polyp formation in MIN mice. *Dig Dis Sci* 2010;55:3086-94.
44. Jung YS, Park CH, Eun CS, et al. Statin use and the risk of colorectal adenoma: A meta-analysis. *J Gastroenterol Hepatol* 2016;31:1823-30.
45. Broughton T, Sington J, Beales IL. Statin use is associated with a reduced incidence of colorectal adenomatous polyps. *Int J Colorectal Dis* 2013;28:469-76.
46. Siddiqui A, Pandove S, Mahgoub A, et al. The long-term use of statins is associated with a decreased incidence of advanced adenomatous colon polyps. *Am J Gastroenterol* 2008;103:S544.

(English Language Editor: J. Jones)

**Cite this article as:** Du JY, Huang GY, Xie YC, Li NX, Lin ZW, Zhang L. High levels of triglycerides, apolipoprotein B, and the number of colorectal polyps are risk factors for colorectal polyp recurrence after endoscopic resection: a retrospective study. *J Gastrointest Oncol* 2022;13(4):1753-1760. doi: 10.21037/jgo-22-491