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肠道菌群的改变与结肠息肉相关性的研究进展

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摘　要

肠道微生物一直被认为是机体的“隐形器官”，在维护人类健康方面发挥着不可替代的功能。近年的研究发现，肠道微生态与各种疾病之间存在一定的相关性，而肠道微生态与结肠息肉之间的相关性是目前研究的热点。结肠息肉是一种常见的消化道疾病，其发病率逐年升高。它不仅会对病人的肠道功能造成损害，而且某些息肉还会发生恶性转化，对人体健康构成极大的危害。而肠道微生物是机体多种生理活动的关键成分，一旦其失衡，即微生态失衡，将引起一系列肠道甚至整个机体的病理生理改变。

关键词

肠道菌群，结肠息肉，肥胖症

Research Progress on the Correlation between Changes in Intestinal Microbiota and Colon Polyps

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Abstract

Gut microbes have long been considered the “invisible organs” of the body and play an irreplaceable function in maintaining human health. Recent studies have found some correlation between intestinal microecology and various diseases, and the correlation between intestinal microecology and colon polyps is a current research hotspot. Colonic polyps are a common digestive tract disease, and their incidence increases year by year. It will not only cause damage to the patient’s intestinal function,

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but also cause some polyps to undergo malignant transformation, posing great harm to human health. Intestinal microbes are the key components of various physiological activities of the body. Once they are unbalanced, that is, the microecological imbalance, it will cause a series of pathophysiological changes in the intestinal tract and even the whole body.

Keywords

Gut Microbe, Colonic Polyp, Obesity

1. 肠道菌群的概况

人类的身体里有数以兆计的微生物，大多数都在肠胃里，他们的数目超过了人全部的细胞。在肠道内，这些菌群与身体的新陈代谢有着紧密的联系[1]。研究发现，肠道菌群与人体多种疾病如糖尿病、心血管疾病、代谢性疾病、肠道炎症及肿瘤等的发生发展相关[2]。

1.1. 肠道菌群的组成

肠道菌群作为寄生在人体肠道中的“器官”，对机体的健康与行为起着极为重要的调控作用。代谢产物短链脂肪酸(SCFAs)在糖尿病和肥胖等代谢性疾病中发挥重要作用[3]。通过影响三种物质的代谢，生成各种代谢产物，如短链脂肪酸，从而影响糖尿病和肥胖等代谢性疾病的发病[4]。成年人的肠道中含有约 330 万个基因，相当于人体基因组的 150~200 倍[5]。肠道微生物包括 3 大类微生物，包括益生菌，致病菌和条件致病菌[6]。益生菌是肠道中的优势微生物，对维持肠道稳态具有重要作用[7]。目前被鉴定的肠道菌群主要包括：硬壁菌、拟杆菌门、变形菌门、放线菌、疣状菌门等，且以硬壁菌和拟杆菌门为主(>98%) [8]。肠球菌、肠杆菌等条件性病原菌通常是不会致病的。但是，当到达一定的病因时，例如：免疫功能下降、寄生部位发生变化、菌群失调等，就会引起它的侵袭[9]。

1.2. 肠道菌群的作用

肠道菌群对宿主有多方面重要的作用：包括维护宿主正常组织学和解剖学结构，形成菌群屏障，抵御外来细菌感染；为宿主提供维生素、氨基酸、脂质和碳水化合物等营养物质；直接参与内源蛋白质等物质代谢，加快肠道蠕动，促进营养消化吸收；预防炎症及癌症发生等，如乳酸菌可激活机体细胞免疫和体液免疫应答，尤其是细胞免疫激活及产生的细胞因子，发挥抗肿瘤作用[10]。肠道中的细菌可以分为两类：一类是位于肠腔内的细菌，另一类是位于肠道粘膜层的细菌。粪样细菌在一定程度上能反应肠道内的菌群结构，而不能反应肠道上皮及隐窝内的微生物[11]。已有研究表明[12]，肠道上皮细胞与肠道上皮细胞的菌群结构有较大差别，肠道上皮细胞对肠道微生物的敏感性较高，且肠道上皮细胞的结构相对稳定，故本项目拟从肠道粘膜及肠道微生物两个层面，来探索外源性及内源性因子对息肉发病的作用机制。肠道微生物对机体代谢具有重要调节作用。它不但可以帮助人体吸收糖类物质，还可以帮助维持稳定的肠道屏障功能，建立起抵抗病原体侵袭和定殖的免疫应答[13]。肠道长期持续的炎症刺激可导致肠粘膜屏障的损伤和破坏，从而导致结肠息肉的形成。研究发现，肠道微生物及代谢物与结肠息肉的发生、发展有密切关系，其中以腺瘤样息肉最为突出[14]。

2. 肠道菌群与结肠息肉的相关性

2.1. 结肠息肉中肠道菌群的作用

研究显示，肠道微生物与 CAP 发病密切相关。前期研究发现，肠息肉病人大便标本中拟杆菌及双歧杆菌属(双歧杆菌)丰度与大肠息肉发病风险呈正相关，而乳杆菌及真菌会对肠道起保护作用[15]。大量研究显示，结直肠癌患者的肠道微生态存在着显著的差异，提示结直肠癌中存在着显著的肠道菌群失调，影响了与其相关的代谢物质的含量，进而影响了肠道的正常功能[16]。菌群紊乱主要表现为：益生菌数量减少，病原菌增多，以及肠道微生物的多样性下降[17]。肥胖时，肠道微生态失衡，胰岛素抵抗，微炎症等异常[18]。按主要菌群类型可将大肠划分为 3 个“肠型”[19]：肠型 1 以拟杆菌为主；肠型 2 以普氏菌为主；肠道 3 型以厚壁菌所占的比例来划分，以瘤胃球菌为主。CAP 及 CRC 病人以类杆菌占优势的肠道类型为主，而正常人则以普氏菌属及瘤胃球菌为主[20]。我们前期通过宏基因组关联分析发现，梭杆菌(Fusobacterium)是 CAP 的主要致病菌，且其丰度较无病理变化的正常组织显著增高[21]。当前有关研究显示，粪便中存在具核梭杆菌，可能是由腺瘤向癌转化的一个潜在风险因子[22]。

近期有研究发现肠道菌群紊乱与大肠腺瘤易感性相关，但仍需进一步研究以探讨菌群与酸性磷酸酶的关系[23]。我们前期研究发现，CAP 病人与正常人群粪便中细菌群落结构没有明显差别，而细菌群落结构(beta 多样性)却有很大差别，主要集中于变形杆菌(*Proteobacteria*)，并不受病人性别的影响，主要包括：假单胞菌科、军团菌科、嗜盐菌科、肠杆菌科、沙雷菌属、沙门菌、泛菌属、大肠埃希菌属、大肠埃希菌属、曲霉菌属、志贺菌属等[24]。近期研究发现，肠道微生物与大肠癌发生发展密切相关[25]。肠息肉病人与健康人比较，其肠道菌群特点存在差异[26]。通过对粪菌进行分析，研究者们发现，不同种类的大肠息肉病人的肠道菌群存在明显差异[27]。Watson 等人在上述研究中发现，与健康体检者相比，在腺瘤病人的肠道粘膜中，细菌 alpha 的多样性呈降低的趋势，但并无统计上的差别[28]。Peters 等的研究还表明，增殖性和非增殖性的锯齿状瘤没有明显的区别，而进展期的结肠腺瘤的肠道微生物群落则有很大的不同[29]。而 α 多样性差异在进展期腺瘤病例中表现明显[30]。最新研究表明，肠道微生态不仅可以调节机体的能量代谢，还可以诱导炎性介质的高表达、胰岛素抵抗，进而诱发肠道微生态失衡[31]。研究显示，大肠息肉，特别是腺瘤样息肉，其发病与肠道微生态失衡及代谢物关系密切[32]。结肠息肉以腺瘤性息肉为主，而结肠腺瘤性息肉(CAP)与大肠癌的发病关系密切，被视为癌前病变[33]。息肉越多、越大，越有可能发生癌症。随着绒毛数量的增加，息肉的癌变几率也会越来越高，主要有管状腺瘤、绒毛管状腺瘤和绒毛状腺瘤[34]。结直肠腺瘤患者常伴有肠道微生态紊乱，我们前期研究发现，结直肠腺瘤中克雷伯杆菌属、梭杆菌属丰度高于正常人，而乳杆菌、双歧杆菌含量却明显下降[35]。

2.2. 结肠息肉患者肠道菌群特点

研究发现，结直肠癌病人的肠道微生物群落结构与正常人群存在显著差异，且结肠癌病人之间也存在显著差异。不同部位和病理类型的大肠息肉病人肠道微生物的丰度和多样性存在差异[36]。特异性菌群数量的改变可以作为一种新的生物学标记，用于筛选包括 ACP、CRC、IBD、肠易激综合症等消化道疾病[37]。目前的理论研究表明，结直肠癌的发生与发展是无法避免的，对其进行积极的治疗是防止结直肠癌发生的一个重要措施[38]。研究表明，结直肠癌及癌前病变病人的肠道微生物群落结构出现了变化，双歧杆菌属、乳杆菌数量下降，肠球菌、肠杆菌和酵母菌增多[39]，在肠道菌群失调的情况下，会发生条件性病原的作用，因为肠道的黏液通透性增加，导致了细菌的移动，免疫系统被激活，从而使炎症细胞释放出了大量的炎症因子，并与细菌的毒性代谢物共同组成了一个肠道炎性微环境[40]。目前研究发现，肠道微生物代谢物(SCFAs，包括丁酸、丙酸、甲酸、乙酸、戊酸等)的变化也与结肠息肉的发生有关[41]。

丁酸盐(Butyrate)作为被研究最为广泛的短链脂肪酸之一，有着诸多重要功效。它能够减少肠道上皮细胞的 DNA 氧化损伤，引导那些遭受 DNA 损伤的细胞走向分化凋亡，还能抑制肿瘤细胞的生长与增殖，降低具有促癌作用酶的活性。凭借这些作用，丁酸盐得以保护肠壁，抑制肠道炎性反应，对预防和对抗如 CRC (结直肠癌)等疾病发挥着积极作用[42]。我们前期研究发现，肠道微生态失调可导致 SCFAs 生成减少，抑制炎症反应，进而导致结直肠息肉乃至大肠癌的发生发展[43]。丁酸可通过抑制 NF-κB，来减少前炎症因子如白介素-6、12 等的表达，从而影响炎症反应。也可以通过调节肠上皮细胞间紧密连接的基因来保持肠粘膜通透性[44]。我们前期研究发现，醋酸能与肠上皮细胞表面 GPR43 结合，引起 Ca^{2+}升高，进而介导 K^{+}依赖的胞膜超极化，活化 NLRP3 炎症小体，进而活化半胱氨酸蛋白酶-1，促进白介素-18 的分泌，保护肠上皮结构完整[45]。文献报道 SCFAs 也能调控结肠粘膜上皮细胞的增殖与分化，上调黏液蛋白 2 的表达，调控免疫及氧化应激，对大肠上皮起到保护作用[46]。SCFAs 水平下降可能与肠道微生态不良相关，提示 SCFAs 可通过其代谢物作用于机体，进而促进结肠息肉乃至结肠癌的发生[47]。

3. 结语

研究肠道微生态与息肉发生发展的相关性，将为进一步揭示其致病机理提供新思路。从目前的研究结果来看，在息肉的发生发展过程中，肠道微生态的失调，无论是在种类，还是数量上的变化，都与息肉的发生发展有着密切的关系。这些微生物的改变，不仅是对肠道微生物的局部调节，还可能通过免疫调节、代谢物的变化，影响肠上皮细胞的增殖和分化，从而促进了息肉的发生。结直肠息肉与肠道菌群的相关性研究显示，结直肠息肉的肠道菌群结构与结直肠息肉发病机制存在较大差异，而肠道菌群在结肠息肉发病机制中发挥着重要作用。通过对肠道菌群的检测，可以作为一种生物标志物，监测宿主的健康状况，预测和评估宿主的疾病风险，指导临床治疗。微生物制剂对预防和治疗结直肠息肉的效果已经获得了一定的证实，但其确切的机理、长期使用益生菌对结直肠癌的预防和治疗效果如何、益生元制剂的安全性如何均需深入研究。

参考文献

[1] Valdes, A.M., Walter, J., Segal, E. and Spector, T.D. (2018) Role of the Gut Microbiota in Nutrition and Health. *British Medical Journal*, **361**, k2179. https://doi.org/10.1136/bmj.k2179

[2] Keku, T.O., Dulal, S., Deveaux, A., Jovov, B. and Han, X. (2015) The Gastrointestinal Microbiota and Colorectal Cancer. *American Journal of Physiology-Gastrointestinal and Liver Physiology*, **308**, G351-G363. https://doi.org/10.1152/ajpgi.00360.2012

[3] Wu, G., Xu, T., Zhao, N., Lam, Y.Y., Ding, X., Wei, D., *et al*. (2024) A Core Microbiome Signature as an Indicator of Health. *Cell*, **187**, 6550-6565.e11. https://doi.org/10.1016/j.cell.2024.09.019

[4] 张静, 王肖枭, 周怡, 等. 肠道菌群与疾病相关性的研究进展[J]. 基础医学与临床, 2020, 40(2): 243-247.

[5] Stephens, R.W., Arhire, L. and Covasa, M. (2018) Gut Microbiota: From Microorganisms to Metabolic Organ Influencing Obesity. *Obesity*, **26**, 801-809. https://doi.org/10.1002/oby.22179

[6] Campbell, C., Kandalgaonkar, M.R., Golonka, R.M., Yeoh, B.S., Vijay-Kumar, M. and Saha, P. (2023) Crosstalk between Gut Microbiota and Host Immunity: Impact on Inflammation and Immunotherapy. *Biomedicines*, **11**, Article 294. https://doi.org/10.3390/biomedicines11020294

[7] 辜柏源, 丁永年. 结直肠息肉相关危险因素研究现状[J]. 齐齐哈尔医学院学报, 2024, 45(14): 1382-1386.

[8] Qin, J., Li, R., Raes, J., Arumugam, M., Burgdorf, K.S., Manichanh, C., *et al*. (2010) A Human Gut Microbial Gene Catalogue Established by Metagenomic Sequencing. *Nature*, **464**, 59-65. https://doi.org/10.1038/nature08821

[9] Chen, Y., Zhou, J. and Wang, L. (2021) Role and Mechanism of Gut Microbiota in Human Disease. *Frontiers in Cellular and Infection Microbiology*, **11**, Article 625913. https://doi.org/10.3389/fcimb.2021.625913

[10] 李惠, 赵菊梅, 师长宏. 基于肠道菌群的免疫调节策略用于结直肠癌联合治疗研究的进展[J]. 中国实验动物学报, 2022, 30(3): 436-443.

[11] Sun, T., Liu, S., Zhou, Y., Yao, Z., Zhang, D., Cao, S., *et al*. (2016) Evolutionary Biologic Changes of Gut Microbiota

in an ‘Adenoma-Carcinoma Sequence’ Mouse Colorectal Cancer Model Induced by 1, 2-Dimethylhydrazine. *Oncotarget*, **8**, 444-457. https://doi.org/10.18632/oncotarget.13443

[12] Hale, V.L., Chen, J., Johnson, S., Harrington, S.C., Yab, T.C., Smyrk, T.C., *et al*. (2017) Shifts in the Fecal Microbiota Associated with Adenomatous Polyps. *Cancer Epidemiology, Biomarkers & Prevention*, **26**, 85-94. https://doi.org/10.1158/1055-9965.epi-16-0337

[13] Zhang, Z., Tang, H., Chen, P., Xie, H. and Tao, Y. (2019) Demystifying the Manipulation of Host Immunity, Metabolism, and Extraintestinal Tumors by the Gut Microbiome. *Signal Transduction and Targeted Therapy*, **4**, Article No. 41. https://doi.org/10.1038/s41392-019-0074-5

[14] 徐艳丽, 尹霞, 常英. 肠道菌群失衡在结直肠癌发病过程中的作用[J]. 国际消化病杂志, 2014, 34(2): 124-127.

[15] 陈辞言, 杜艳. 肠道菌群及其代谢产物与结肠腺瘤性息肉相关性的研究进展[J]. 中国微生态学杂志, 2019, 31(9): 1092-1096.

[16] Huyghe, J.R., Bien, S.A., Harrison, T.A., Kang, H.M., Chen, S., Schmit, S.L., *et al*. (2018) Discovery of Common and Rare Genetic Risk Variants for Colorectal Cancer. *Nature Genetics*, **51**, 76-87. https://doi.org/10.1038/s41588-018-0286-6

[17] Qu, R., Zhang, Y., Ma, Y., Zhou, X., Sun, L., Jiang, C., *et al*. (2023) Role of the Gut Microbiota and Its Metabolites in Tumorigenesis or Development of Colorectal Cancer. *Advanced Science*, **10**, Article 2205563. https://doi.org/10.1002/advs.202205563

[18] DeGruttola, A.K., Low, D., Mizoguchi, A. and Mizoguchi, E. (2016) Current Understanding of Dysbiosis in Disease in Human and Animal Models. *Inflammatory Bowel Diseases*, **22**, 1137-1150. https://doi.org/10.1097/mib.0000000000000750

[19] Cotillard, A., Kennedy, S.P., Kong, L.C., Prifti, E., Pons, N., Le Chatelier, E., *et al*. (2013) Dietary Intervention Impact on Gut Microbial Gene Richness. *Nature*, **500**, 585-588. https://doi.org/10.1038/nature12480

[20] Arumugam, M., Raes, J., Pelletier, E., Le Paslier, D., Yamada, T., Mende, D.R., *et al*. (2011) Enterotypes of the Human Gut Microbiome. *Nature*, **473**, 174-180. https://doi.org/10.1038/nature09944

[21] Goedert, J.J., Gong, Y., Hua, X., Zhong, H., He, Y., Peng, P., *et al*. (2015) Fecal Microbiota Characteristics of Patients with Colorectal Adenoma Detected by Screening: A Population-Based Study. *EBioMedicine*, **2**, 597-603. https://doi.org/10.1016/j.ebiom.2015.04.010

[22] Hussan, H., Clinton, S.K., Roberts, K. and Bailey, M.T. (2017) fusobacterium’s Link to Colorectal Neoplasia Sequenced: A Systematic Review and Future Insights. *World Journal of Gastroenterology*, **23**, 8626-8650. https://doi.org/10.3748/wjg.v23.i48.8626

[23] Guo, S., Li, L., Xu, B., Li, M., Zeng, Q., Xiao, H., *et al*. (2018) A Simple and Novel Fecal Biomarker for Colorectal Cancer: Ratio of Fusobacterium Nucleatum to Probiotics Populations, Based on Their Antagonistic Effect. *Clinical Chemistry*, **64**, 1327-1337. https://doi.org/10.1373/clinchem.2018.289728

[24] Kim, M., Vogtmann, E., Ahlquist, D.A., Devens, M.E., Kisiel, J.B., Taylor, W.R., *et al*. (2020) Fecal Metabolomic Signatures in Colorectal Adenoma Patients Are Associated with Gut Microbiota and Early Events of Colorectal Cancer Pathogenesis. *mBio*, **11**, 16. https://doi.org/10.1128/mbio.03186-19

[25] Saeed, M., Shoaib, A., Kandimalla, R., Javed, S., Almatroudi, A., Gupta, R., *et al*. (2022) Microbe-Based Therapies for Colorectal Cancer: Advantages and Limitations. *Seminars in Cancer Biology*, **86**, 652-665. https://doi.org/10.1016/j.semcancer.2021.05.018

[26] Lou, Y., Song, M., Han, M., Zhong, J., Tian, X., Ren, Y., *et al*. (2022) Tumor Necrosis Factor-A-Induced Protein 8-Like 2 Fosters Tumor-Associated Microbiota to Promote the Development of Colorectal Cancer. *Cancer Immunology Research*, **10**, 354-367. https://doi.org/10.1158/2326-6066.cir-21-0666

[27] Peters, B.A., Dominianni, C., Shapiro, J.A., Church, T.R., Wu, J., Miller, G., *et al*. (2016) The Gut Microbiota in Conventional and Serrated Precursors of Colorectal Cancer. *Microbiome*, **4**, Article No. 69. https://doi.org/10.1186/s40168-016-0218-6

[28] Watson, K.M., Gardner, I.H., Anand, S., Siemens, K.N., Sharpton, T.J., Kasschau, K.D., *et al*. (2021) Colonic Microbial Abundances Predict Adenoma Formers. *Annals of Surgery*, **277**, e817-e824. https://doi.org/10.1097/sla.0000000000005261

[29] 尹莉莉. 菌群失调参与远端结直肠息肉复发的机制研究[D]: [硕士学位论文]. 南昌: 南昌大学, 2024.

[30] Li, J., Zhu, Y., Yang, L. and Wang, Z. (2022) Effect of Gut Microbiota in the Colorectal Cancer and Potential Target Therapy. *Discover Oncology*, **13**, Article No. 51. https://doi.org/10.1007/s12672-022-00517-x

[31] Barna, I., Nyúl, D., Szentes, T. and Schwab, R. (2018) A bélmikrobiom, a metabolikus betegségek és a hypertonia kapcsolatának irodalmi áttekintése. *Orvosi Hetilap*, **159**, 346-351. https://doi.org/10.1556/650.2018.30787

[32] Pop, O.L., Vodnar, D.C., Diaconeasa, Z., Istrati, M., Bințințan, A., Bințințan, V.V., *et al*. (2020) An Overview of Gut

Microbiota and Colon Diseases with a Focus on Adenomatous Colon Polyps. *International Journal of Molecular Sciences*, **21**, Article 7359. https://doi.org/10.3390/ijms21197359

[33] Jass, J.R. (2006) Classification of Colorectal Cancer Based on Correlation of Clinical, Morphological and Molecular Features. *Histopathology*, **50**, 113-130. https://doi.org/10.1111/j.1365-2559.2006.02549.x

[34] Hu, H., Gong, X., Xu, K., Luo, S., Gao, W., Li, B., *et al*. (2023) Risk Factor Analysis of Malignant Adenomas Detected during Colonoscopy. *Frontiers in Medicine*, **10**, Article 1106272. https://doi.org/10.3389/fmed.2023.1106272

[35] Burrows, M.P., Volchkov, P., Kobayashi, K.S. and Chervonsky, A.V. (2015) Microbiota Regulates Type 1 Diabetes through Toll-Like Receptors. *Proceedings of the National Academy of Sciences*, **112**, 9973-9977. https://doi.org/10.1073/pnas.1508740112

[36] 杨丽萍, 马臻棋, 王学红, 等. 肠道菌群与结肠息肉的关系研究进展[J]. 中国医刊, 2022, 57(2): 139-141.

[37] Gao, M., Zhong, A., Patel, N., Alur, C. and Vyas, D. (2017) High Throughput RNA Sequencing Utility for Diagnosis and Prognosis in Colon Diseases. *World Journal of Gastroenterology*, **23**, 2819-2825. https://doi.org/10.3748/wjg.v23.i16.2819

[38] Rezasoltani, S., Ghanbari, R., Looha, M.A., Mojarad, E.N., Yadegar, A., Stewart, D., *et al*. (2020) Expression of Main Toll-Like Receptors in Patients with Different Types of Colorectal Polyps and Their Relationship with Gut Microbiota. *International Journal of Molecular Sciences*, **21**, Article 8968. https://doi.org/10.3390/ijms21238968

[39] Dai, Z., Zhang, J., Wu, Q., Chen, J., Liu, J., Wang, L., *et al*. (2019) The Role of Microbiota in the Development of Colorectal Cancer. *International Journal of Cancer*, **145**, 2032-2041. https://doi.org/10.1002/ijc.32017

[40] Brennan, C.A. and Garrett, W.S. (2016) Gut Microbiota, Inflammation, and Colorectal Cancer. *Annual Review of Microbiology*, **70**, 395-411. https://doi.org/10.1146/annurev-micro-102215-095513

[41] Morrison, D.J. and Preston, T. (2016) Formation of Short Chain Fatty Acids by the Gut Microbiota and Their Impact on Human Metabolism. *Gut Microbes*, **7**, 189-200. https://doi.org/10.1080/19490976.2015.1134082

[42] Tailford, L.E., Crost, E.H., Kavanaugh, D. and Juge, N. (2015) Mucin Glycan Foraging in the Human Gut Microbiome. *Frontiers in Genetics*, **6**, Article 81. https://doi.org/10.3389/fgene.2015.00081

[43] Oh, T.J., Sul, W.J., Oh, H.N., Lee, Y., Lim, H.L., Choi, S.H., *et al*. (2019) Butyrate Attenuated Fat Gain through Gut Microbiota Modulation in Db/Db Mice Following Dapagliflozin Treatment. *Scientific Reports*, **9**, Article No. 20300. https://doi.org/10.1038/s41598-019-56684-5

[44] Abu-Ghazaleh, N., Chua, W.J. and Gopalan, V. (2020) Intestinal Microbiota and Its Association with Colon Cancer and Red/Processed Meat Consumption. *Journal of Gastroenterology and Hepatology*, **36**, 75-88. https://doi.org/10.1111/jgh.15042

[45] Elinav, E., Strowig, T., Kau, A.L., Henao-Mejia, J., Thaiss, C.A., Booth, C.J., *et al*. (2011) NLRP6 Inflammasome Regulates Colonic Microbial Ecology and Risk for Colitis. *Cell*, **145**, 745-757. https://doi.org/10.1016/j.cell.2011.04.022

[46] Bonder, M.J., Kurilshikov, A., Tigchelaar, E.F., Mujagic, Z., Imhann, F., Vila, A.V., *et al*. (2016) The Effect of Host Genetics on the Gut Microbiome. *Nature Genetics*, **48**, 1407-1412. https://doi.org/10.1038/ng.3663

[47] Cremon, C., Barbaro, M.R., Ventura, M. and Barbara, G. (2018) Pre and Probiotic Overview. *Current Opinion in Pharmacology*, **43**, 87-92. https://doi.org/10.1016/j.coph.2018.08.010