cognitive capability and affects the microbiotaof large intestine of tree shrew model for Alzheimer's disease[J]. Mol Med Rep, 2021, 23(4): 291.
[25] Yang Y, Li S, Huang H, et al. Comparison of the protective effects of ginsenosides Rb1 and Rg1 on improving cognitive deficits in SAMP8 mice based on anti-neuroinflammation mechanism [J]. Front Pharmacol, 2020, 11: 834.
[26] Sun Y, Yang Y, Liu S, et al. New therapeutic approaches to and mechanisms of ginsenoside Rg1 against neurological diseases[J]. Cells, 2022, 11(16): 2529.
[27] Xu T Z, Shen X Y, Sun L L, et al. Ginsenoside Rg1 protects against H_2O_2-induced neuronal damage due to inhibition of the NLRP1 inflammasome signalling pathway in hippocampal neurons in vitro[J]. Int J Mol Med, 2019, 43(2): 717-726.
[28] Luo C L, Wu G Y, Li Z Z, et al. Research progress on iron homeostasis in ferroptosis in Alzheimer's disease[J]. Int J Geriatr, 2025, 46(4): 477-482.
[29] Kong L, Liu Y, Li J, et al. Ginsenoside Rg1 alleviates chronic inflammation-induced neuronal ferroptosis and cognitive impairments via regulationof AIM2 - Nrf2 signaling pathway [J]. J Ethnopharmacol, 2024, 330: 118205.
[30] Guo J, Chen L, Ma M. Ginsenoside Rg1 suppresses ferroptosis of renal tubular epithelial cells in sepsis-induced acute kidney injury via the FSP1-CoQ10-NAD(P)H pathway[J]. Curr Med Chem, 2024, 31(15): 2119-2132.
[31] Li J Y, Li N J. Research progress on pharmacological effects and mechanisms of ginsenosides in the treatment of Alzheimer's disease[J]. Pract Geriatr, 2017, 31(7): 606-608,612.
[32] Su P, Wang L, Du S J, et al. Advance in studies of Panax notoginseng saponinson pharmacological mechanism of nervous system disease[J]. Zhongguo Zhong Yao Za Zhi, 2014, 39(23): 4516-4521.
[33] Shi C, Na N, Zhu X, et al. Estrogenic effect of ginsenoside Rg1 on APP processing in post-menopausal platelets [J]. Platelets, 2013, 24(1): 51-62.
[34] Zhang X, Wang J, Xing Y, et al. Effects of ginsenoside Rg1 or 17β-estradiol on a cognitively impaired, ovariectomized rat model of Alzheimer's disease [J]. Neuroscience, 2012, 220: 191-200.
[35] Wang S S, Liu P. Research progress on the correlation between gut microbiota and pathogenesis of Alzheimer's disease[J]. Beijing Med J, 2022, 44(7): 633-635,639.
[36] Zha X, Liu X, Wei M, et al. Microbiota-derived lysophosphatidylcholine alleviates Alzheimer's disease pathology via suppressing ferroptosis[J]. Cell Metab, 2025, 37(1): 169-186. e9.
[37] Wang L, Lu J, Zeng Y, et al. Improving Alzheimer's disease byaltering gut microbiota in tree shrews with ginsenoside Rg1[J]. FEMS Microbiol Lett, 2020, 367(4): fnaa011.

片语健康

碳水化合物毒性

“碳水化合物毒性（Carbotoxicity）”的说法出现在 2018 年，可用来描述碳水化合物在诱发代谢综合征中的作用[1]。碳水化合物由碳、氢、氧组成，是人的主要能量来源，包括糖、淀粉和纤维等。糖又分为单糖（如葡萄糖、果糖）、双糖（如蔗糖）和多糖（如淀粉和糖原），淀粉和纤维是含糖碳水化合物。

在以谷物为主食的农业时代（约 1 万年前）之前，人作为狩猎采摘者（Forager）已进化了 200 多万年[2-3]。这些狩猎采摘者以动物性食物（肉类、鱼和昆虫等）为主食，辅以植物性食物（果实、种子、块茎、坚果、根茎和球茎等）。因为来源有限，碳水化合物是支持他们生存的“挚友”，没有什么毒性[1]。

在 19—20 世纪，从甜菜中提取的糖在欧洲出现。由于产量有限，糖首先成为了王公贵族的奢侈品。当他们消耗多了的时候，碳水化合物毒性开始显现，导致肥胖和糖尿病患者增多。随着技术的成熟，糖从奢侈品变为日常食品。再晚些，果酱、甜点、含糖饮料等大规模进入家庭餐桌，碳水化合物毒性开始大规模凸显[1]。

第二次世界大战后，食品工业驱动的超加工食品及碳酸饮料消费量在全球范围内迅速攀升。自 1975 年起，高果糖玉米糖浆作为新型添加糖在食品工业中广泛使用，进一步强化了现代饮食中的碳水化合物毒性。这空前提高了肥胖、2 型糖尿病、非酒精性脂肪肝、高血压和心血管疾病的发生率[1]。

参考文献

[1] Kroemer G, López-Otín C, Madeo F, et al. Carbotoxicity-noxious effects of carbohydrates[J]. Cell, 2018, 175(3): 605-614.
[2] Arbuckle B S. Genomic insights into social life in NeolithicAnatolia[J]. Science, 2025, 388(6754): 1372-1373.
[3] Gibbons A. The riddle ofcoexistence[J]. Science, 2025, 389(6758): 338-343.

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