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TyG及其衍生指数在保留比值受损肺功能诊疗方面的研究进展

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摘　要

保留比值受损肺功能(PRISm)是一种具有独特肺功能表现的功能障碍表型，与心血管代谢紊乱、全身性炎症反应及不良预后风险密切相关。当前PRISm的早期识别与综合管理面临挑战，亟需能够反映其潜在病理生理机制的生物标志物以指导临床实践。反映胰岛素抵抗与代谢紊乱状态的甘油三酯－葡萄糖指数(TyG)及其衍生指标(如TyG-BMI、TyG-WC等)，因其与全身低度炎症、氧化应激及多器官功能损害相关，在慢性呼吸系统疾病中的作用日益受到关注。作为综合代谢炎症标志物，这些指标可能为PRISm的病理分型、风险评估及预后预测提供新视角。本文系统阐述PRISm的临床特征与病理基础，综述现有生物标志物研究进展，重点探讨TyG及其衍生指标在慢性呼吸系统疾病特别是PRISm人群中的研究证据，以期为PRISm的早期干预与个体化防治提供理论依据与策略参考。

关键词

甘油三酯－葡萄糖指数，代谢，炎症，保留比值受损肺功能

Research Progress on TyG Index and Its Derived Indices in the Diagnosis and Treatment of Preserved Ratio Impaired Spirometry

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Abstract

Preserved ratio impaired spirometry (PRISm) is a distinct functional impairment phenotype identified through clinical pulmonary function assessment, characterized by its close associations with cardiovascular metabolic disorders, systemic inflammatory responses, and increased risk of adverse outcomes. Currently, the early identification and comprehensive management of PRISm face significant challenges, highlighting an urgent need for biomarkers that reflect its underlying pathophysiological mechanisms to guide clinical practice. In recent years, the triglyceride-glucose (TyG) index and its derived metrics (such as TyG-BMI and TyG-WC), which reflect insulin resistance and metabolic dysregulation, have garnered increasing attention in the context of chronic respiratory diseases due to their links with systemic low-grade inflammation, oxidative stress, and multi-organ functional impairment. As integrated metabolic-inflammatory markers, these indices offer potential new perspectives for the pathological subtyping, risk assessment, and prognosis prediction of PRISm. This article systematically outlines the clinical characteristics and pathological basis of PRISm, reviews advances in existing biomarker research, and focuses on the evidence regarding TyG and its derived indicators in chronic respiratory diseases, particularly in PRISm populations. The aim is to provide theoretical foundations and strategic insights for the early intervention and personalized management of PRISm.

Keywords

Triglyceride-Glucose Index, Metabolism, Inflammation, Preserved Ratio Impaired Spirometry

1. 引言

保留比值受损肺功能(Preserved ratio impaired spirometry, PRISm)是一种过渡状态，可能向正常肺功能或慢性阻塞性肺病(Chronic Obstructive Pulmonary Disease, COPD)进行双向转变[1] [2]。2014 年 Wan 等人明确定义 PRISm 为：支气管舒张后第一秒用力呼气容积(Forced Expiratory Volume in the first second, FEV1)/用力肺活量(Forced Vital Capacity, FVC)比值正常(≥0.7)，FEV1 低于预计值 80% [3]。不同研究中报道的 PRISm 患病率存在显著差异[4]，可能与性别、种族、地理位置及风险因素分布不同有关[5]。PRISm 不仅与呼吸道症状相关[6]，还伴随医疗资源使用增加，与肥胖、糖尿病、心脏病等多种合并症共存，与全因死亡率、心血管和呼吸相关疾病的发生率和死亡率升高存在关联[1] [7]-[12]。一项队列研究表明，约 12%的 PRISm 患者会进展为符合 COPD 诊断的气流受限，但多数患者的 PRISm 表型表现出可逆性与潜在可治疗性[6]。通过公共卫生措施(如降低吸烟率、控制体重指数等)或有助于减少 PRISm 的患病率[6]。鉴于上述特点，针对 PRISm 高危人群开展系统筛查与早期预防干预具有重要意义。研究表明，代谢紊乱可能通过影响肺微血管系统与间质对肺部健康产生负面影响，因为肺组织正常功能的维持依赖于稳定的代谢内环境[13]。糖尿病、血脂异常和代谢综合征以胰岛素抵抗(Insulin Resistance, IR)为病理基础，有研究证实会对肺部产生损害[14]-[16]。甘油三酯－葡萄糖指数(Triglyceride-Glucose Index, TyG)是用于综合评估代谢功能的一项指标。相较于单独的血糖或甘油三酯指标，在识别代谢功能障碍方面更具优势，在代谢综合征的诊断方面的准确性甚至高于胰岛素抵抗稳态模型评估(Homeostatic Model Assessment of

Insulin Resistance, HOMA-IR) [17]。目前，TyG 指数已被证明与呼吸道症状、慢性支气管炎和限制性肺功能模式存在显著关联[18]，此外通过整合肥胖参数(如腰围、体重指数)和炎症标志物(如 C 反应蛋白)而衍生的指标，如甘油三酯－葡萄糖－腰围指数(Triglyceride-Glucose-Waist Circumference Index, TyG-WC)、甘油三酯－葡萄糖－体重指数(Triglyceride-Glucose-Body Mass Index, TyG-BMI)、甘油三酯－葡萄糖－腰高比指数(Triglyceride-Glucose-Waist-to-Height Ratio Index, TyG-WHtR)、C 反应蛋白－甘油三酯－葡萄糖指数(C-reactive protein-Triglyceride-Glucose Index, CTI)等，则进一步提升其对肺部疾病风险的预测能力[18] [19]。本文聚焦于 TyG 指数及其衍生指标对 PRISm 的预测价值进行综述，旨在为临床实现 PRISm 的早期识别与干预提供参考，以延缓或阻断其向 COPD 进展，从而改善患者长期预后。

2. PRISm 的临床概况

2.1. PRISm 的流行病学

PRISm 在全球范围内的患病率存在显著差异，文献报道在 4.7%至 25.2%之间[20]，这种差异与地域分布密切相关：例如，拉丁美洲、美国、英国报告的患病率分别约为 5% [21]、17.3% [22]和 11% [6]，而亚洲的韩国和日本则分别为 8.9% [23]和 10% [1]，我国学者基于中国慢性病前瞻性研究(China Kadoorie Biobank, CKB)的数据发现，≥40 岁人群中 PRISm 患病率高达 24.8% [24]。患病率的差异可能受多重因素影响，包括自然环境、社会经济条件、种族特征(如日本人群肥胖率较低而吸烟率较高[1] [25]-[27])、城乡暴露源差异(如农村生物质燃烧[28]、城市空气污染[29])以及研究设计本身的异质性(如一项美国研究基于心肌梗死患者[22])。因此，开展结合本土环境与人群特征的深入研究，对于制定有针对性的防治策略至关重要。在人口学分布方面，多项研究表明 PRISm 患者中女性比例较高[30] [31]，这一发现更新了以往认为肺功能下降主要见于男性吸烟者的传统观念。在年龄分布上，尽管≥40 岁人群的患病风险显著升高，但年轻群体，尤其合并哮喘或肥胖的患者，其患病风险同样不容忽视。澳大利亚 BOLD 研究进一步描绘了 PRISm 人群的特征轮廓：与肺功能正常或存在典型气流受限的患者相比，PRISm 受试者往往年龄相对较轻、女性占比更高、非吸烟者居多，并且更可能来自社会经济地位较低的区域或本身即为肥胖个体[32]。

2.2. PRISm 的病理生理与潜在机制

PRISm 的病理生理机制核心在于代谢紊乱及其引发的系统性慢性低度炎症共同作用于呼吸系统。代谢紊乱特别是 IR 与内脏脂肪堆积，是关键的始动因素。

首先，代谢紊乱通过影响呼吸肌结构与功能，直接参与限制性通气功能障碍的形成。IR 可导致骨骼肌(包括呼吸肌)葡萄糖摄取障碍和能量代谢异常，可能直接削弱呼吸肌的收缩功能与耐力[33] [34]。同时堆积的内脏脂肪组织不仅作为机械性负担限制膈肌活动与胸廓扩张，加剧限制性通气障碍，更作为一个活跃的内分泌器官，通过分泌促炎因子(如 TNF-α、IL-6)和失衡的脂肪因子(瘦素水平升高、脂联素水平降低)，诱导并维持全身性的慢性低度炎症状态[35]-[37]。

其次，在代谢－炎症轴的驱动下，高血糖与高甘油三酯血症可通过激活氧化应激与内质网应激通路直接损伤肺泡上皮并促进肺间质纤维化。高糖环境通过线粒体电子传递链过度活跃及葡萄糖自身氧化增加(Reactive oxygen species, ROS)生成[38]，而高甘油三酯的代谢产物游离脂肪酸(如棕榈酸)则通过激活 NADPH 氧化酶协同放大氧化应激信号[39]。过量 ROS 一方面直接攻击肺泡上皮细胞膜，引发脂质过氧化与细胞凋亡，另一方面激活 NF-κB，促使肺泡上皮释放 TNF-α、IL-6 等促炎因子，加剧局部炎症损伤[40]。同时高糖高脂环境干扰内质网腔内蛋白质正常折叠，导致未折叠蛋白蓄积，激活 PERK/eIF2α、ATF6 及 IRE1α/XBP1 三条未折叠蛋白反应通路[41]。当内质网应激超出细胞自救能力时，信号通路功能发生转换：一方面 PERK 通路持续激活转录因子 CHOP，诱导肺泡 II 型上皮细胞凋亡；另一方面未折叠蛋白反

应信号还可通过诱导上皮–间质转化直接促进纤维化重塑[42]。氧化应激与内质网应激共同激活 TGF-β1/Smad 信号通路进一步促进肺泡上皮向间质转化，刺激成纤维细胞增殖并转化为肌成纤维细胞，导致胶原蛋白(Col I, Col III)与纤维连接蛋白在肺泡间隔异常沉积，破坏肺实质弹性[43] [44]。

最后，循环中的炎症介质与局部免疫细胞活化共同作用，加剧肺组织损伤和气体交换功能障碍。全身性低度炎症可激活肺泡巨噬细胞，诱导局部 ROS 生成，引起肺泡上皮细胞与肺毛细血管内皮的氧化应激损伤、细胞凋亡及细胞外基质重塑，最终影响肺实质弹性与弥散功能。炎症信号还趋化中性粒细胞、淋巴细胞等炎症细胞浸润肺间质和气道，加剧局部炎症微环境。此外，IR 和慢性炎症共同诱发的全身性血管内皮功能障碍同样累及肺循环，引起肺微血管病变，损害肺组织的灌注与气体交换效率[45]。

上述病理过程协同作用，在疾病早期主要表现为肺弹性回缩力下降与肺容积扩张受限，导致肺总量(Total Lung Capacity, TLC)及用力 FVC 降低[46]；而由于此时大气道尚未出现明显阻塞性病变，FEV_1/FVC 比值得以维持正常。因此，PRISm 的特征性肺功能改变可视为全身性代谢–炎症异常在呼吸系统的早期、特异性表现。

2.3. PRISm 的危险因素

目前研究已识别多种与 PRISm 相关的危险因素，主要涵盖以下几个方面：(如女性[47]、高龄[48]、低社会经济地位及教育水平)、体重指数异常[49]、环境与职业暴露(如吸烟[23]、职业性粉尘[50]、生物燃料及重金属暴露[51] [52])、共患疾病(如哮喘[7]、糖尿病、高血压)以及肺功能特征(如小气道功能障碍、总肺活量降低[53])等。其中，一项大规模队列研究分析显示，女性、当前吸烟状况、哮喘史及超重/肥胖(尤其是中心性肥胖)是 PRISm 明确且独立的风险因素[6]。

2.4. PRISm 的临床表现与合并症

PRISm 人群的临床表现与 COPD 相似，常见症状包括慢性咳嗽、咳痰、喘息及呼吸困难等[6] [54]。进一步研究表明 PRISm 患者存在显著临床异质性。依据 He D 等人提出的分型标准，PRISm 可分为轻度和重度两种亚型，其中重度亚型患者普遍具有年龄较大、受教育程度较低、已婚或有伴侣比例较低、当前吸烟率较高以及体力活动水平明显下降等特征[55]。

此外，PRISm 与多种全身性疾病密切相关，包括高龄、肥胖、高血压、糖尿病、心力衰竭、冠心病、卒中及严重肾功能下降等[56]，韩国学者开展研究进一步揭示，相较于其他人群，PRISm 患者中上述合并症的患病比例呈现出显著升高态势[23]。这些共病的进展与恶化常导致患者死亡风险上升，例如心力衰竭会增加呼吸系统相关死亡率，而冠心病则直接推高心血管死亡风险[57] [58]。值得注意的是，研究显示因心脏并发症住院的 PRISm 患者数量呈上升趋势。英国的一项队列研究数据显示，PRISm 组的糖尿病患病率(8.6%)显著高于对照组(3.7%)及气流受限组(4.7%)，且其心绞痛、心肌梗死及卒中的患病率均约为对照组的两倍[6]。综上所述，对 PRISm 患者实施临床管理需要更为全面和系统的策略。

3. PRISm 生物标志物的研究进展

3.1. 系统性炎症标志物

如 C 反应蛋白(C-reactive Protein, CRP)、纤维蛋白原和白细胞介素-6 (Interleukin-6, IL-6)、白细胞介素-2 (Interleukin-2, IL-2)、白细胞介素-5 (Interleukin-5, IL-5)和白细胞介素-17A (Interleukin-17A, IL-17A)、外周血嗜酸性粒细胞计数(Peripheral Blood Eosinophil Count, EOS 计数)等，在 PRISm 个体中常被发现水平升高[59]-[61]，这些标志物不仅提示低度全身性炎症状态，且与肺功能加速下降及不良预后相关，为理解 PRISM 与心血管等全身性共病之间的桥梁提供分子依据，并指向潜在的抗炎干预靶点。

3.2. 肺组织本身的损伤与修复标志物

例如晚期糖基化终末产物可溶性受体(Soluble Receptor for Advanced Glycation End products, sRAGE)以及基质金属蛋白酶(Matrix Metalloproteinases, MMPs)家族成员[62]-[65]，其水平变化暗示了肺泡上皮细胞的损伤和细胞外基质的重塑，这有助于识别早期、亚临床的肺实质病变，进而实现对 PRISm 中可能存在的不同病理生理亚型进行有效区分。

3.3. 代谢与心血管相关标志物

如胰岛素[66]、瘦素、脂联素、尿酸和 N-末端脑钠肽前体(N-terminal pro-B-type Natriuretic Peptide, NT-proBNP)的异常[61] [67]，强调 PRISm 与肥胖、代谢综合征及潜在心功能不全存在紧密关联，有助于临床进行多系统综合评估与管理。

3.4. 多组学与新兴生物标志物

近年来高通量技术的演进为新型生物标志物的探寻开辟全新路径。借助蛋白质组学筛查手段，研究者已成功识别出与 PRISm 相关的生物标志物，例如 α1-抗胰蛋白酶(Alpha-1-Antitrypsin, AAT) [68]。基因组学研究亦揭示部分与 COPD 和肺纤维化存在重叠的遗传位点，诸如基质金属蛋白酶 12 (Matrix Metallo-proteinase-12, MMP12) [69]、转化生长因子-β (Transforming Growth Factor-beta, TGF-β)通路基因 [70]。上述发现为深入理解 PRISm 的遗传根基与分子调控网络提供重要线索。

3.5. 其他

呼气挥发性有机化合物(Exhaled Volatile Organic Compounds, VOCs)作为呼吸系统疾病诊断领域的一种新兴无创检测技术，正逐步受到学界广泛关注。研究显示特定 VOC (如丙酮)水平降低可能与 PRISm 相关，提示呼气流量受限[71]。此外，激素类(如脱氢表雄酮(Dehydroepiandrosterone, DHEA)、硫酸脱氢表雄酮(Dehydroepiandrosterone Sulfate, DHEA-S)) [72] [73]、维生素(如维生素 D) [74]、以及环境暴露相关的重金属(如镉[52])等标志物也可能分别指向 PRISm 不同亚型的潜在驱动机制。

4. TyG 及其衍生指标

4.1. TyG 的定义与计算方法

TyG 可通过如下公式来表达：TyG = Ln[空腹甘油三酯(mg/dL) × 空腹血糖(mg/dL)/2]。该指数通过整合脂质代谢与葡萄糖代谢两大核心参数，反映机体胰岛素敏感性状态[75]。相较于 IR 的传统评估方法，如高胰岛素 - 正常血糖钳夹试验及 HOMA-IR，TyG 具有计算简便、成本低、时效性强且灵敏度与特异性较高等优势，可作为 IR 的有效替代生物标志物[76]。目前 TyG 在呼吸系统疾病研究领域也日益受到重视，正逐步应用于 COPD 等慢性气道疾病的风险分层与预后评估，为相关临床研究及人群筛查提供新的实用工具。

4.2. TyG 衍生指标的生物学基础

TyG-WC 同时反映 IR 和中心性肥胖，中心性肥胖通过脂肪组织内分泌功能失调和游离脂肪酸释放加剧 IR，而 IR 又进一步促进脂肪异常分布，形成恶性循环，共同增加代谢及心血管风险，研究证实 TyG-WC 在预测 2 型糖尿病和非酒精性脂肪肝方面优于单一指标[77] [78]。肥胖伴随的慢性低度炎症和脂毒性可损害胰岛素信号通路，TyG-BMI 将 IR 与全身肥胖程度整合，能更全面评估肥胖相关的整体代谢紊乱负荷，与高血压及动脉粥样硬化性心血管疾病的发生风险呈现出显著相关性[79] [80]。TyG-WHtR 进一步

优化中心性肥胖的评估，通过腰围与身高的比值校正体格差异，更敏感地反映内脏脂肪堆积，与代谢综合征及其相关心肾并发症的风险密切相关[81]-[83]。动脉粥样硬化发生发展的核心机制是 IR 与慢性低度炎症相互促进，共同构成“代谢性炎症”状态，CTI 将 IR 与 CRP 这一经典急性期炎症标志物相结合，是心血管事件和卒中风险的强力预测指标[84]。

5. 在慢性呼吸系统疾病及 PRISm 患者中的研究

美国国家健康与营养检查调查(National Health and Nutrition Examination Survey, NHANES)的一项研究结果表明 TyG 与多种肺功能指标与 PRISm 呈现出显著相关性。进一步分析发现，TyG 在膳食质量指标与肺功能指标(包括 FEV1、FVC 及 PRISm 状态)之间的关联中发挥部分中介作用[85]。另一项研究发现 TyG 水平升高可能拮抗维生素 D 对 PRISm 的部分保护效应，这揭示了维生素 D、氧化应激与代谢功能障碍间的复杂交互作用，提示针对该通路的干预或可作为潜在治疗策略[74]。在慢性呼吸系统疾病中，慢性低度炎症与代谢紊乱相互促进，共同推动气道病变与病情进展。TyG 及其衍生指标(TyG-WC, TyG-BMI, TyG-WHtR, CTI)作为同时评估 IR 与全身炎症的新型综合标志物，为该类疾病的临床评估提供重要工具。近期证据表明，TyG 升高与自我报告的慢性支气管炎诊断几率增加 21%相关[18]。TyG-BMI、TyG-WC 和 TyG-WHtR 能更综合地反映代谢异常与中心性肥胖对气道高反应性及气流受限的影响，其中 TyG-BMI 及 TyG-WC 已被发现与中国成年人群肺腺癌风险呈正相关，有助于识别高危个体[86]。此外，在空腹血糖受损人群中，TyG-WHtR 与结核感染风险亦呈显著正相关[87]。同时，整合炎症指标的 CTI 可同步捕捉代谢异常与全身炎症负荷，基于 CTI 构建的列线图模型已证实 CTI 是慢性阻塞性肺疾病急性加重的独立风险因素，具有良好的预测效能[88]。

尽管目前尚未有研究直接阐明 TyG 衍生指标与 PRISm 之间的关联，但上述证据提示 TyG 及其衍生指标与整体肺功能及多种慢性呼吸系统疾病存在显著相关。这些发现为进一步探索其在 PRISm 发生发展中的潜在作用提供了重要线索，也凸显了未来开展系统化、针对性研究的科学价值与临床意义。

6. 展望

现有研究表明，TyG 及其衍生指标与 PRISm 显著关联，为早期识别代谢相关性肺功能障碍提供了新思路。然而若要将其真正用于临床预测，仍需开展大规模前瞻性队列研究验证其效能，并通过多中心临床试验探索其干预价值。后续可通过前瞻性队列设计，对 PRISm 人群进行 TyG 指数分组，经过 5 至 10 年的长期随访，比较各组进展为 COPD 或实现肺功能缓解的差异，以明确 TyG 指数对疾病转归的预测能力；同时可在合并高 TyG 的 PRISm 患者中开展随机对照试验，考察生活方式优化或药物干预对 TyG 指数及肺功能的影响，从而探讨其作为潜在干预靶点的可行性与临床价值。上述研究的开展，将为构建基于 TyG 指数的标准化评估体系及其在肺功能管理中的临床应用路径提供重要循证依据。

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