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溃疡性结肠炎及大肠湿热病证结合动物模型的研究现状及思考

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【摘要】 溃疡性结肠炎(ulcerative colitis,UC)是一种慢性炎症性肠病,病因复杂,治疗面临诸多挑战。中医药在 UC 治疗中展现出良好疗效,病证结合动物模型已成为中西医融合研究的重要工具。系统归纳了 UC 动物模型的主流构建方法,并重点梳理了与“大肠湿热证”相结合的造模策略,包括饮食、环境及微生态等诱因的复合建模路径。在模型评价方面,强调将现代组学技术(如代谢组、宏基因组等)引入中医证候动物模型,用以提升证候识别的客观性与量化水平。同时,总结了基因修饰动物模型与大肠湿热证诱导手段整合的研究进展,提出复合模型构建思路,以更贴近 UC 复杂病因与个体体质的交互特征。该类模型在完善构建技术与评价体系的基础上,有望增强中医病证模型的科学性与临床相关性,为中医药干预 UC 的基础研究与机制探索提供支撑。

【关键词】 溃疡性结肠炎;病证结合;动物模型;组学评价
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Syndrome current research landscape and critical reflections on disease-syndrome integrated animal models of ulcerative colitis with large-intestine damp-heat syndrome

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【Abstract】 Ulcerative colitis (UC) is a chronic inflammatory bowel disease with a multifactorial etiology that poses considerable challenges to effective treatment. Traditional Chinese medicine (TCM) has demonstrated favorable therapeutic outcomes in the management of UC, and the development of disease-syndrome integrated animal models has emerged as a critical approach for bridging TCM and modern biomedical research. We systematically reviewed the main strategies for constructing UC animal models, with a specific focus on modeling protocols tailored to the TCM syndrome of large intestine damp-heat. These approaches encompass multifactorial induction pathways involving dietary, environmental, and microbial stimuli. In terms of model evaluation, emphasis was placed on integrating modern omics technologies, such as metabolomics and metagenomics, to enhance the objectivity and quantifiability of TCM syndrome identification in experimental models. We also summarize recent advances in the integration of gene-edited models with damp-heat induction method, providing a conceptual framework for composite models that better simulate the complex interplay between UC pathogenesis and individual constitutional characteristics. These integrative models, supported by refined construction techniques and multidimensional evaluation systems, have the potential to improve scientific rigor and translational relevance, offering a robust experimental foundation for elucidating the mechanisms of TCM-based interventions in UC.

【Keywords】 ulcerative colitis; disease-syndrome combination; animal model; omics-based evaluation

Conflicts of Interest: The authors declare no conflict of interest.

溃疡性结肠炎(ulcerative colitis, UC)是一种由环境、遗传、微生物及免疫等多因素共同作用引发的慢性非特异性炎症性疾病,临床上以黏液脓血便、腹痛、腹泻等症状为主要表现,发病机制复杂[1]。目前西医治疗 UC 主要依赖于氨基水杨酸类、糖皮质激素、免疫抑制剂及生物制剂等,但长期应用可能带来副作用及并发症,且易反复发作,影响患者生活质量[2]。相较而言,中医药在调控免疫、减轻炎症、缓解症状及预防复发方面具有明显优势;例如,传统中药能够降低糖皮质激素的副作用、增强西药疗效及改善患者预后质量[3-4]。此外,中医药还能有效减少病情复发,节省医疗成本,因此其作用机制与疗效受到广泛关注[5]。

中医认为,UC 的发生与“湿热”“脾虚”“肝郁”等内生病机密切相关,其中“大肠湿热”证型最具代表性[6];大肠是 UC 的重要中医证型,占中医辨证分类的 25.93%[7],贯穿疾病发展的整个过程[8],《沈氏尊生书》中亦有记载:“大抵痢之疡根,皆由湿蒸热壅”[9]。湿热证对应的中医治疗能降低促炎反应、提高抗炎反应[10],针对大肠湿热证的病证结合动物模型研究有助探讨 UC 的生物学机制。近年来,研究者尝试通过饮食干预、高温高湿环境、灌服白酒等手段诱导“大肠湿热证”表型,并与化学、微生物或基因修饰等 UC 模型结合,从而实现“病”与“证”的协同模拟。然而,现有模型多停留在单因素诱导或主观评价阶段,缺乏系统、量化的评价标准。

因此,针对现有模型在诱导方式、评价标准以及中医证候量化方面的不足,本文将系统梳理溃疡性结肠炎动物模型的构建策略,并重点关注与“大肠湿热证”相结合的病证结合模型的发展

现状,尝试探讨中西医融合视角下模型优化的方向及可能路径。

1 UC 模型研究概述

目前用于研究 UC 的动物模型主要分为化学诱导模型、微生物诱导法、基因修饰模型和其他特殊模型。

1.1 化学诱导模型

常用的有葡聚糖硫酸钠(dextran sodium sulfate, DSS)模型[11]:DSS 通过饮水方式诱导肠黏膜损伤,通常为 3% DSS 溶液自由饮用 7 d,是 UC 研究中最常用的模型之一。其优点是操作简便,可诱发典型的炎症反应和黏膜损伤;但由于炎症机制单一,无法全面反映 UC 的复杂病理特征。2,4,6-三硝基苯磺酸(2,4,6-trinitrobenzene sulfonic acid, TNBS)和乙醇模型[12]:大鼠模型中使用 0.6 mL 的混合溶液,终浓度为 33.3% 的 TNBS 与无水乙醇,进行灌肠;也有方法用 10 mg 的 TNBS 溶解于 0.25 mL 的 50% 乙醇溶液中,在距肛门约 8 cm 处进行滴注灌肠[13]。对于小鼠模型,则采用 200 mg/kg 的 TNBS 溶解于 30% 乙醇,在距肛门 3 ~ 4 cm 处进行灌肠以建立模型[14]。此模型能够模拟一定的免疫炎症特征。但其病理过程与人类 UC 存在一定差异。

1.2 微生物诱导法

肠道菌群是指肠道内的微生物,对维持肠道功能及宿主免疫稳态至关重要[15],新模型注重通过菌群失调来诱发结肠炎。例如,将无菌小鼠定植人源菌群或特定致病菌可复制出类似 UC 的病变,通过灌胃致病菌以打破菌群平衡,并结合 DSS 诱导肠炎,常用方法:单次灌注-适用于短期急性结肠炎模型,灌注后观察 24 ~ 72 h 的病理变化。每日灌注适用于慢性结肠炎或反复感染模型,灌注持续时间可能为 7、14 d 或更长,观察肠道反应的演变过程[16]。该方法贴近肠道菌群失调与 UC 发病的关系,适用于研究菌群与炎症的相互作用,但实验稳定性和重复性仍需提高。此外,一些基因修饰小鼠模型的表型依赖于肠道菌群,如 *Mdr*1*a* 小鼠因黏膜屏障缺陷导致微生物产物蓄积和慢性炎症,表现出与人类结肠炎相似的病理[17]。研究显示 *Mdr*1*a* 缺陷引发结肠上皮屏障功能紊乱,T 细胞异常激活并产生氧化应激和免疫反应,与人 UC 的炎症通路相符。这些“菌群驱动”模型突出了微生态失调在 UC 发病中的关键作用,可用于探索菌群-宿主相互作用机制。

1.3 屏障蛋白基因敲除法

肠黏膜屏障结构和功能的维持对大肠免疫内环境、稳态至关重要,屏障蛋白基因敲除法构建 UC 动物模型的成功率高,在实验过程中无自愈倾向,如通过敲除如白细胞介素-22(interleukin 22, *Il*22)基因破坏肠道屏障功能,导致炎症加重、细胞增殖与凋亡失衡[18]。该模型高度模拟 UC 的病理特征,且炎症损伤部位精准可控。

1.4 炎症调控基因敲除法

肠道内炎症平衡被破坏是 UC 的发病机制之一[19],经典例子是敲除小鼠白细胞介素-10(interleukin 10, *Il*10)基因、肿瘤坏死因子诱导蛋白 3(tumor necrosis factor alpha-induced protein 3, *tnfaip*3)基因,因先天缺乏抗炎细胞因子而在无特定病原条件下自发发生慢性结肠炎,反映免疫失衡对肠炎的促进作用[20-21]。新的基因靶向模型更关注免疫和炎症信号的调控,如甲基转移酶样蛋白 14 基因(methyltransferase-like 14, *Mettl*14)条件性敲除小鼠在 T 细胞中删除 m6A 甲基转移酶 METTL14,可自发出现严重结肠炎,伴有 Th1/Th17 型炎症,并证实由调节性 T 细胞功能缺陷所致[22]。该模型的结肠炎还能被抗生素明显减轻,说明炎症表型依赖于肠道微生物,为研究表观遗传-免疫-菌群轴提供了新工具。同样, *Mrgprb*2 基因敲除[23]通过影响肠道肥大细胞功能加重了 DSS 结肠炎:Mas 相关 G 蛋白偶联受体 B2(Mas-related G protein-coupled receptor member B2, *Mrgprb*2)基因小鼠结肠炎症更重,伴随有益菌减少(如乳杆菌)和有害菌增多(如大肠杆菌属),黏膜屏障蛋白 2(mucin 2, oligomeric mucus/gel-forming, Muc2)和闭合蛋白(Occludin)下降。这提示敲除炎症调控或屏障相关基因(如 *Mrgprb*2、*Il*22 等)会破坏黏膜稳态,导致菌群紊乱和炎症恶化。此外,新型自发性 UC 模型 Winnie 小鼠(*Muc*2 基因错义突变)[24]近年来受到关注,该小鼠因杯状细胞黏蛋白异常折叠而逐渐发生慢性结肠炎,炎症随年龄加重,并伴有与人 UC 类似的固有免疫和适应免疫反应及菌群改变;Winnie 小鼠甚至出现直肠脱垂和肠外表现等并发症,增强

了模型的临床相关性。总之,基因缺陷模型能够精确模拟特定通路对 UC 发病的贡献,例如黏膜屏障功能障碍(*Muc*2, *Mdr*1*a* 缺陷)、抗炎失衡(*Il*10 缺失)或炎症通路过度激活(*tnfaip*3 缺失等)。这些模型大多具有可遗传、表型稳定的优点,利于深入机制研究,是近年的研究热点和未来方向,病理更接近人类 UC[25]。不过需注意单一基因缺陷通常不能完全再现 UC 复杂病因,常需结合环境或菌群因素以提高外推性。以上各种方法具体见表 1。

表 1　UC 动物模型的造模方法

Table 1　Modeling methods of UC animal models

诱导方法 Induction method	方法 Methods	造模周期 Mold cycle	模型评价 Model evaluation
DSS 法[11] DSS method[11]	自由饮用或间歇性饮用诱导慢性炎症模型 A model of chronic inflammation induced by ad libitum or intermittent drinking	1 ~ 2 周 1 ~ 2 weeks	建模简便,急性炎症特征典型,适用于初步药效筛选和病理观察,但病理机制相对单一,难以再现慢性复发病程,对中医“虚实夹杂”“湿热交错”等证型拟合度不高 Modeling is simple and the characteristics of acute inflammation are typical, which is suitable for preliminary pharmacodynamic screening and pathological observation, but the pathological mechanism is relatively simple, it is difficult to reproduce the course of chronic recurrence, and the fit degree of syndrome types such as “virtual and real mixture” and “damp-heat interlacing” in traditional Chinese medicine is not high
TNBS、乙酸法[12] TNBS, acetic acid method[12]	TNBS 溶于乙醇中灌肠 TNBS dissolved in ethanol enema	1 ~ 2 周 1 ~ 2 weeks	
微生物诱导法[16-17] Microbial induction method[16-17]	提取 UC 患者粪便菌群移植或单一菌株灌肠 Extraction of fecal microbiota from UC patients for transplantation or single-strain enema	1 ~ 2 周 1 ~ 2 weeks	通过肠道菌群失调诱导肠炎,较好模拟 UC“菌群-免疫-黏膜”相互作用,便于中医“湿热由内生、脾虚为本”等理论的演绎。但实验条件要求较高,重复性受菌群稳定性影响大 Intestinal flora disorder induces enteritis, which better simulates the interaction of UC “microbiota-immunity-mucosa”, which is convenient for the interpretation of theories such as “dampness and heat are endogenous and spleen deficiency is the foundation” in traditional Chinese medicine. However, the experimental conditions are highly demanding, and the reproducibility is greatly affected by the stability of the microflora
抗炎因子、屏障蛋白基因敲除[18,20-24] Knockout of anti-inflammatory factor and barrier protein genes[18, 20-24]	*Il*10、*Il*22、*tnfaip*3、*Mrgprb*2 基因敲除;*Muc*2 基因错义突变 *Il*10, *Il*22, *tnfaip*3, *Mrgprb*2 gene knockout, missense mutation in the *Muc*2 gene	3 ~ 4 周 3 ~ 4 weeks	能精准模拟特定致病机制(如 IL10 缺失引发免疫失衡),适合深入机制研究与靶点干预,尤其适用于“内在体质偏颇”型中医病机研究,但技术复杂、周期长、成本高,不利于中药多成分、多靶点研究的整体评估 It can accurately simulate specific pathogenic mechanisms (such as immune imbalance caused by IL10 deficiency), which is suitable for in-depth mechanism research and target intervention, especially for TCM pathogenesis research with “internal constitution bias”. However, the technology is complex, the cycle is long, and the cost is high, which is not conducive to the overall evaluation of multi-component and multi-target research in traditional Chinese medicine

2　UC 大肠湿热病证结合模型研究现状

中医认为,常说的“湿热证”是广义的病理范畴,而“UC 大肠湿热证”则特指以大肠湿热为主的证型。围绕大肠湿热证展开,分析传统与复合模型的策略。

2.1　传统大肠湿热证模型

以往的大肠肠湿热证模型通过简单叠加“病”(UC 病变)和“证”(湿热症状)来构建。常用方法包括内因饮食(高脂高糖饮食)结合外因环境(高温高湿箱)致大鼠出现泄泻、黏液脓血便等湿热症状诱导肠黏膜溃疡[26]。具体来说,高脂饲料 + 高温高湿环境[27-28]或高脂高糖白酒灌胃[29]连续 2 ~ 3 周可诱导湿热内盛表现;高脂高糖饮食配合湿热环境[30]也能在 2 周内诱发类似症候;若再加入间歇性饥饱失调和白酒灌胃[31]等,应激强度加大,但模型周期延长至 3 周以上。

这些模型一定程度上再现了大肠湿热证的复杂病因(饮食不节、嗜酒、暑湿环境等),符合中医“湿热既可外感亦可内生”的理论。然而,现有研究多为对造模条件的简单调整,缺乏对其他影响因素的深入考虑。模型成功与否主要凭借动物的粪便性状、体质量变化等指标判断,定量评价不足,在科学性和可重复性方面仍有提升空间。

2.2 湿热与结肠炎模型的复合策略

近年来,研究趋向将“湿热证”诱因与结肠炎动物模型(如 DSS、TNBS)叠加,构建“病”与“证”并重的复合型模型。包括饮食干预、高压氧或病原感染等方式,结合化学诱导法(如 DSS),通过多种手段共同诱发肠炎。例如,通过高温高湿环境暴露并给予高脂饮食、酒精摄入、高脂高糖饮食联合高温高湿或单独高脂饮食处理,最终结合 DSS 建模[32-34]。此方法能够模拟湿热环境的影响,接近部分 UC 大肠湿热发病机制,但建模时间长,成功率有待提高。具体见表 2。

3 UC 病证结合动物模型评价

3.1 多组学技术在模型评估中的应用

传统模型评价多采用疾病活动指数(disease activity index, DAI)、组织病理评分等指标。如今,多组学(基因组、转录组、蛋白质组、代谢组等)在动物模型评估中展现出巨大潜力。以代谢组学为例[35-36],有研究比较了炎症性肠病(inflammatory bowel disease, IBD)大肠湿热证与脾虚证患者的代谢谱差异,发现两者在甘油磷脂代谢等通路上存在显著不同。多种芳香族代谢物和脂质衍生物可作为潜在标志物,将湿热型 IBD 与非湿热型区分开来。这些发现不仅丰富了证候的生物学内涵,也为动物模型的客观评价提供了依据—可检测模型动物血浆/粪便中的代谢物谱是否贴近湿热型患者特征,从而判断模型成功与否。同样地,宏基因组学和 16S rRNA 测序[37-38]已用于量化模型的菌群失调程度。此外,蛋白质组和转录组技术可检出模型中炎症通路的整体变化。例如,通过 RNA 测序可了解湿热模型中核因子 κB (nuclear factor kappa B, NF-κB),贾纳斯激酶/信号转导与转录激活因子(Janus kinase/signal tansducer and activator of transcription, JAK/STAT)等通路基因的上调情况,验证其与人类大肠湿热证 UC 的吻合度。这些组学数据作为客观指标,有助于将以往依赖经验的证候判断转化为可量化的分子指标,使模型评价更加科学精细[39]。

3.2 生物标志物与影像学评价

随着对 UC 生物学机制认识加深,诸多炎症和损伤指标被用于模型评价。传统炎症标志物如 C 反应蛋白(c-reactive protein, CRP)[40]、红细胞沉降率(erythrocyte sedimentation rate, ESR)[41]等在动物模型中同样适用,可反映全身炎症水平。更有特异性的指标如粪便钙卫蛋白(fecal calprotectin, FC)[42],已经在临床用于监测肠道炎症活动度,在模型研究中,若造模成功往往能检测到粪便钙卫蛋白显著升高,提示结肠中性粒细胞浸润。近期还有学者建立了微生物失调指数(dysbiosis index)[43]用于量化小鼠菌群紊乱程度,从而客观评估诸如 DSS 模型的肠道生态失衡状况。除了体液标志物,成像技术也逐渐用于无创监测模型炎症。红外热成像(热红外热图)能够捕捉体表温度异常,反映炎症部位的血流和代谢变化;有报告指出,在 IBD 患者中红外热像可作为评估疾病活动的一种辅助手段。对应到动物实验中,如果造模成功,引发结肠局部炎症,则模型动物腹部或肛周皮肤的温度分布会有异常“热点”,可被红外相机检测到。这种方法实现了对模型炎症的实时、在体监测,避免了过早处死取材。此外,新近发展起来的分子影像探针也用于 UC 模型评价,例如近红外荧光探针检测结肠黏膜的通透性和中性粒细胞活性[44]、光学相干断层成像[45]评估肠黏膜结构损伤等。这些影像手段提供了独特视角,可动态观察模型炎症进展和对治疗反应。最后,行为学量化[46]越来越受到重视。UC 模型不仅表现在结肠局部病变,还伴随动物全身状态和行为的改变。研究发现,结肠炎小鼠常出现活动力下降、焦虑样行为增加等现象。例如在开放场实验中,DSS 模型小鼠的自主活动距离显著减少,更多时间停留在边缘(提示焦虑增强)。又如一些模型大鼠表现出肠病相关的抑郁行为,可通过强迫游泳、不愉快刺激反应等实验加以量化。这些行为学指标从神经-肠道轴角度为模型功能性影响提供了评价手段,与分子和组织学指标相互印证,使模型表征更全面。

表 2　大肠湿热证动物模型的造模方法

Table 2　Modeling methods for animal models of colorectal dampness and heat syndrome

诱导方式 Induction method	方法 Method	造模周期 Mold cycle	模型评价 Model evaluation
高脂 + 高温高湿[27-28] High fat + high temperature and high humidity[27-28]	高脂饮食 + 湿热环境(人工气候箱,设置参数温度 35 ℃,湿度 85%;8 h/d)14 d High-fat diet + humid and hot environment (artificial climate chamber, set parameter temperature 35 ℃, humidity 85%; 8 h/d) for 14 d	2 ~ 3 周 2 ~ 3 weeks	优点:符合中医大肠湿热证的发病特点、能够全面模拟大肠湿热证的复杂病理机制、造模方法多样,易于根据实验需求调整强度。不足:周期较长,不同动物对饮食、环境和药物的敏感性不同,湿热程度不稳定 Advantages: it conforms to the pathogenesis characteristics of damp-heat syndrome in traditional Chinese medicine, can comprehensively simulate the complex pathological mechanism of damp-heat syndrome, has a variety of modeling methods, and is easy to adjust the intensity according to experimental needs. Disadvantages: the cycle is long, different animals have different sensitivities to diet, environment and drugs, and the degree of dampness and heat is unstable
高脂高糖 + 乙醇[29] High fat and high sugar + ethanol[29]	高糖饮食 + 隔日油脂灌服与隔日灌服乙醇交替干预 20 d High-fat and high-sugar diet + alternate fat administration and alternate day alcohol were alternately intervened for 20 d		
高脂高糖 + 高温高湿[30] High fat and high sugar + high temperature and high humidity[30]	高脂高糖饮食 + 湿热环境 14 d High-fat and high-sugar diet + humid and hot environment for 14 d		
高脂高糖 + 高温高湿 + 饥饱交替 + 乙醇[31] High fat and high sugar + high temperature and high humidity + hunger and satiety alternately + ethanol[31]	前 14 d 里,自由饮用蜂蜜水,单号管饲猪油,双号禁食不禁饮,经受饥饱交替;第 15 ~ 22 天,自由饮用蜂蜜水,自由采食普通饲料,每日管饲乙醇 + 每日湿热环境 In the first 14 days, he drank honey water freely, fed lard with a single tube, fasted and did not drink at double number, and experienced hunger and satiety. On the 15th ~ 22nd day, drink honey water freely, eat ordinary feed ad libitum, and feed alcohol per day + daily hot and humid environment		
湿热与结肠炎模型的复合策略[32] A composite strategy of damp heat and colitis models[32]	高温高湿环境暴露并给予高脂饮食、乙醇摄入,最终结合 5% DSS Exposure to high temperature and humidity and high-fat diet, alcohol intake, and finally 5% DSS	6 ~ 12 周 6 ~ 12 weeks	优点:贴近人类 UC 的发病背景,能同时观察饮食、环境和宿主基因等多方面的综合作用,饮食和环境条件易控制,成本较低。不足:造模周期较长,饮食和环境诱导的炎症表型可能因动物个体差异较大,结果波动性高,病变轻微,机制不明,食物或环境诱导的特定分子机制仍需深入研究 Advantages: close to the pathogenesis background of human UC, it can observe the comprehensive effects of diet, environment and host genes at the same time, and the dietary and environmental conditions are easy to control and the cost is low. Disadvantages: the modeling cycle is long, the inflammatory phenotype induced by diet and environment may vary greatly from animal to individual, the results are highly volatile, the lesion is mild, the mechanism is unknown, and the specific molecular mechanism induced by food or environment still needs to be further studied

3.3　行为学与中医证候观察

临床与模型症状互相对应,大肠湿热的临床表现包括发热、口渴、腹胀腹痛、下痢脓血、便黏腥臭、肛门灼热、小便短黄等;而动物模型则表现为肛温升高、饮水量增加、蜷缩、便溏便血、肛周污秽、小便色深量少等体征[47]。结合病理学检测,如小鼠舌组织与肠道组织的病理学检查[48],观察炎症细胞浸润及组织损伤情况。此外,湿热

证的特征性血液学指标如血小板(platelet,PLT)、ESR、CRP、纤维蛋白原(fibrinogen,Fg)、D-二聚体(D-dimer)水平升高明显[49]。免疫学指标例如胃动素、胃泌素等指标的免疫组化阳性表达水平[50]。

但动物模型中的行为多停留在简单的指标上(如饮水次数、肛温等),缺乏系统性和定量化,难以与现代医学标准对接。见表 3。

表 3 病证结合 UC 动物模型的多维评价体系

Table 3 Multi-dimensional evaluation system of disease syndrome combined with UC animal model

评价维度 Evaluation dimensions	指标类别 Indicator category	代表性指标或技术 Representative indicators or techniques	评价意义 Evaluative significance
中医证候特征[47] TCM syndrome characteristics[47]	体征观察 Signs observed	肛温升高、饮水量增加、便稀肛污、小便黄等 Increased anal temperature, increased water intake, loose stools, anal dirt, yellow urine, etc	表征“湿热”体质外在反应 To characterize the external reaction of “damp heat” constitution
组织病理学[40-42] Histopathology[40-42]	苏木-伊红(HE)、PAS 染色 Hematoxylin-Eosin(HE), PAS staining	肠黏膜结构破坏、炎性细胞浸润 Structural destruction of the intestinal mucosa and inflammatory cell infiltration	反映 UC 炎症病理进展 It reflects the pathological progression of UC inflammation
炎症标志物[49] Inflammatory markers[49]	血清/组织 ELISA Serum/tissue ELISA	TNF-α、IL-1β、CRP、FC 等 TNF-α、IL-1β、CRP、FC and so on	判断炎症程度与治疗效果 Determine the degree of inflammation and the effect of treatment
微生态评价[35,38] Microecology evaluation[35,38]	宏基因组、16S rRNA 测序 Metagenomic, 16S rRNA sequencing	α 多样性、优势菌变化 α diversity and changes in dominant bacteria	反映肠道菌群与 UC/大肠湿热证的相关性 Reflects the correlation between intestinal microbiota and UC/damp-heat syndrome
代谢组学[35] Metabolomics[35]	LC-MS、GC-MS	甘油磷脂类、胆汁酸代谢物等 Glycerophospholipids, bile acid metabolites and so on	识别湿热型 UC 的潜在生物标志物 dentify potential biomarkers of damp-heat UC
行为学指标[46] Behavioral indicators[46]	开放场、强迫游泳实验 Open field, forced swimming experiment	活动减少、焦虑样行为增加 Decreased activity and increased anxiety-like behavior	模拟肠脑轴异常引发的情绪行为变化 Simulates changes in mood and behavior caused by abnormalities in the gut-brain axis
成像学观察[44-45] Imaging observations[44-45]	红外热成像、近红外探针 Infrared thermography, near-infrared probes	腹部热点、通透性变化、局部热区增强 Abdominal hot spots, permeability changes, and increased local hot spots	实现非侵入性动态监测 Achieve non-invasive dynamic monitoring

4 UC 病证结合动物模型建立及评价体系优化的思考

综上所述,尽管已有一些成果,但现有 UC 病证结合模型在方法和评价上仍存在局限:多数模型依赖单一因素构建,评价缺乏量化和系统标准,难以同时模拟 UC 复杂的致病环境和个体体质背景。因此,有必要提出改进策略,进一步优化造模手段和评价体系。

4.1 模型构建策略优化

可在传统化学诱导法(如 DSS、TNBS)基础上,可考虑引入微生态干预与基因修饰手段,提高模型的复杂性与临床相关性。例如,可将高脂高糖饮食 + 湿热环境诱导的“大肠湿热证”模型,与致病菌移植[51-52]或基因敲除模型(如 *Il*10、*Muc*2、*Mrgprb*2 等)相结合,综合模拟环境、菌群与遗传因素共同作用下的发病状态。特别是“菌群-免疫-屏障”轴线的干预设计,能更好契合中医病机中“湿热内生”“脾虚为本”的理论内涵。复合建模还应结合研究目标权衡实验可控性与证候适配性:基础性研究中可优先使用 DSS 模型,机制研究可聚焦基因修饰模型,而病证结合模型则适用于中药作用机制及疗效验证,强调“整体调控”与“辨证论治”的干预特征。

4.2 评价体系优化思路

为提升病证结合模型的科学性与适配性,可尝试建立一套结构清晰、指标统一的综合评价体系[53]。在整合过程中,可考虑从以下 3 个方面着手:首先,可考虑构建“中医证候-现代医学-多组学”三维融合的评价框架。中医部分强调“湿热”体征的可观察性与标准化(如便溏、肛温、饮水量等);现代医学侧重炎症指标(如 CRP、白细胞介

素-6(interleukin 6,IL-6)、FC)及组织病理变化;组学层面则引入宏基因组、代谢组、行为学等新兴技术,量化微生态与功能状态,增强评价的客观性。例如,可通过检测模型动物血浆/粪便的代谢物谱是否与湿热型患者一致来判断模型的"湿热度"。其次,推动关键指标的定量化和可重复化,提升与临床相关性的对接程度。未来可探索以炎症因子水平、菌群多样性指数及特征性代谢产物为核心的评价模块,作为模型可靠性与稳定性的基础衡量标准。最后,应根据不同研究目的建立分层次的评价标准体系,明确基础研究、机制探讨与中药干预验证所需的模型拟合度与评价重点,提升模型评价的针对性和推广性。通过上述整合路径,有望实现从经验判断向精准评价的转变,为中医药干预 UC 机制研究提供坚实模型支撑。

综上,UC 大肠湿热病证结合动物模型是连接中医理论与现代医学的重要桥梁。通过优化模型的构建方法与评价体系,病证结合模型在中药作用机制研究以及中西医结合治疗探索中具有巨大潜力。结合微生物诱导法、基因修饰模型以及特殊造模技术,与大肠湿热证的饮食和环境模拟相配合,以期提升 UC 模型的复杂性和临床相关性。然而,模型设计需平衡不同方法的优缺点,避免单一化或盲目叠加。此外,目前病证结合模型在中医证候量化、慢性炎症的长期模拟等方面仍有明显不足。未来可尝试中医表型的量化标准,将中医大肠湿热证特征与西医客观指标(如炎症标志物)有机结合,构建多维评价体系。优化造模方案,精确模拟 UC 的环境因素、遗传背景和诱导方式,提升模型在复杂病理状态下的稳定性与适用性。此外,利用微生物组学、代谢组学和基因组学等现代技术,揭示大肠的生物学机制,亦可为模型优化提供新思路。优化模型的构建与评价,进一步增强其科学性、实用性和推广性,病证结合模型有望为 UC 的基础研究与临床治疗提供有力支撑。

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本刊是由中国实验动物学会与中国医学科学院医学实验动物研究所主办的全国性高级学术刊物(月刊),以理论与实践、普及与提高相结合为宗旨,征稿的范围是与实验动物与动物实验相关的生命科学各分支学科,栏目设置包括研究报告、研究快报和进展与综述。要求来稿材料翔实、数据可靠、文字简练、观点明确、论证合理,有创新、有突破、有新意。

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