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李兰娟，中国工程院院士，浙江大学教授、主任医师、博士生导师。现为传染病诊治国家重点实验室主任，感染性疾病诊治协同创新中心主任，兼任教育部科技委生物与医学学部主任，中华预防医学会副会长，中国卫生信息协会副会长，国家卫计委人口健康信息化专家咨询委员会主任等。承担国家“863 计划”、“973 项目”、“十五”攻关、国家自然科学基金重点项目等课题 20 余项，发表论文 400 余篇，其中在 *Nature*、*Lancet*、*NEJM*、*Nature Communication* 等 SCI 收录杂志发表 200 余篇。获国家科技进步一等奖 2 项，国家科技进步奖(创新团队) 1 项，国家科技进步二等奖 2 项，浙江省科技进步一等奖 6 项。获“全国优秀科技工作者”和“全国杰出专业技术人才”称号、何梁何利基金科技进步奖、光华工程科技奖、谈家桢科学奖临床医学奖等。

人体微生态与健康和疾病

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摘要：人体微生态是人体内的微生物生态群落，是存在于人体组织和体液中的共生和病原微生物的总和，也是近年来发现的 “ 新器官 ”，它在维持人体健康过程中扮演着重要角色。微生态与宿主间有着全面广泛的相互作用机制，微生态失衡与疾病的发生发展密切相关。本文概述了人体微生态与健康和疾病研究的重要意义，并对国内外研究进展作总结评述。

关键词：人体微生态，健康，疾病

微生态是存在于植物或动物体内的包括共生微生物和病原微生物的共生生态群落。人体微生态是近年来发现的具有重要作用的“新器官”[1]。人体存在数目庞大(超 10^{14} 个，干重约占人体总重 1%至 2%)且结构复杂(包括细菌、古细菌、原生生物、真菌和病毒等)的微生物群落，定殖于胃肠道、口腔、皮肤、泌尿生殖道、呼吸道等，它们所编码的基因数量可达人体自身基因数量 150 倍，相当于人体的“第二个基因组”，包含重要的遗传信息[2–3]。人体微生态在维持人体健康和疾病的发生发展过程中都扮演着重要角色。一方面，它是宿主消化吸收、免疫反应、物质能量代谢的重要维持者，直接或间接调控消化系统、免疫系统、神经系统和大脑等器官功能[4]；另一方面，人体微生态失衡与多种疾病的发病机制密切相关，同时也是药物代谢、微生物耐药的中间站；并且随着年龄增长，微生态不断变化，与人的衰老、寿命息息相关。

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目前人体微生态研究已进入高速发展期，宏基因组、宏转录组、代谢组等研究技术不断革新，推动了人体微生态研究的突飞猛进。本文概述了人体微生态与健康和疾病研究的重要意义，并对近年来国内外相关研究进展进行总结和评述。

1 “人体微生态与健康和疾病”研究具有重大意义

人体微生态与健康和疾病研究正在引发生命科学、医学、药学、机械、信息等领域的重大变革。人体微生态“器官”的确立，翻开了生命起源、进化、发育等科学问题研究的新篇章，颠覆了医学上关于感染、肝病、肿瘤、代谢等重大疾病的传统认识，催生了药物研发新靶点、新途径的应用，激起了科学研究、临床诊断相关新技术、新设备的研发浪潮，推动了大数据分析、信息产业的发展。

全球各国都高度重视人体微生态研究。近 10 年，欧盟已先后启动 2 项人体微生态研究计划；美国NIH也先后启动了2项人体微生态研究计划，并且美国政府于 2016 年启动“国家微生物组计划”；加拿大、日本、法国、新加坡等也分别实施相关计划，投资数十亿美元，试图在研究人体微生态、解决重大科学问题的同时，撬动医药、设备、信息等相关行业的发展，成为国家经济增长点[5–6]。

我国是人体微生态研究起步较早、成果突出的国家。“十一五”和“十二五”期间，“973 项目”、“863 计划”、国家自然科学基金等项目大力支持人体微生态的基础研究、关键技术开发和资源平台建设。2007 年，李兰娟院士领衔的“肠道微生态与感染”项目首次获得“973 项目”资助，与全国 11 家单位共同研究，《Science》杂志对该项目给予了专门报道，是全球最早的人体微生态研究项目之一；2013 年，该项目再次获得“973 项目”支持，极大推动了国内相关研究的发展。

2 “人体微生态与健康和疾病”的国内外研究进展

2.1 微生态“器官”的功能

微生态作为额外的“器官”在宿主生命活动中发挥不可或缺的作用：促进宿主本身难以消化的营养物质的代谢与吸收，生产生命必需维生素；参与上皮细胞的发育和分化，诱导黏膜免疫、免疫应答；保护宿主免于条件致病菌的侵袭；参与维持组织内稳态，预防疾病发生。此外，在生命早期，微生态对宿主器官结构和功能的成熟，免疫、神经、消化等系统的发育，以及其他正常生理机能的完善都有重要作用。

胃肠道是微生物群落最主要的栖息地，也是微生物与宿主免疫系统发生相互作用的最主要场所，微生态对肠道免疫系统的发育与功能调节起重要作用。大量研究表明，肠道微生态参与胃肠相关淋巴组织的发育和成熟，无菌动物体内免疫系统发育、免疫细胞分化、免疫应答等都存在缺陷：其孤立淋巴滤泡发育不完全，派尔集合淋巴结、肠系膜淋巴结和脾白髓均小于或少于 SPF 动物；天然杀伤细胞(NK cell)的分化减少，恒定天然杀伤 T 细胞(iNKT cell)增多，调控 T 细胞(T_{Reg} cell)和辅助 T 细胞(T_H cell)分化比例失调，导致炎症相关细胞因子异常释放；TLR2、TLR4、NOD2 等模式识别受体和 MYD88、TRIF 等衔接蛋白表达障碍，抗原提呈细胞功能受损；抗菌肽、抗体、免疫球蛋白等产量减少[7–10]。微生态也参与宿主黏膜免疫的建立。黏液层由杯状细胞分泌的黏蛋白构

成，是肠道内容物和肠上皮细胞间的物理-化学屏障。黏蛋白上丰富的聚糖结构域可为某些分泌特定凝集素或糖苷酶的微生物提供结合位点，达到选择性粘附目的[11]。黏液层-微生物相互作用对于维持肠道内平衡、保持宿主健康有重要意义。Xia 等人发现黏蛋白异常糖基化会引起黏膜屏障功能受损、肠道微生物过度增长，导致自发性结肠炎发生[12]。黏液层的厚度和构成主要受肠道微生态影响：无菌动物肠道内杯状细胞减少，黏液层更薄，中性黏蛋白的比例增高；而饲喂脂多糖、肽聚糖等细菌产物后无菌动物肠道黏液层迅速恢复[13–14]。

肠道微生态同样参与宿主中枢神经系统、肠道神经系统等的发育。神经蛋白 PSD-95 和突触小泡蛋白与中枢神经系统的突触形成以及神经兴奋突触的成熟相关，异常表达会影响大脑发育。Diaz 等人发现与 SPF 动物相比，无菌动物生命早期的纹状体中 PSD-95 和突触小泡蛋白的表达升高，而微生态干预后这两种蛋白的表达恢复正常水平[15]。小神经胶质细胞(microglia，MG)是中枢神经系统中的巨噬细胞，在神经元的生理活动中起着支持、营养、保护及修复等重要功能。Prinz 等人发现微生态对 MG 的体内平衡有重要作用，无菌小鼠和微生态复杂性受限小鼠(体内仅有 *Bacteroides distasonis*，*Lactobacillus salivarius*，*Clostridium cluster XIV* 三株菌定殖)体内 MG 都存在全局性缺陷，细胞形态改变且呈未成熟表型，MG 的不稳定状态导致机体固有免疫应答机制受损；而通过重建微生态多样性或补充微生物发酵产生的短链脂肪酸可修复受损的 MG，使其达到稳态[16]。Ratcliffe 等人发现相比于 SPF 小鼠，无菌小鼠由于缺少肠道微生物定殖，在出生第 3 天就出现肠肌间神经丛结构异常，神经元数量减少且氮能神经元比例升高，这一变化导致肠肌收缩的频率和幅度下降，影响肠动力[17]。肠道内的短链脂肪酸、脂多糖等微生物产物会调节肠上皮细胞中血清素转运体的表达及其活性，影响宿主体内 5-羟色胺水平，从而起到调节肠道神经系统功能的作用[18]。Hsiao 等人将人源和鼠源的肠道微生物移植到无菌小鼠，发现其结肠和血清中 5-羟色胺水平升高，与无菌相关的肠动力障碍症状得到缓解[19]。此外，肠道微生态信号可被 TLR2、TLR4 等 Toll 样受体(TLR)识别，微生物作用也会上调肠神经间质细胞 TLR 的表达[20]；而 TLR-微生态通路对促进肠神经细胞的成熟、肠神经网络的发育、肠神经系统的内稳态调节及其功能的组织都有重要意义[18,21]。

2.2 人体微生态“器官”的特点

人体微生态的多样性和特异性与遗传、饮食习惯等密切相关。Ley 等人对同卵/异卵双胞胎成年人的肠道微生态进行比较，发现同卵双胞胎之间的微生物类型更为接近，尤其是克里斯滕森菌科(Christensenellaceae)细菌，具有高度遗传性且在身材苗条的个体内丰度更高[22]，这一发现确定了宿主遗传因素的重要性。根据人体内微生物的种类和数量，可将不同人群按“肠型”(Enterotypes)进行划分。肠型的定义是多维空间内高度聚集的微生物群落结构，它代表了宿主-微生物共生稳态时的人体微生态结构特征[23]；目前研究确定了 4 种肠型：拟杆菌型(Bacteroides)、普氏菌型(Prevotella)、瘤胃球菌型(Rminococcus)和厚壁菌型(Firmicutes)[23–24]。Wu 等人研究发现，肠型的形成与个人长期饮食习惯密切相关，即使短期内饮食发生改变，肠型仍能保持相对稳定[25]。然而即便如此，人体肠道微生物组成仍可在 1 日之内发生快速、可重复的改变，以应对人类多元化饮食习惯导致的食物成分

突然变化[26]。微生态的改变也会影响宿主。将在身材苗条者体内有更多富集的小克里斯滕森氏菌(*Christensenellaceae minuta*)移植到无菌小鼠肠道，能够抑制小鼠体重的增加[22]；Gordon 等人从 4 对一胖一瘦的人类双胞胎获取肠道细菌并移植到无菌小鼠，发现尽管所有小鼠饮食量相同，但接受了胖者细菌的小鼠增加了更多体重和脂肪；而将它们与接受了瘦者细菌的小鼠合住后，其微生态发生变化，肥胖状况得到改善[27]。这些研究提示某些肠道微生物不仅可遗传，还可通过传播影响其他个体的微生态，而且某些肠道微生物可作为标志物用于预测疾病风险，具有进一步挖掘潜力。

2.3 人体微生态与感染性疾病

我国感染病患者众多，广谱抗菌药物的长期大量使用不可避免地加重人体微生态失衡，导致细菌易位、肠源性感染甚至致命感染发生，也会增加住院患者遭受院内感染的几率。

健全的人体微生态可以抵御病原菌的侵染。粪便移植成功治疗艰难梭菌反复感染是感染微生态学理论的成功体现，其疗效明显优于抗生素治疗[28]。但是，粪便移植还存在许多未知风险，因此提取肠道有益菌群用于疾病治疗将是未来医疗发展的重要方向。肠道菌群除了直接抵御病原菌入侵外，还可以通过影响宿主免疫调控感染。幽门螺旋杆菌感染会引发胃溃疡、慢性萎缩性胃炎、胃癌和黏膜相关淋巴瘤等疾病[29–30]。全世界人口中超过 50%携带幽门螺旋杆菌(*Helicobacter pylori*，HP)，而携带者中发病人群比例约为 10%。研究表明，健康人群(非 HP 携带者)、HP 携带者、HP 感染患者的胃部微生态结构有明显差异[31–33]，暗示人体内存在与微生态直接相关的免疫机制以抵御 HP 感染。通过补充某些有益菌重建胃部微生态平衡治疗 HP 感染的研究已有相关报道[34–36]。此外，Buffie 等人发现肠道 *Clostridium scindens* 菌可通过改变肠道中胆汁酸的成分来抑制艰难梭菌感染[37]；Soares 等人发现人肠道菌群中某些特异性细菌可表达 α-半乳糖苷酶，诱导宿主产生天然抗体触发自然防御机制，预防疟疾传播[38]；Jones 等人发现某些肠杆菌可在宿主体内表达特定型组织血型抗原(HBGA)，促进诺如病毒感染 B 淋巴细胞[39]。这些研究为通过调节肠道菌群治疗感染性疾病提供了宝贵依据。

近年来，使用微生态调节剂辅助治疗新突发传染病取得了重大进展。Li 等人发现 H7N9 患者肠道微生态严重失衡，双歧杆菌与肠杆菌比值(B/E)值远小于健康人群，部分患者双歧杆菌量甚至低于检测线；患者接受抗生素治疗后肠道菌群丰度大幅波动，有益菌急剧下降；经益生菌治疗，患者肠道中双歧杆菌、乳杆菌、产丁酸菌丰度明显上升[40]，显示了维持微生态平衡在传染病诊治中的重要意义。

2.4 人体微生态与消化道疾病

肠道微生态改变与炎症性肠病(IBD)、肠易激综合征(IBS)、胃肠道肿瘤等诸多消化道疾病的发生发展密切相关。IBD 患者肠道微生态显著改变，导致免疫细胞的非正常活化、肠道通透性增加，进而失去免疫耐受功能[41–42]；O'Toole 等人发现部分 IBS 患者也存在肠道菌群紊乱现象：厚壁菌增多、拟杆菌减少[43]；Ahn 等人研究发现结直肠癌患者肠道微生物多样性减少，患者肠道内能抑制炎症反应的梭状芽孢杆菌减少而促炎细菌增多[44]，这些研究表明肠道微生态平衡被破坏会引发宿主的免疫、炎症反应，导致上述疾病的发展。Wang 等

人研究发现结肠腺瘤和结肠癌患者的肠型多为拟杆菌型，而作为对照的健康人群以瘤胃球菌型居多，随着结肠癌的发生和发展，患者肠型还存在由瘤胃球菌型向拟杆菌型转变的趋势[45]。拟杆菌型肠型人群对应的饮食习惯为高蛋白、高动物脂肪的摄入[25]，这也印证了红肉和加工肉制品消费是直结肠癌发生的风险因素之一[46]。Yu 等人发现 I 型溶血性大肠杆菌促进女性结肠肿瘤发生，是女性大肠癌发生的潜在致病因子，可作为女性大肠癌易感性分析的生物标志物[47]；而具核梭杆菌可作为大肠肿瘤诊断的粪便细菌标志物[48]，这些研究提示肠道微生物既可作为预防和治疗肠道癌症的靶点，也有助于推动肠道肿瘤诊断技术向早期化、精确化、低成本化、非损伤化方向发展。

目前，微生态制剂已在 IBS、IBD、结肠癌、感染性腹泻、抗生素相关性腹泻、坏死性结肠炎等多种消化道疾病的治疗中获得良好效果[49–52]。Kweon 等人发现，肠道病毒通过 TLR3 和 TLR7 介导的 β 干扰素的产生改善肠道炎症[53]。Atarashi 等人从健康成人中筛选出的 17 株梭菌属，能够显著促进 T_{Reg} 细胞增殖分化并在结肠炎和过敏性腹泻模型中起治疗作用[54]。

2.5 人体微生态与肝病

肝脏与肠道存在特殊的生理与解剖关系，微生物通过肝-肠循环和微生态-肝脏轴，在肝脏炎症、损伤、慢性纤维化发生发展中发挥重要作用。Li 等人发现肝硬化患者肠道菌群结构与健康对照存在显著差异，并且肠道微生态失衡程度与肝硬化病情严重程度有显著相关性[55]。为进一步揭示肝硬化患者肠道菌群的组成特征，Li 等人通过研究建立了世界首个肝硬化患者肠道菌群基因集，阐明肝硬化患者肠道菌群的宏基因变化，并建立疾病预测模型，从肠道菌群紊乱的角度揭示肝硬化的发生发展机制[56]。非酒精性脂肪性肝病(NAFLD)近年来发病率不断增高，危害人类健康。Flavell 等人发现炎症小体 NLRP6 和 NLRP3 参与非酒精性脂肪性肝炎(NASH)的发病，而肠道菌群组成改变会促进 NASH 发展，加剧肝脏损伤和肝纤维化[57]；Yu 等人发现趋化因子受体 CXCR3 通过促进炎症、巨噬细胞聚集、脂肪酸合成以及自噬损伤在 NASH 的发病中起关键作用，而 CXCR3 拮抗剂可逆转 NASH 的发生[58]。这些研究揭示了保持微生态平衡在肝病治疗中的重要意义，表明对宿主与微生物群相互作用的操控调节将是肝病新疗法的重点，也为探索微生态干预治疗肝病提供了新靶点与新思路。目前，微生态制剂在肝病治疗中的应用研究也取得了重要进展。一项临床随机对照试验显示，服用益生菌(VSL#3) 6 个月能显著降低肝性脑病患者住院风险以及 Child-Pugh 和 MELD 评分[59]。类似的微生态制剂在治疗 NAFLD、急性肝衰竭的应用中也有报道[60–62]。

大量动物实验表明，微生态改变与肝癌等肿瘤的发生发展密切相关[63–65]，因此肠道微生态调节参与肿瘤治疗也成为研究热点。Iida 等人发现肠道微生态通过调节肿瘤微环境来控制肿瘤对治疗的反应，因此对肿瘤的最佳治疗必须要有完整的、可调节肿瘤微环境的肠道微生物群存在[66]；Darnaud 等人提出可通过肠道微生态靶向治疗阻止肝癌发展[67]；Viaud 等人指出肠道微生态调节环磷酰胺的抗肿瘤免疫作用[68]，表明肠道微生态有助于机体形成抗肿瘤的免疫反应。此外，人体微生态组成还会影响人体的自然癌症免疫监测以及肿瘤治疗诱导的免疫应答[69]。这些发现突显了人体微生态在肝癌等肿瘤治疗过程中的作用，为发展新型的癌症治疗方法指出方向。

2.6 人体微生态与肥胖、糖尿病、心血管病等代谢性疾病

研究表明肠道微生态与肥胖、糖尿病、心血管病等代谢性疾病有密切关系。

Blaser 等人发现生命早期的抗生素治疗会改变小鼠肠道微生态结构，并影响体内与糖类和脂质代谢相关基因的活性以及某些激素的水平，从而导致肥胖[70]；随后，他们对处在不同生长期的幼鼠给予青霉素，发现短时间、低剂量的抗生素即可对初生小鼠产生长久影响，改变微生物-宿主间代谢交互作用，导致小鼠在中年时肥胖[71]。这表明在生命早期的某个时间窗，肠道微生态有可能影响宿主体内一些代谢信号通路的形成。Ehrlich 等人对非肥胖和肥胖人群进行肠道菌群分析，发现两类人群的肠道菌群在基因和丰富程度上均存在显著差别，低基因丰富性的个体有更显著的肥胖特征：胰岛素抗性、脂代谢紊乱现象并伴有其他炎症表征；仅对少数几种细菌标记进行分析就可区分个体细菌丰富程度的高低[72]。因此，肠道菌群作为新的切入点为日益困扰现代人生活的肥胖问题提供了新的预防和诊疗思路，也是近来的研究热点之一。Cotillard 等人对肥胖和超重人士进行饮食诱导的体重干预研究，发现高纤维低脂饮食可提高肥胖者肠道菌群基因丰度，并改善与肥胖相关的临床表征[73]。

已有很多研究表明微生态失调与代谢紊乱和糖尿病相关，但肠道菌群与 II 型糖尿病间的关系有待进一步研究。Qin 等人发现Ⅱ型糖尿病患者肠道微生态存在中度失调：产丁酸菌减少、条件致病菌增多；然而该疾病并不是造成患者与健康人之间微生态结构差异的主导因素，其贡献度为第 5 位[74]。II 型糖尿病的发病由患者自身基因和环境间复杂的相互作用导致，其致病因素包括年龄、家族史、饮食、生活习惯和肥胖等。肠道微生态结构作为重要的环境因素，可用于 II 型糖尿病的预测诊断。基于宏基因组分析，学者们通过筛选得到了不同生物标记物，分别建立了适合中国人和欧洲人的 II 型糖尿病肠道菌群预测模型[74–75]。

冠心病是人类健康头号杀手，动脉粥样硬化是其主要的病理基础。血氧化三甲胺(TMAO)浓度的升高与动脉粥样硬化形成有关[76]，而人体内 TMAO 的形成过程并非由基因决定，而是依赖于肠道菌群作用[76–77]。Karlsson 等人发现动脉粥样硬化患者的肠道微生态发生明显改变[78]，提示调控肠道菌群可为预防和治疗心血管疾病提供新思路。目前，通过补充肠道有益菌预防和改善动脉粥样硬化的研究也有较多报道[79–82]。

2.7 人体微生态与精神类疾病

人体微生态结构的改变会影响激素水平和神经递质产生，调节外周和中枢神经系统，导致大脑功能发生变化，从而影响人的心理、情绪和行为[83]。研究表明，大脑与肠道微生态、肠道免疫间的异常调节很可能是许多精神类疾病的发病机理，包括焦虑、抑郁、神经退行性病变、精神分裂、自闭、情感障碍、强迫症、注意力缺陷多动症、神经性厌食症、发作性嗜睡病、应激障碍和慢性疲劳等[84–87]。

研究表明 α-突触核蛋白聚集体在脑细胞中堆积，破坏脑细胞间通讯，是帕金森病、多系统萎缩症和路易体痴呆等神经退行性病变的病理基础之一[88]。帕金森病患者普遍伴有胃肠道损伤及功能紊乱。Scheperjans 等人发现帕金森病患者肠道微生态中普氏菌属显著降低，且肠杆菌丰度与某些症状严重程度相关[89]，提示肠道微生态结构变化可能会影响肠道微生物与肠神经元的相互作用，导致 α-突触核蛋白在肠道神经系统中聚集[90–91]，增加

患病风险。自闭症患者的肠道菌群结构发生改变。与正常儿童相比，自闭症儿童肠道菌群多样性降低，肠道内普氏菌属、粪球菌属和韦荣球菌科显著降低；进一步研究发现自闭症儿童肠道菌群多样性和数量的降低均与自闭症程度显著相关[92–93]，提示通过调节肠道微生态治疗此类疾病的潜在可能性。目前使用益生菌辅助治疗儿童自闭症的临床研究已有相关报道[94–95]；而动物研究也表明自闭症小鼠在接受脆弱拟杆菌治疗后，不仅肠道微生物群恢复正常，自闭症行为也得到改善[96]。焦虑症是最常见的神经性疾病。动物实验表明，肠道感染和肠道炎症会影响脑源性神经营养因子表达，使大脑内影响神经功能的化学物质和蛋白质水平发生变化，导致焦虑行为产生；而给予益生菌(长双歧杆菌)治疗可降低肠道神经元兴奋性，这一变化作为信号被传导至中枢神经系统后激活迷走神经调节机制，消除焦虑行为[97–98]，暗示了微生态治疗用于治疗人类焦虑症的前景。

2.8 人体微生态与其他疾病

肠道微生态失衡与肠道外免疫疾病有关，肠道菌群紊乱可导致全身免疫系统过度活跃[99]，导致自身免疫疾病发生。Li 等人研究发现原发性胆汁性肝硬化(PBC)患者肠道微生态明显改变，酸杆菌、毛型杆菌、拟杆菌等有益菌减少，变形菌、肠杆菌、奈瑟氏菌等条件致病菌增多；而某些条件致病菌的代谢产物会影响患者代谢、免疫和肝功能，与 PBC 的发生相关[100]。人体内出现某些与细菌产物类似的分子，可能是自身免疫性疾病发生的原因之一。例如，具有人类白细胞抗原 HLA-B27 的人群中约 90%患有强直性脊髓炎。Ebringer 研究发现肺炎克雷伯菌菌体成分与某些人的 HLA-B27 十分相似，免疫系统误将 HLA-B27 作为有害菌攻击导致自身抗体水平上升，引起多种自身免疫疾病[101]。Scher 等人发现新发类风湿性关节炎患者肠道菌群中普氏菌数量显著增多，动物实验显示由普氏菌主导的肠道微生态结构能提高化学药物诱导结肠炎的敏感性，从而更易激活机体对炎症的免疫应答反应[102]，提示普氏菌在类风湿性关节炎发病中具有潜在作用。

3 展望

当前，大量基础实验数据的积累正在促使人体微生态与健康和疾病相关研究从量变进入质变，研究重点开始从菌群结构功能变化的表象揭示，向菌群之间、菌群与人体相互作用等更高维度发展，并注重对微生态在发育、疾病和药物应用中的作用与机制研究。后续研究中，应加强方法学研究，建立标准化的人体微生态研究方法，标准的模式动物模型；建立中国人的肠道微生态研究计划和中国人群重大疾病患者肠道菌群研究的临床标本库和数据库；促进肠道微生态研究向功能基因组学研究方向发展；开发具有自主知识产权、具有医疗和保健价值的肠道微生态调节剂等。

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The links among disease, health and human microbiota

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Abstract: The human microbiota is the ecological community of commensal, the aggregate of symbiotic and pathogenic microorganisms that resides on or within any of several human tissues and biofluids. Human microbiota can be considered as a new 'organ' due to its important role in maintaining good health. The interaction mechanisms between microbe and host are comprehensive and complex. The dysbiosis of human microbiota is closely related to the process from diseases occurrence to development. Here we outlined the importance of microbiota study and reviewed the current researches worldwide on the links among disease, health and human microbiota.

Keywords: human microbiota, health, disease

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