

# 抗牙本质敏感的研究进展

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## 摘要

牙本质敏感是一种普遍的口腔疾病, 发病概率逐年上升, 温度、机械或化学刺激会使牙齿产生的短暂而尖锐的疼痛症状, 影响着人们的饮食与生活质量, 还或许因长期敏感造成排斥口腔清洁行为, 进而引起龋齿、牙髓炎、牙周炎等一系列口腔疾病, 寻找高效的抗牙本质敏感治疗举措, 重建牙体生理功能至正常水平, 成为口腔医学领域的关键研究方向, 本文将研究其发病机制、诊断方式及相应治疗策略, 回顾近年来在抗牙本质敏感上的研究进展, 随后对其未来研究方向进行总结与展望。

## 关键词

牙本质敏感, 发病机制, 治疗策略, 研究进展

# Research Progress on the Treatment of Dentin Hypersensitivity

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## Abstract

Dentin hypersensitivity is a common oral disease with an increasing incidence year by year. It is

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characterized by short and sharp pain in the teeth triggered by thermal, mechanical, or chemical stimuli. This condition affects people's diet and quality of life, and may even lead to avoidance of oral hygiene practices due to long-term sensitivity. Consequently, it can further cause a series of oral diseases such as dental caries, pulpitis, and periodontitis. Therefore, finding effective therapeutic measures for dentin hypersensitivity and restoring the physiological functions of teeth to a normal level have become key research directions in the field of stomatology. This article will explore its pathogenesis, diagnostic methods, and corresponding treatment strategies, review the research progress in anti-dentin hypersensitivity in recent years, and then summarize and prospect its future research directions.

## Keywords

Dentin Hypersensitivity, Pathogenesis, Treatment Strategies, Research Progress

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## 1. 引言

伴随现代生活方式的改变和人们口腔健康意识的进一步提高,牙本质敏感已成为全球范围内高发性口腔疾病,由流行病学调查结果显示,有42%的成年群体遭受它的困扰[1],鉴于牙本质小管产生暴露,患者在进食冷热的食物、碰到酸甜的物质或刷牙之际,常常出现短暂且尖锐的疼痛,这种疼痛不能归结为任何牙齿的缺陷或病症[2] [3]。即使牙本质敏感不是那种致命性疾病,然而其高发特性和治疗难度大,始终是口腔医学领域亟待处理的难题[4] [5],口腔医学技术不断改进且实现突破,研究者在牙本质敏感的发病机制、诊断途径以及治疗办法等方面均得到了阶段性进展,更多脱敏方案正陆续应用到缓解牙本质敏感患者的牙痛表现上,目前的相关研究还是有诸多争议与空白,临床治疗的成效也参差不齐,本文要围绕抗牙本质敏感方面的研究进展做综述,期望能为后续科研工作以及临床应用提供理论支撑和实践范例。

## 2. 牙本质敏感的发病机制

牙本质是一种生物矿化硬组织,其内部含有大量牙本质小管,这些小管呈现出紧密排列的形态[6],在正常的生理状态下,矿化的牙釉质将牙本质与复杂的口腔环境有效隔离[7],但当频繁摄入酸性食物、长期采用错误的口腔卫生清洁方式、存在功能障碍习惯时,牙釉质可能被破坏,牙龈也可能出现退缩,进而导致牙本质层和其中的牙本质小管暴露,外界环境中的温度刺激、化学刺激与机械刺激,能够直接作用于牙本质小管,进而诱发敏感症状[8]。解释牙本质敏感疼痛的三种理论已被学术界提出,分为神经学说、牙本质纤维传导理论和流体动力学说[9] [10],神经学说聚焦于外周神经末梢被直接激活的现象,牙本质纤维传导学说聚焦于成牙本质细胞在敏感机制中的关键功能,流体动力学说核心观点聚焦于牙本质小管内液体流动产生的效应。

### 2.1. 神经学说

牙本质里分布着神经末梢,当外界刺激直接施加给这些神经末梢的时候,会带动神经冲动的生成,该冲动进一步传导抵达中枢神经系统后,就会出现疼痛的感觉,从解剖学角度去看,牙髓里的神经纤维可延展至牙本质内层,甚至一部分可延伸至牙本质小管的中1/3范围。当牙本质暴露这一情况出现,外界

诸如温度、化学或机械的刺激等, 会直接刺激此类神经末梢, 致使神经冲动的产生[11], 当牙齿碰到过冷和过热的食物的时候, 牙本质里面的神经末梢受刺激, 把信号传导进大脑, 让人产生疼痛体验, 并非所有牙本质的区域都有神经分布, 尤其就外层牙本质而言, 这导致该学说无法完整阐释牙本质敏感引发的疼痛现象[12], 即便牙本质当中存在神经分布, 但鉴于敏感的外层牙本质缺少神经的支配, 让神经学说在说明牙本质敏感疼痛机制方面存在一定局限。

## 2.2. 牙本质纤维传导学说

牙本质小管里面存在着成牙本质细胞突, 其能起到传导痛觉的功效, 成牙本质细胞突从牙髓伸展进入牙本质小管里, 当外界刺激施加时, 成牙本质细胞突出现形变或位置的移动, 此类变化可借助细胞间隙连接等结构传递给牙髓神经, 从而引起疼痛[13]。当牙齿承受机械性的摩擦刺激之际, 牙本质小管里面的成牙本质细胞突会被干扰, 这种状态的变动会进一步传至牙髓神经, 最终引起痛觉产生, 然而该学说也碰到若干挑战, 成牙本质细胞突是否能跟神经纤维一样有效地传导痛觉信号, 而其传导机制的具体细节还是不明确[8] [14]。

## 2.3. 流体动力学说

现有研究主要基于获得公认的流体动力学说[15], 该学说的核心主张为: 牙本质小管内含液体, 牙本质外层直接接触外界环境后, 外界刺激能够引起小管中液体流动[16], 该流体运动可触发牙本质小管神经及髓腔表浅部神经感受器的反应, 从而形成疼痛反馈, 牙齿接触冷刺激物时, 受温度变化驱动, 牙本质小管中的液体开始流动, 流动的液体会对神经末梢形成刺激, 最终引起疼痛感, 从微观范畴的角度看, 牙本质小管的直径极其微小, 液体在那里流动会引起压力变化, 随后刺激神经末梢[17]。后续的研究表明, 进入牙本质小管的液体运动加剧会激活牙本质-牙髓连接处的 C 纤维和 A $\delta$  纤维, 基于此激活参与调控牙齿疼痛的相关信号通路[18] [19], 初级传入神经元内热敏与机械敏受体, 以及参与疼痛信号传导的电压门控离子通道的表达[20], 这也进一步支持了流体动力学说, 诸多研究表明, 经由堵塞牙本质小管以减少管中液体流动, 可以显著缓解牙本质敏感症状[21]-[23], 这也为该学说的合理性提供了极为重要的支撑证据。

## 3. 牙本质敏感的诊断

为了确定适当和有效的治疗, 对牙本质敏感做出正确的诊断是至关重要的。许多口腔疾病会出现与牙本质敏感相似的症状, 只有当体征和症状不符合任何其他口腔疾病或疾病的标准时, 才能做出牙本质敏感的诊断[8] [24]。

国际上普遍认可的牙本质敏感诊断标准, 核心要素为“短而尖锐的疼痛”和“牙本质暴露” [2] [3], 在中华口腔医学会发布的《牙本质敏感的诊断和防治指南(2019 修订版)》里, 同样是明确说明了[25], 诊断牙本质敏感的关键判定依据是: 患者碰到温度、化学、机械等刺激后产生短时疼痛, 同时呈现牙本质暴露的体征。若要准确诊断牙本质敏感, 需要充分时间收集患者的主诉和现病史, 然后做全面的口腔临床检查, 还需借助辅助检查去排除症状相仿的其他的口腔疾病, 通常采用的诊断工具主要有冷、热测试仪、牙周探针、X 光片、咬合评估等[26], 要实现对牙本质敏感的明确诊断, 得有完整的病史采集以及细致的临床检查, 这两项工作是诊断过程中的关键基础。

在临床实际应用中, 常见的需鉴别的牙体疾病及其关键鉴别要点如下: (1) 龋病: 探针可探及龋洞, 刺激入洞时可感到疼痛, 刺激去除后疼痛可消失[27]; (2) 牙隐裂: 裂纹常在咬合面的窝沟点隙或磨牙尖端, 可用探针或在牙科显微镜下发现裂纹, 疼痛一般为咬合痛[28]; (3) 牙髓炎: 牙髓活力测试异常, 疼痛往往不随刺激去除而消失, 且可有自发痛或夜间痛表现[29]。

## 4. 牙本质敏感的治疗

### 4.1. 牙本质小管闭塞

传统牙本质小管封闭材料里, 氟化物作为代表, 氟化物可在牙本质表面生成氟磷灰石晶体, 靠沉积作用来堵塞小管口[30]-[32], 临床研究证实, 含氟亚锡牙膏连续用上 10 天之后, 可实现降低牙本质敏感程度的效果[33], 这类材料存有封闭层薄、易被唾液溶化掉的不足, 难以契合长期抗敏感要求[34] [35], 因此, 需要研发出新的抗敏剂以解决这一缺陷。

生物活性玻璃、纳米羟基磷灰石等新型材料被研究领域密切关注着, 逐渐成为该领域的热门研究方向[36], 生物活性玻璃一旦与牙本质接触后, 可放出钙离子、磷酸根离子等活性离子, 这些离子可在牙本质小管内生成羟基磷灰石晶体, 以此种途径实现牙本质小管的深度封闭[37] [38]。纳米羟基磷灰石因为其主要成分和天然牙体组织较为相仿[39], 具备渗透至牙本质小管的特点, 造就了紧密的晶体层面, 可以使其封闭效果更为持久[38] [40] [41], 含有纳米羟基磷灰石(10% nHAP 和 15% nHAP)的牙膏用 14 天后能有效封闭牙本质小管[42]。

### 4.2. 神经脱敏

钾盐类药物是现阶段临床应用较为广泛的神经脱敏药物[43], 其主要的作用机制是依靠钾离子的跨膜运输, 影响到神经纤维的细胞膜电位, 若钾离子进入神经细胞的里面, 可使细胞膜呈现超极化现象, 增加神经纤维的兴奋临界值, 由此减少神经冲动的生成[44] [45], 常见的钾盐类药剂里有硝酸钾、氯化钾等, 这些药物大量添加到了牙膏、漱口水等口腔护理产品里[46], 对牙本质敏感不同治疗途径有效性的系统归纳和随访分析说明, 使用硝酸钾脱敏剂可大幅降低对冷热刺激的敏感水平[47]。

钙通道在神经冲动的产生与传导方面起关键作用, 钙通道阻滞剂可以专一性地跟钙通道蛋白相黏合, 凭借阻止钙离子流入, 能进一步抑制神经递质往外释放, 同时还对神经冲动的传导起到阻断效应[48] [49], 在进行牙本质敏感治疗期间, 钙通道阻滞剂可以缓解牙髓神经对刺激的反应[50], 但因其药物递送和作用持久方面存在问题, 目前在临床实际应用中还未广泛普及, 依旧处于科研攻关的关键阶段。

### 4.3. 激光治疗

有研究成果显示, 不管采用低功率激光还是高功率激光, 都可以有效治疗牙本质敏感状况[51]-[53], 相关实验表明, 低功率激光可能借助抑制牙髓神经的兴奋性发挥功效[54]; 高功率激光可诱导牙本质小管闭合, 从而发挥治疗作用, 以减轻牙本质敏感症的表现[55], 就牙本质敏感的治疗范畴而言, 目前临床主要采用以下几种激光: Nd:YAG 激光、Er:YAG 激光等[56]。

脱敏治疗中常用的 Nd:YAG 激光, 运用激光辐照诱导牙本质表面光热效应, 能引发牙本质表面瞬时熔融与结晶转变, 最终实现牙本质小管的封闭, 改善牙本质小管的封闭性以降低通透性[57]。研究发现波长 1064 nm, 频率 10 Hz, 功率 1 W 的 Nd:YAG 激光在体外具有优越的牙本质小管闭塞作用, 它在治疗牙本质敏感中的应用克服了传统牙本质敏感治疗方法的缺点, 但是由于酸性饮食和刷牙会导致沉淀物和表面涂层的持续消除, 因此需要反复应用以实现持续缓解疼痛[58]。有研究评估了 Nd:YAG 激光对牙本质敏感前牙牙髓血部分氧饱和度的影响, 得出结论, 在脱敏参数的范围内, Nd:YAG 激光产生的热量虽会扩散至牙髓, 但这种热量并不会对牙髓造成不可逆的损伤[59]。

Er:YAG 激光可跟水及羟基磷灰石起反应, 通过阻塞牙本质小管达成牙齿脱敏[60], 采用 0.25 W、0.5 W 的 Er:YAG 激光治疗对降低牙本质敏感度具有显著效果[61], 多项实验证实, Nd:YAG 激光的脱敏治疗效果明显胜过 Er:YAG 激光[62], 主要归因于 Nd:YAG 激光的光热转化更充分, 能实现牙本质表层更理想的熔融状态, 从而更充分地实现牙本质小管的闭合; Er:YAG 激光能量被水分子与羟基磷灰石接收, 实

施脱敏治疗期间发热不明显, 安全性方面更胜一筹。

4.4. 生物治疗

在牙本质敏感症治疗相关的研究里, 生物材料成为重点开展创新的对象, 生物治疗依靠其独特的修复与再矿化的长处, 为临床治疗提供了不一样的思路, 跟传统的治疗方法相比, 生物治疗更聚焦于从生物学层面改善牙体组织的微环境, 推动组织自身修复, 拥有精准、长效等潜在好处。

聚电解质-钙复合物前体(PCCP)矿化脱敏剂在体外和体内实验中都表现出牙本质小管的深度闭塞(超过 200  $\mu\text{m}$ )和脱矿牙本质基质的快速再矿化[63]。基于表没食子儿茶-3-没食子酸酯包封的纳米羟基磷灰石/介孔二氧化硅纳米颗粒(EGCG@nHAp@MSN)可致密封闭牙本质表面小管结构, 减弱牙本质渗透效应, 兼具突出的耐酸蚀与耐磨耗能力, EGCG@nHAp@MSN 展现出 EGCG 与钙磷的缓释特性, 显著阻碍牙本质表面变异链球菌生物膜的形成过程及其生长[64]。Ziji Ling [65]等建立了由寡肽刺激牙本质基质蛋白 1、三氧化矿物聚集体和琼脂糖水凝胶仿生矿化模型组成的仿生矿化模型(DMP-1@MTA@AHBMM), 在脱矿质的牙本质表面上使用 DMP-1@MTA@AHBMM 闭塞了牙本质小管, 可在牙本质表面诱导出含氟化羟基磷灰石晶体的类釉质组织, 形成的釉质样再生体硬度超过脱矿牙本质。Bailei Li 等[66]提出了一种基于  $\beta$ -壳寡糖移植衍生物(CAD)的具有显著生物安全性和持久治疗价值的新型疗法, CAD 恢复牙本质小管中的氨基多糖保护膜, 显著促进钙和磷离子沉积和骨合成代谢, 并调节唾液中的免疫球蛋白和血浆中的细胞炎症因子水平, 暴露的牙本质小管被深度超过 70  $\mu\text{m}$  的再矿化羟基磷灰石封闭。与空白组相比, CAD 组在 2 周内 Sprague-Dawley 大鼠磨牙牙本质的骨密度增加了 10.96%, 骨小梁厚度增加到约 0.03  $\mu\text{m}$ 。尽管生物治疗在牙本质敏感领域已取得一定进展, 但目前大多处于基础实验研究阶段, 仍面临诸多挑战。

4.5. 临床治疗路径

临床上对牙本质敏感的患者, 基于病因及敏感的严重程度, 可进行个性化的阶梯式治疗如图 1 所示。

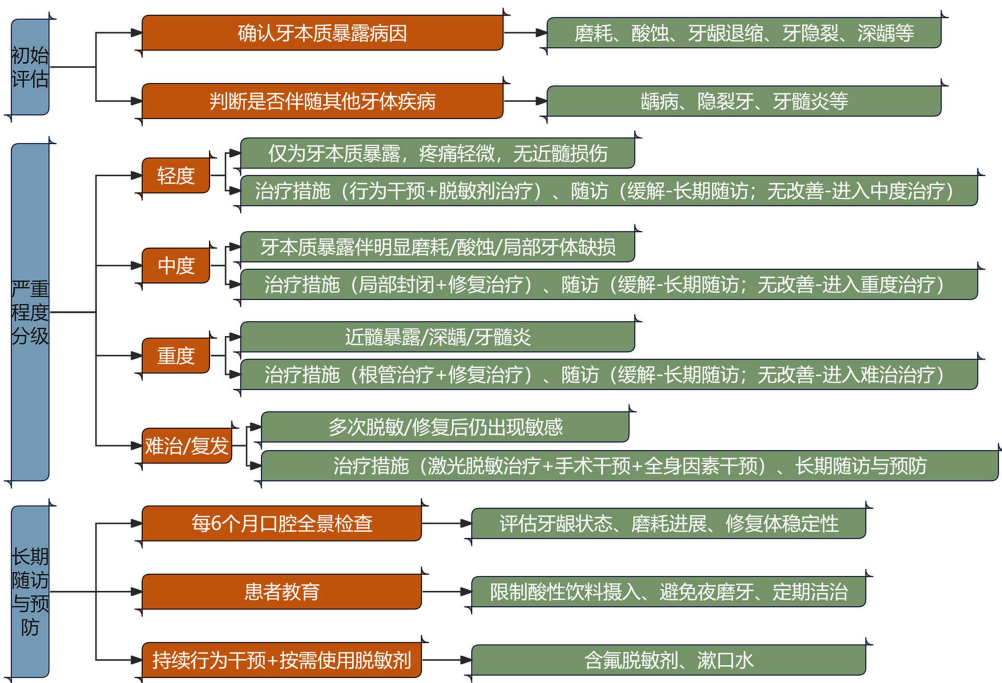


Figure 1. Clinical management of dentin hypersensitivity  
图 1. 牙本质敏感的临床治疗

## 5. 总结与展望

抗牙本质敏感研究在机制探索、材料开发以及治疗技术上有显著推进,但现今的研究依然存在局限性,部分材料长期的临床效果与安全性有待证实,对牙本质敏感发病机制的认识还没有完全搞明白,不管不同治疗手段的协同应用策略,或是个体化治疗方案的制定,目前均需进一步补充与完备,未来抗牙本质敏感研究还会进一步深入展开探索。

从材料研发这一相关方面,进一步提高材料性能水平,提高材料跟牙本质在生物相容性、结合紧密程度以及长期稳定性方面的表现,开发带有智能响应属性的材料,让其可根据口腔环境的改变动态调整所起到的作用。已有研究报道 pH 刺激响应型聚合物能智能控制药物释放[67],且纳米羟基磷灰石在口腔环境中具有调控优势,使设计 pH 响应性载氟纳米羟基磷灰石复合材料用于抗敏和防龋一体化治疗成为可能。

在进行机制探究方面,采用多组学技术、单细胞测序等先进技术手段,透彻阐明牙本质敏感在细胞与分子水平的调节机制,留意疾病从发生到发展阶段的动态变动,为精准治疗给予必要的理论支撑。多组学与 AI 融合已在生命科学中形成成熟框架,如 AI 在基因组学、单细胞分析中的应用[68],构建“多组学-单细胞”平台,系统描绘牙本质敏感的细胞与分子网络可成为未来研究方向。

就治疗技术的角度看,推进多模态治疗的扩充,让激光治疗、生物材料修复跟生物活性因子调控配合起来,摸索非侵入性或微创治疗的新招法,支持临床研究开展,建立标准化的疗效考核体系,同时结合患者存在的个体差异,采用大数据、人工智能技术设计个性化治疗方案,由此明显增强抗牙本质敏感治疗的临床成效。目前抗牙本质敏感的联合多种疗法尚缺乏大样本、长期随访的临床数据,因此未来开展多中心、长期随访的随机对照临床试验具有重要意义。

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