

近视前期的危险因素识别及干预策略研究进展

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摘要

近视前期作为近视防控的关键窗口期, 以远视储备快速消耗、眼轴增长加速为核心特征, 其防控对于降低近视发生风险具有重要临床意义。近年来, 随着儿童青少年近视患病率持续上升, 近视前期的流行病学特征、危险因素及干预措施受到广泛关注。现有研究表明, 遗传因素、近距离用眼负荷增加、户外活动不足及屈光储备下降等与近视前期发生密切相关。行为与环境干预、光学及药物干预均被证实可在一定程度上延缓近视发生, 其中周边离焦光学设计、低浓度阿托品及新型光学疗法在近视前期人群中的应用显示出较为稳定的防控效果。近年来, 多模式联合干预及个体化干预策略逐渐成为研究热点, 但其长期疗效、安全性及适用人群仍有待高质量研究进一步明确。本文系统综述近视前期的流行病学特征、主要危险因素及最新干预研究进展, 为儿童青少年近视的早期防控提供循证依据。

关键词

近视前期, 危险因素, 干预措施

Risk Factors Identification and Intervention Strategies for Premyopia

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Abstract

Premyopia represents a critical transitional stage preceding the onset of myopia and is character-

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ized by accelerated axial elongation and progressive depletion of hyperopic reserve. Early identification and intervention during this period may reduce the risk of myopia development. With the rising prevalence of myopia among children and adolescents, increasing attention has been directed toward the epidemiology, associated risk factors, and preventive interventions for premyopia. Current evidence suggests that genetic susceptibility, increased near-work exposure, insufficient outdoor activity, and reduced hyperopic reserve are key factors associated with the development of premyopia. A range of interventions, including behavioral and environmental modifications, optical approaches, and pharmacologic treatments, have demonstrated varying degrees of efficacy in delaying myopia onset. In particular, peripheral defocus-based optical designs, low-dose atropine, and emerging optical therapies have shown consistent preventive effects in premyopic populations. Recently, multimodal and individualized intervention strategies have been proposed; however, robust evidence regarding their long-term efficacy, safety, and optimal target populations remains limited. This review summarizes current evidence on the epidemiology, major risk factors, and recent advances in intervention strategies for premyopia, providing an evidence-based framework for early myopia prevention in children and adolescents.

Keywords

Premyopia, Risk Factors, Intervention Strategies

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1. 引言

近视已成为全球范围内最重要的视觉公共卫生问题之一，其患病率在儿童和青少年中持续攀升[1]。多项系统评价及预测模型显示，在缺乏有效干预的情况下，中国儿童近视患病率至 2050 年可能超过 60%，然而在学龄期早期通过系统性防控措施，可显著降低近视患病率[2]。学龄期近视的早期控制可有效降低成年后高度近视并发症风险，这凸显了针对近视前期进行公共卫生干预的必要性[3]。值得关注的是，近视的发生并非突然事件，其之前往往经历一个以远视储备快速消耗和眼轴(Axial Length, AL)增长加速为特征的“近视前期”阶段[4]。

近年来，国际近视研究所(IMI)及我国相关专家共识均将近视前期视为近视防控的关键窗口期，特别是在 5~15 岁儿童群体中，在该阶段开展早期识别和干预，有助于延缓甚至阻断近视的发生[5] [6]。目前我国相关专家共识将近视前期的定义为：睫状肌麻痹验光检测的远视储备量小于或等于同年龄段儿童远视储备量的下限(6 岁为+0.75D, 7~8 岁为+0.50D, 9~10 岁为+0.25D)，且等效球镜度数(spherical equivalence refraction, SER)为 $\leq +0.75D$ 且 $> -0.50D$ [6]。这一年龄相关的屈光储备阈值为临床早期识别提供了可操作标准，其病理特征包括眼轴加速增长、视网膜离焦模式改变以及脉络膜厚度变薄等生物标志物异常[7]。近年来，国内外围绕近视前期的流行病学特征、危险因素及干预措施开展了大量研究，本文拟就近视前期的危险因素及干预研究进展进行综述，以期临床防控提供参考。

2. 近视前期的流行病学特征和筛查指标

流行病学研究显示，在不同地区人群中，近视前期的分布亦存在差异[8]。东亚地区 6~7 岁儿童近视前期患病率高达 35.7%，显著高于欧洲同龄人群的 18.3% [9]，欧洲近视患病率低于亚洲，城市儿童近视比例高于农村儿童。这种地域差异或许与教育压力、户外活动时间等环境因素密切相关[10]。近视前期在学龄儿童中呈现明显的年龄特异性分布，6~7 岁儿童中约 30%存在屈光储备不足，而这一比例在 10~12

岁群体中上升至 45% [4]。我国 5 至 18 岁儿童和青少年的远视储备显著不足, 近视发病年龄越来越小, 尤其是接近青春期的女性和更幼的儿童[11]。这种年龄相关性变化提示屈光储备的消耗速度与生长发育阶段密切相关, 近视早期干预黄金期主要集中在小学低年级阶段。

单独未矫正远视力(Uncorrected Distance Visual Acuity, UDVA)对近视筛查有一定效果。在非散瞳条件下, AL/角膜曲率半径(Axial Length/Corneal Radius ratio, AL/CR)、SER、UDVA 等指标组合分析可精准实现近视前期筛查。中国研究构建并验证近视发生预测模型, 模型整合了散瞳 SER、既往 1 年 AL 变化、性别、父母近视史四大预测因子, 并开发了开源在线风险计算器, 为儿童近视早期预警与干预提供了精准工具[12]。研究证明散瞳后分析的近视患病率低于非散瞳, 这提示在筛查近视前期时散瞳 SER 这一指标更精准。根据两步法筛查策略: “先筛查近视, 再针对非近视人群筛查近视前期”的模式, 可提高近视的筛查效率与精准度[4]。

3. 近视前期的危险因素

3.1. 遗传因素

大量研究证实, 遗传因素在近视前期的发展中扮演着重要角色, 父母近视史与儿童近视前期显著相关。父母一方近视时, 儿童近视患病率为 14.9%; 当父母双方均近视时, 儿童近视发生风险可提高至 40% 以上, 这一现象反映出父母近视史与儿童近视易感性之间存在密切的正相关关系[13]。双生子研究进一步量化了遗传贡献, 发现近视前期的遗传度可达 60%~80%之间, 表明遗传变异对屈光发育的调控具有主导作用[14]。年龄特异性屈光储备数据进一步显示, 3 岁儿童正常远视储备应为 2.64D, 若低于此阈值则预示近视风险上升[15]。

3.2. 屈光发育特征

晶状体和角膜屈光力和 AL 是影响屈光状态的主要因素[16], 在屈光学特征方面, 远视储备不足被认为是近视前期最重要的预测指标[17]。儿童早期屈光状态越接近正视, 其未来发生近视的风险越高。研究发现, SER 年变化和眼轴增长速度与初始屈光状态的密切相关。眼轴增长和屈光度数加速变化往往始于轻度远视阶段, 尤其在 SER 接近+1.00D 甚至更低时最为明显[18]。通过年龄特异性远视储备分析显示, 6~9 岁儿童每年平均消耗 +0.50D 屈光储备, 其中基线 SER \leq +0.19D 的儿童在 10 岁前发生近视的风险增加 6.28 倍, 6~7 岁时 SER \leq +0.19D、AL \geq 23.19 mm 及至少一位家长近视(SER \geq -3.00D)是儿童 10 岁前发生近视的主要高危因素[9]。

3.3. 行为与环境危险因素

除遗传易感性外, 行为与环境因素在近视前期的发生中具有明确的可干预性, 近距离用眼时间过长、阅读距离过近、电子屏幕暴露增加以及户外活动不足, 是近视前期最主要的环境危险因素[19]。中国 6~17 岁学生近视率达 55.5%, 其中高学业压力群体发病率更高[20]。在近距离用眼行为中, 阅读距离小于 30 厘米、单次用眼时间超过 30~40 分钟, 或每日累计近距离用眼时间超过 2 小时, 均会加速远视储备消耗并增加近视风险, 这种行为可能通过视网膜离焦机制来影响近视进展[5]。相反, 户外活动时间与近视发生呈显著负相关, 每日增加 40 分钟户外活动可使近视发生风险降低约 20%~25% [21] [22]。因此, 近年来的防控策略更强调“用眼模式”的调整, 这些发现为行为干预提供了坚实的循证基础。

4. 近视前期的干预策略

4.1. 行为与环境干预

行为与环境干预是近视前期防控中最基础的且成本效益高的措施, 也是目前证据相对充分的干预方

式。政府和学校实施的长期干预措施增加户外时间,可能有助于减少儿童近视的发生[23]。每日增加 40 分钟户外活动,3 年内近视发病率相对减少 23% [19]。部分随机对照研究显示,在学校层面增加课间户外活动时间,可在一定程度上延缓屈光状态向近视方向发展,尽管其具体机制尚未完全明确,可能与多巴胺介导的视网膜信号通路有关[21]。光照强度阈值研究通过可穿戴智能设备监测光照强度及户外活动时间,发现每日持续 15 分钟、光照强度不低于 2000 lux 的户外暴露能有效进行近视干预[22]。为实现最佳预防效果建议每天至少 2 小时的户外活动时间,当然更长或更频繁的户外活动可能更有效,但需注意紫外线防护措施。在临床实践中,合理控制近距离用眼时间、改善阅读姿势及用眼环境,仍被视为近视前期管理的重要内容。虽然部分用眼行为为指导缺乏高等级循证证据,但其安全性高、可操作性强,具有较好的推广价值。

4.2. 药物干预

低浓度阿托品作为目前近视前期防控最有效的药物干预手段,其应用证据主要来自两类研究:一类是以近视发生率为结局的预防试验,另一类是以 SER、AI 增长为结局的控制试验,部分研究结果存在差异。欧洲一研究证实 0.01% 的阿托品可显著降低近视转化率并抑制 AL 增长[24]。AMPP 研究纳入 5 至 12 岁阶段的近视前期儿童,连续 1 年每晚使用 0.01% 阿托品滴眼液可使近视进展减少 53%,但眼轴变化没有明显异常[25]。Yam 等的随机对照试验表明,0.05% 阿托品在 2 年随访中可使近视前期儿童的近视累积发病率降低 24.6%,其效果优于 0.01% 组及安慰剂组,而 0.01% 组与安慰剂组差异无统计学意义,提示存在一定的浓度依赖性疗效[26]。这一发现与 Lee 的 meta 分析结果部分吻合,但提示不良反应并非严格呈剂量依赖性,个体差异可能起重要作用[27]。综合现有证据,0.05% 阿托品在延缓近视发生方面可能优于 0.01% 浓度,在低远视储备(SER < 0.75D)的儿童最为明显,且不良反应多在可耐受范围内,但潜在反弹效应仍需关注[28]。研究亦提示 0.01% 阿托品疗效存在种族差异,东亚儿童获益相对有限,临床上可根据近视进展速度进行个体化给药,如快速进展者考虑 0.05% 每日一次,进展缓慢者采用 0.01% 或隔日用药[29]。此外,DIMS 镜片联合 0.01% 阿托品较单用镜片可进一步减少约 62% 的眼轴增长[29],提示联合干预对于高风险人群如伴有间歇性外斜视的儿童中可能具有优势。

目前针对近视前期儿童使用低浓度阿托品的长期随访数据仍较有限。阿托品的不良反应具有剂量依赖性,使用浓度越高,其不良反应越明显[30]。低浓度阿托品滴眼液短期应用后可能出现因瞳孔散大导致的畏光、因睫状肌麻痹导致的调节能力降低等副作用,症状多处于轻度至中度,多数儿童可耐受,但长期使用对眼表健康、眼压调节、晶状体浑浊及视网膜功能的潜在影响尚未明确[26]。尤其对于低龄儿童(5~6 岁),眼部组织仍处于发育阶段,药物代谢能力与青少年存在差异,长期用药的安全性更需谨慎评估。目前随访发现 0.01% 阿托品滴眼液的全身及眼部不良反应症状最轻微且发生率最低。在短暂使用后阿托品滴眼液停药后会出现一定的反弹效应,且与使用浓度和年龄相关,尤其在高浓度或更低年龄阶段的儿童中反弹效应更明显[31]。这些研究进一步指出阿托品滴眼液维持持续疗效所需要的使用时间、何时可以停止治疗将反弹效果降至最低是未来主要的研究方向。

4.3. 光学干预

光学干预在近视前期儿童及青少年中的防控作用已在多项随机对照试验中得到验证,其核心机制是在保证中央视力清晰的同时,于视网膜周边区域诱导相对近视性离焦,从而抑制眼轴过度增长。基于该理论,临床常用的干预方式包括周边离焦设计框架眼镜、点扩散技术镜片、角膜塑形镜及离焦软性接触镜等。多区正向光学离焦(Defocus Incorporated Multiple Segments, DIMS)镜片作为代表性干预手段,在 2 年随机对照试验中显示其可较单光镜片使近视进展减缓 52%,眼轴增长减少 62%,且 21.5% 的受试者未

出现近视进展[32]。进一步随访至3年发现,持续佩戴者干预效果稳定,而由单光镜片转为DIMS的儿童亦获得显著控制效果,提示该干预具有持续性及一定可逆性[33]。不同光学设计的干预效果存在差异。高度非球面镜片(Highly Aspherical Lenslets, HALs)在8~13岁华裔儿童的5年随访中显示明显降低高度近视发生率,且佩戴时间越长,控制效果越显著[34],尤其在佩戴后前三个月内[35],但在近视前期阶段的控制效果还需进一步研究。此外,DOT镜片在CYPRESS试验中于12个月内使屈光进展减少74%,进一步验证了周边离焦策略在近视前期防控中的潜力[36]。现有研究还提示,近视前期的光学干预效果与个体基线周边屈光状态密切相关。DIMS镜片在基线存在远视性周边离焦的儿童中控制效果更为显著,提示个体化光学设计可能进一步优化干预结局[37]。角膜塑形镜(Orthokeratology, OK)作为夜间佩戴的代表性光学干预手段,其延缓眼轴增长的效果已得到多项随机对照试验验证。Meta分析显示,OK镜在6个月时可实现64%的近视控制率,但对于中度近视患者人群效果更佳[38],但对于近视前期阶段的控制效果尚未明确。上述结果进一步提示,在近视前期防控中,结合个体视网膜周边屈光特征进行定制化光学干预具有重要意义。

4.4. 重复低强度红光治疗(Repeated Low-Level Red-Light, RLRL)

RLRL疗法作为一种新兴的、非侵入性光学干预手段,短期临床数据显示其近视控制率可达60%以上[39],最新研究报告指出,RLRL治疗后儿童眼部血流量增加、眼轴增长趋于稳定,可能与改善巩膜缺氧状态有关,但其具体作用机制仍有待进一步阐明[40]。我国一项为期1年的随机对照试验纳入139名近视前期儿童且至少一方父母近视,结果显示RLRL组近视发作率较对照组降低54.1%,提示该疗法在高风险近视前期人群中具有一定预防作用[41]。针对近视前期的儿童,重复强化中心凹红光治疗可有效降低了近视前期儿童的近视发生率、AL,并提高了SER和脉络膜厚度[42]。RLRL能有效降低近视前期儿童的患病率,但RLRL对已近视儿童的影响更强[43]。RLRL长期预防近视的疗效及安全性仍缺乏充分证据,尤其是其对视网膜细胞潜在生物学效应的影响,尚需开展更长期、严格设计的研究加以评估[44]。

近期系统性回顾的结果表明,RLRL疗法未发现导致不可逆损伤的视功能丧失或眼部结构改变[45]。RLRL治疗后最常见的眼部症状是后像。在数十项已发表的研究中,仅有两项报告了同一女孩出现可逆性视功能下降及黄斑中心凹椭圆体带与嵌合体带中断的单一病例,该病例在停用四个月完全康复[46]。虽然不良事件的发生率极低,但是仍需在整个治疗过程中进行细致监督,以确保RLRL的安全实施。适当措施包括:在治疗开始前及每次常规检查时通过眼底照相和OCT记录视网膜状况,追踪视力变化并记录任何后像的持续时间。

此外,Xiong等[47]进行了为期两年的研究,观察到第二年RLRL-SVS组、SVS-SVS组AL增长分别为 (0.42 ± 0.20) mm、 (0.28 ± 0.14) mm, SER进展分别为 (-0.91 ± 0.48) D、 (-0.54 ± 0.39) D,表明停止RLRL照射治疗1年后的患者中观察到明显的反弹效应,突然停止RLRL照射治疗是不合适的,逐渐减少治疗时间可能有助于减少反弹效应。

4.5. 联合治疗

联合治疗模式通过多靶点协同作用展现出突破性潜力。药物与光学手段两者联用可产生叠加效应。临床研究显示,DIMS镜片联合0.01%阿托品较单用镜片可额外减少约62%的眼轴增长[39],而角膜塑形镜联合0.01%阿托品其控制效果明显优于单一干预[48];RLRL治疗与各种光学手段联合使用,在儿童和青少年近视防控中展现出明显优势[49][50]。总体来看,药物与光学手段的联合应用在抑制眼轴增长方面表现出一定优势。此外,Eppenberger等提出,多模态干预策略(如增加户外活动联合低浓度阿托品及光学矫正)可能更适用于近视进展风险较高的人群,包括伴有间歇性外斜视的儿童[51],然而,联合干预的长

期疗效、安全性及适用人群仍有待进一步研究明确。

5. 结论

在近视发生前,眼球的生长速度和屈光度的变化最快,因此研究人员一直在寻找近视前期干预治疗[44]。尽管现有的干预措施在一定程度上显示出疗效,但其临床应用仍面临多方面挑战,其中依从性问题尤为突出。光学干预如角膜塑形镜需长期规范佩戴,但青少年常因舒适度及护理复杂性而中断使用;低浓度阿托品虽被证实具有一定效果,但瞳孔散大及调节相关不良反应仍可能影响持续用药,长期安全性亦有待进一步评估[52]。行为干预如增加户外活动受社会和教育环境制约明显,东亚地区儿童日均户外活动时间普遍不足1 h,反映出有防控措施在实施层面的现实局限[9]。

在此背景下,多模式联合干预被认为是提升防控效果的重要方向。个体化干预理念逐渐引入近视防控领域,通过将光学矫正、药物治疗及行为干预进行动态组合,可能在一定程度上弥补单一措施的不足[53]。Mohr提出的行为干预技术模型为此提供了参考,其核心在于借助智能设备监测用眼行为并调整干预强度[54]。然而,此类技术依赖较高的医疗和信息化资源,可能在不同地区间进一步加剧防控手段的可及性差异[55]。

此外,个体生物学差异亦是近视防控技术转化中的关键问题。角膜生物力学特性[56]和屈光储备的年龄特异性变化[57]使固定参数的干预方案在不同人群中的效果存在明显差异。例如,DIMS镜片在8~10岁儿童中可使眼轴增长减少约62%,而在12岁以上青少年中效果明显减弱[32]。低浓度阿托品同样存在个体代谢差异,较高浓度虽疗效更优,但可能增加调节障碍风险[39]。上述异质性提示,近视防控策略需具备动态调整能力,而目前基于群体数据构建的模型尚难精准反映个体生理变化[58]。因此,未来近视防控应从单一干预向覆盖筛查、风险评估、干预及随访的全流程管理模式转变,通过机制研究、临床验证与技术创新协同推进,实现由群体防控向精准干预的过渡,以响应儿童近视早期阻断的公共卫生策略[7]。

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