

发展性阅读障碍与小脑异常： 小脑的功能和两者的因果关系*

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摘要 发展性阅读障碍(下文简称为“阅读障碍”)不仅会影响个人的终身发展,还会对社会造成沉重的经济负担,深入探讨相关的神经机制,对实现阅读障碍的早期预测和干预十分重要。以往关于阅读障碍神经机制的模型多集中于大脑,近些年的研究发现,阅读障碍也与小脑异常有关,但到目前为止我们仍不清楚两者的关系。通过总结最新的研究进展,我们发现小脑在阅读障碍中可能发挥着多种功能,且小脑异常与阅读障碍可能互为因果。在此基础之上,我们提出了“阅读中小脑与大脑的功能映射假说”,旨在从一个全新的角度揭示小脑与阅读的关系,以及两者与大脑的关系。相关内容对全面揭示阅读障碍的神经机制,以及小脑在高级认知加工中的作用,具有重要的启示意义。

关键词 发展性阅读障碍, 小脑异常, 阅读的神经机制, 功能映射假说, 因果关系

分类号 B845

发展性阅读障碍(以下简称为“阅读障碍”)是一种常见的学习障碍,特指儿童或成人智力正常,有足够的学习动机和受教育机会,但依然表现出明显的阅读困难现象(Peterson & Pennington, 2012)。阅读障碍在人群中的比例较高,约为 5%~17% (Shaywitz & Shaywitz, 2005)。阅读障碍不仅仅影响个人的学业水平,还会进一步降低自我效能感,增强学业焦虑,提高辍学率,并最终对社会造成严重的经济负担(刘丽, 何茵, 2018; Livingston et al., 2018)。

阅读依赖于一定的脑结构基础,而阅读障碍也与脑结构的发展异常有关。深入探讨与阅读过程相关的神经基础,将有利于揭示阅读障碍的形

成机制,进而为阅读障碍的早期预测和干预提供必要的理论指导。以往关于阅读障碍神经机制的模型多集中于大脑:研究发现阅读障碍与颞顶区域及枕颞区域的激活减弱或者灰质体积减少有关(Linkersdörfer et al., 2012; Maisog et al., 2008; Norton et al., 2015; Richlan et al., 2013)。但近些年的研究发现,大脑异常并不是阅读障碍唯一的神经基础,阅读障碍者的小脑也存在结构或功能活动的异常(Stoodley & Stein, 2013; 彭聆, 杨静, 2004; 赵婧 等, 2015)。

针对小脑异常与阅读障碍的关系, Nicolson 等人(2001)提出了小脑缺陷假说,此假说认为小脑结构或功能的异常可能是导致阅读障碍的原因(Mariën et al., 2014; Nicolson et al., 2001),且小脑可以通过运动相关的能力(比如运动/自动化加工或者发音)来影响阅读(Alvarez & Fiez, 2018; Ben-Yehudah & Fiez, 2008; Mariën et al., 2014; Nicolson & Fawcett, 2011; Nicolson et al., 2001; Stoodley & Stein, 2013)。但这种单一功能或单一因果关系的视角已很难解释现有的研究发现,同时该假说较少涉及阅读障碍中小脑与大脑的关系。本综述从

收稿日期: 2021-04-15

* 国家自然科学基金项目(32100846, 32000793); 中国博士后科学基金资助项目(2020M682846)、认知神经科学与学习国家重点实验室开放课题基金资助项目(CNLYB2005)、广东省科技计划项目(2019A050510048); 深港脑科学创新研究项目(2021SHIBS0003); 深圳大学青年教师科研启动项目(QNJS0304)。

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小脑功能以及因果关系两个方面,总结了小脑异常与发展性阅读障碍的研究进展,并在此基础上提出了关于两者以及两者与大脑关系的新假说。

1 小脑在阅读中的作用

1.1 小脑异常与自动化加工缺陷或语言加工缺陷的关系

1.1.1 与运动/自动化加工或发音相关的小脑异常

运动序列学习任务经常被用于考察小脑与运动/自动化加工的关系,此任务可稳定地诱发小脑的激活(Danelli et al., 2013; Hung et al., 2019; Nicolson et al., 1999; Penhune & Steele, 2012)。序列学习任务要求被试根据屏幕上依次呈现的刺激进行相应的按键反应。序列学习的早期和晚期所涉及的认知过程略有不同。在学习的早期,认知负荷较大,刺激和按键匹配的学习过程依赖工作记忆的参与。随着被试成功习得固定刺激序列的呈现规律,被试的按键反应会变快,正确率会变高,行为逐渐自动化(Ashby et al., 2010; Janacsek et al., 2019)。

阅读障碍者小脑异常与运动/自动化加工相关的脑成像证据,主要来自于正常组和障碍组在运动序列学习任务中的对比研究。Nicolson 等人(1999)要求被试在神经影像数据采集之前学习一串数字序列,在神经影像采集过程中根据所学的数字序列进行按键反应,此时的任务表现更多地反映了个体的自动化加工能力。研究发现,阅读障碍者在右侧小脑 VI 区的激活显著弱于正常读者。Menghini 等人(2016)利用此任务区分了在学习早期和晚期,阅读障碍者小脑的神经活动与序列学习的关系。在序列学习的早期,正常组和障碍组在小脑处的神经活动并未表现出显著的组间差异,但在学习的晚期,阅读障碍组在双侧小脑 VI 区的激活显著强于正常组。Yang 等人(2013)采用与 Menghini 等人(2016)类似的任务,在左侧小脑 IV 区发现了类似的效应。考虑到序列学习晚期被试的反应逐渐自动化,而阅读障碍者的小脑在此阶段的神经活动出现异常,说明此异常可能与自动化加工有关。

如前所述,小脑缺陷假说认为小脑也可以通过发音影响阅读。目前,虽然有研究发现小脑内存在负责发音的区域,比如右侧小脑 VI 区的内侧(Ashida et al., 2019; Frings et al., 2006),但并没有

研究直接探讨这一区域与阅读障碍的关系。总体上,小脑缺陷假说得到了一些研究的支持。这些研究发现阅读障碍者的小脑异常可能与运动/自动化加工能力的异常有关。

1.1.2 与语言加工相关的小脑异常

值得注意的是,运动/自动化加工似乎并不足以解释小脑在阅读中的作用,因为即使在较少涉及这些加工过程的词汇阅读中,阅读障碍者的小脑也表现出神经活动的异常。Richlan 等人(2010)比较了阅读障碍者与正常读者在加工真词或假词时的神经活动。相对而言,真词更多地涉及语义加工,而假词更多地依赖语音加工(Cattinelli et al., 2013)。结果显示,阅读障碍者无论是在加工真词还是假词,其右侧小脑 VI 区的激活都显著强于正常读者(Richlan et al., 2010)。这样的结果说明右侧小脑 VI 区可能参与语音和语义加工。这一推论也得到其他研究的支持。例如,在偏重语音加工的押韵判断任务中,阅读障碍者在右侧小脑 VI 区的激活显著弱于正常读者(Meng et al., 2016; Raschle et al., 2012)。在偏重语义加工的(不发声的)动词产出任务中,正常读者和阅读障碍者在右侧小脑 V 区、VI 区以及右侧 VII 区的激活也存在显著差异(Baillieux et al., 2009)。

以上研究多是关注小脑异常与单一功能的关系,比如与运动(运动/自动化加工)或与语言加工(语音或语义加工)的关系。仅仅考察单一类型的功能,可能难以全面揭示小脑异常与阅读障碍的内在联系。基于此,Cullum 等人(2019)采用了多任务范式,考察了不同阅读能力的被试在单字默读任务中和快速命名任务中的神经活动。其中,前者更多涉及语言加工,包含正字法的识别及语音和语义的提取,而后者则更多涉及自动化加工。研究发现,在被动注视的单字默读任务中,阅读障碍者小脑内多个区域的激活显著弱于正常读者;但在快速命名任务中,两组人在小脑处的激活并不存在显著差异(Cullum et al., 2019)。此研究表明阅读障碍者的小脑异常更可能与语言加工方面的缺陷有关。值得注意的是,在快速命名任务中两组人不存在显著差异可能与感兴趣区的选择有关。

以上研究结果表明,小脑与阅读的关系并不单一。小脑异常既可以通过运动相关的能力(运动/自动化加工)来影响阅读,也可以通过语言加工相

关的过程(语义或语音加工)来影响阅读。

1.2 亚类型的研究

比较阅读障碍的亚类型是考察小脑功能的另外一种可行的方式。根据个体在不同认知能力上的表现, 阅读障碍可被分成不同的亚类型, 如由语音缺陷、视觉正字法加工缺陷或自动化加工缺陷导致的阅读障碍。如果某一亚类型与小脑异常有关, 则说明小脑异常可能与该类型相关的认知缺陷有关。Norton 等人(2014)将被试分为由语音加工缺陷导致的阅读障碍者和由快速命名加工缺陷导致的阅读障碍者。结果发现, 后者的右侧小脑 IV 区在押韵任务下的激活显著弱于正常读者, 但前者的小脑并未出现异常。个体在快速命名中的表现经常被用于衡量自动化加工的能力(Raberger & Wimmer, 2003)。这些结果说明, 小脑缺陷与阅读障碍的关系更可能与自动化加工缺陷有关。与此结果一致, Jednorog 等人(2014)根据阅读障碍者在多种认知测验上的表现, 将其聚类成不同的亚类型。结果发现在信息加工速度(自动化加工能力)及注意转换上存在异常的阅读障碍组的左侧小脑的灰质体积显著小于其他组(Jednorog et al., 2014)。

也有研究发现小脑不同方面的异常与不同类型的阅读障碍有关。Fernandez 等人(2013)将被试分为语音解码能力存在缺陷和阅读流畅性存在缺陷的阅读障碍者。结果发现, 后者整个小脑的灰质体积与个体在真词测验上的得分呈显著正相关, 而前者的左侧小脑 VII/VIII 区的灰质体积与其在真词测验以及流畅性测验上的表现呈显著正相关(Fernandez et al., 2013)。

亚类型的研究发现不同方面的小脑异常可能与不同认知能力的异常有关, 这说明小脑异常与阅读障碍的关系可能是多维度的。总之, 无论是组间对比还是基于亚类型的研究均支持小脑可能通过多种方式影响阅读, 除了通过运动/自动化加工或者发音外, 小脑异常也可能通过语言加工相关的能力(比如语音或者语义加工)影响阅读, 即小脑在阅读中可能发挥着多功能作用。

2 阅读障碍与小脑异常的因果关系

目前大部分研究多集中探讨小脑的功能, 较少有研究探讨小脑异常与阅读障碍的因果关系。简单来说, 小脑异常与阅读障碍可能存在着 4 种

关系: (1)两者无直接的因果关系。(2)小脑异常是阅读障碍的原因。(3)小脑异常是阅读障碍者阅读能力低导致的结果。(4)互为因果关系, 即小脑异常即可能导致阅读障碍, 也可能被阅读障碍者的阅读能力所影响。

2.1 小脑异常与阅读障碍直接相关

需要指出的是, 除了阅读能力存在缺陷, 阅读障碍者在视知觉加工、注意等方面也可能存在缺陷(Ahissar et al., 2006; Jednorog et al., 2014; Qian & Bi, 2015; Zhao et al., 2014)。这也引发了一个问题, 阅读障碍者的小脑异常是与阅读障碍直接相关还是与其共患的其他障碍有关。对比阅读障碍与其他障碍的神经机制, 是澄清此问题的一个很好的方式(Bishop, 2002)。

以往研究发现, 阅读障碍和多动症具有较高的共患率。大约 15~40% 的阅读障碍者也患有多动症, 而大约 10~30% 的多动症患者也有阅读障碍(Holborow & Berry, 1986; Shaywitz et al., 1994), 而且多动症患者的小脑也存在异常(Valera et al., 2007)。行为上的研究发现, 阅读障碍者在平衡能力测验(用于测试小脑功能)、词汇命名测验以及非词重复测验上的表现均显著差于正常读者和多动症患者(Kasselimis et al., 2008)。神经影像的研究发现, 相比于阅读障碍者, 非阅读障碍者(多动症+正常读者)的小脑呈现出更大的右偏趋势。但相比于非多动症患者, 多动症患者的小脑并不存在偏侧化的异常(Kibby et al., 2008), 这说明阅读障碍者的小脑异常可能与多动症无关。

除了与多动症进行比较, 也有研究对比了阅读障碍与自闭症(Bennett & Lagopoulos, 2015)及书写障碍(Richards et al., 2015)的神经基础。研究发现自闭症患者左侧小脑 VIII 区、IX 区, 以及右侧小脑 Crus I 区的灰质体积显著小于正常读者(Stoodley, 2014), 而阅读障碍者在双侧小脑 VI 区的灰质体积显著小于正常读者(McGrath & Stoodley, 2019; Stoodley, 2014)。在任务态(字母产出任务)及静息态下, 阅读障碍者左侧枕颞区域与双侧小脑 VI 区的功能连接显著强于正常读者, 但在书写障碍组中并未发现大、小脑的连接异常(Richards et al., 2015)。以上的研究表明, 阅读障碍的神经机制可能与自闭症以及书写障碍的神经机制存在差异。这些结果进一步表明, 小脑内可能存在特异于阅读障碍的神经异常。

2.2 小脑异常可能是阅读障碍的原因

如果小脑异常与阅读障碍直接相关,那么小脑异常究竟是阅读障碍的原因还是结果呢?阅读障碍的研究一直存在一个困境:只有等到儿童具有一定的阅读能力之后才能判断其是否患有阅读障碍,但此时已错过了最佳的预测和干预时机(Ozernov-Palchik & Gaab, 2016a, 2016b)。

为了解决此问题,研究者将研究对象提前至学龄前,并试图通过比较有无家庭阅读障碍史的学龄前儿童(Hosseini et al., 2013; Im et al., 2015; Raschle et al., 2011; Raschle et al., 2012; Vandermosten et al., 2015),或患阅读障碍高、低风险的儿童(Saygin et al., 2013; Specht et al., 2009),来探讨阅读障碍与小脑异常的因果关系。如果阅读障碍者的小脑异常出现在学龄前,则说明此异常可能是阅读障碍的原因(李何慧等, 2017)。Raschle等人(2012)比较了有无家庭阅读障碍史的学龄前儿童的神经活动。结果发现有家庭阅读障碍史的儿童右侧小脑VI区在语音任务中的激活弱于无家庭阅读障碍史的儿童。Specht等人(2009)发现,阅读障碍高风险儿童右侧小脑IX区在正字法任务中的激活显著强于低风险儿童(Specht et al., 2009)。这两个研究的结果表明小脑异常可能出现在学龄前,即小脑异常可能是阅读障碍的原因。

干预研究也可以用于探讨小脑异常与阅读障碍的因果关系。如果小脑异常是阅读障碍的原因,那么改善小脑功能应该能改善阅读能力。干预小脑最常用是“阅读障碍-协调障碍-注意障碍干预项目(DDAT)”。此干预项目包含多种提高小脑功能的训练任务。研究发现,经过训练后,阅读障碍者的小脑功能及阅读能力均得到改善(Reynolds et al., 2003)。但值得注意的是,此研究筛选出的阅读障碍者中有65%的个体存在小脑缺陷。而对于其他的阅读障碍群体,研究者并未发现DDAT可以显著改善阅读能力(Rack et al., 2007)。

从学龄前开始的纵向研究也可以回答因果问题。在结构水平上,Bruckert等人(2019)探讨了儿童6岁时小脑内白质纤维的完整性与其8岁时阅读能力的关系。研究发现左侧小脑脚白质纤维的平均各向异性可以显著预测儿童在8岁时的口语阅读能力(Bruckert et al., 2019)。控制社会经济地位、非言语智商以及性别后,此结果依然显著(Bruckert et al., 2019)。在功能水平上,Li, Kepinska

等人(2021)关注了儿童在学龄早期时小脑的神经活动与当前以及一年后阅读能力的关系。结果发现右侧小脑VII区的激活强度与一年后的阅读能力呈显著正相关。这些研究结果表明,早期小脑的结构以及功能特性可能影响未来的阅读发展。换言之,早期小脑的异常可能会导致阅读障碍。

总之,基于学龄前儿童的研究、干预研究以及纵向研究都存在支持小脑异常可能是导致阅读障碍原因的证据。

2.3 小脑异常可能是阅读障碍的结果

小脑缺陷假说一个备受质疑的地方是:为什么一个被认为负责运动的脑区会导致阅读障碍(Stoodley & Stein, 2013)。Zeffiro和Eden(2001)认为,如果小脑缺陷是导致阅读障碍的原因,那么阅读障碍者的首要症状应该是运动功能的异常(如四肢不协调或运动技能差),且小脑异常者应该存在阅读障碍。但事实是,阅读障碍的常见症状是语音加工缺陷。而且也并不是所有小脑存在异常的个体都有阅读障碍。基于此,小脑异常更可能是阅读障碍的结果,而非导致阅读障碍的原因(Zeffiro & Eden, 2001)。

现有的研究也为上述推论提供了线索。研究发现阅读障碍者双侧小脑后外侧区域的神经活动,及其与大脑的功能连接强度显著强于正常读者(Feng et al., 2017; Linkersdörfer et al., 2012; Hancock et al., 2017; Greeley et al., 2020)。例如,Feng等人(2017)在字形加工任务中发现,儿童双侧小脑VI区的前部在阅读障碍者中的激活显著强于正常读者。Ashburn等人(2020)以双侧小脑VI区以及Crus I区为感兴趣区,发现阅读障碍者右侧小脑Crus I区与阅读网络的功能连接强于正常读者。在结构连接水平上,研究发现中脑脚纤维束的完整性与阅读能力呈显著负相关(Travis et al., 2015),而阅读障碍者小脑脚与左侧颞顶区域白质纤维的FA值显著高于正常读者(Fernandez et al., 2016)。激活、功能连接以及白质纤维完整性的增强,有可能是为了补偿大脑内功能或者结构存在缺陷的区域,即小脑异常可能是阅读障碍者阅读能力低所导致的结果。

总体而言,现有的结果表明,小脑异常与阅读障碍直接相关。但研究间得到了不一致的结论:有研究支持小脑异常是导致阅读障碍原因,也有研究发现小脑异常可能是阅读障碍的结果。这种

不一致可能与研究范式或者所招募的被试有关,也可能说明小脑异常与阅读障碍互为因果(符合第四种关系类型),即早期小脑异常可能会影响未来的阅读能力,同时阅读能力的异常也可能在一定程度上调节小脑的神经活动。

3 进展总结及研究展望

发展性阅读障碍在人群中的比例较高,而且已对个人和社会造成了较多负面影响。准确了解相关的认知神经机制,将有利于对其进行尽早识别和干预。以往关于阅读障碍神经机制的模型多集中于大脑,近些年,越来越多的研究发现阅读障碍也与小脑异常有关(Ashburn et al., 2020; D'Mello & Gabrieli, 2018; 彭聘龄, 杨静, 2004)。但目前并不十分清楚两者的关系。小脑缺陷假说认为小脑异常是导致阅读障碍的原因,且小脑通过运动/自动化加工或者发音影响阅读(Nicolson et al., 2001)。但近期的研究结果与此假说并不完全一致:近些年的研究发现阅读障碍者的小脑可以通过高级的语言加工过程(比如语音或者语义加工)影响阅读(D'Mello et al., 2020; Gatti, van Vugt, & Vecchi, 2020; Li et al., in preprint; Meng et al., 2016);而且小脑异常也可能是阅读障碍者阅读能力低所诱发的补偿机制,即阅读障碍的结果(Feng et al., 2017; Hancock et al., 2017)。

一方面,这些不一致可能与研究间采用的实验范式或者招募的样本群体有关;另一方面,这也可能说明小脑异常与阅读障碍存在“多种关系”:在功能方面,小脑在阅读中的作用既可能与运动/自动化加工过程有关,也可能与高级的语言加工过程有关;在相互作用方面,小脑异常与阅读障碍可能互为因果。小脑内的功能分区支持了这种“多种关系”。研究发现小脑内存在多个参与阅读的区域,不同的区域参与不同的认知加工过程。例如,双侧小脑 I 区到 V 区可能与感知运动相关(Stoodley & Schmahmann, 2009; Stoodley et al., 2012),右侧小脑 VI 区可能与语音加工相关(Frings et al., 2006; Li et al., in preprint; Meng et al., 2016; Tan et al., 2005),右侧小脑 VII 区可能与语义加工相关等(D'Mello et al., 2020; D'Mello et al., 2017; Gatti, van Vugt, & Vecchi, 2020; Gatti, Vecchi, & Mazzoni, 2020),而且这些脑区的异常都与阅读障碍有关(Norton et al., 2014; Stoodley &

Stein, 2013; Yang et al., 2013)。小脑内的功能分区可能进一步调节了阅读障碍与小脑异常的因果关系。研究发现,小脑内负责运动加工的区域在阅读障碍者中的神经活动强于正常读者,这种增强可能是阅读障碍的补偿机制,属于阅读障碍者阅读能力低所导致的结果(Li et al., 2020);而小脑内负责语音或者语义加工的区域在学龄前的神经活动已经出现异常(Raschle et al., 2012),说明小脑内语言区的异常可能是导致阅读障碍的原因。多功能以及互为因果关系有利于解释前人研究结果的不一致,突出小脑在阅读中的重要作用,并为理解小脑与阅读关系提供了一个重要视角。

但这也引发了进一步的问题,小脑内存在多个参与阅读的区域,大脑内也存在多个参与阅读的区域,那么小脑与大脑内的阅读区域之间是什么关系呢?来自小脑功能分区的研究发现,小脑内与大脑内功能相似的区域可能存在同步性的神经活动,比如小脑内的运动区与大脑内负责运动的区域存在显著的功能连接,而小脑后外侧负责高级认知加工的区域与大脑的额叶和颞顶区域等存在显著的功能连接(Buckner et al., 2011; Guell et al., 2018; King et al., 2019; Seitzman et al., 2020)。基于此,我们提出了小脑与大脑在阅读中的“功能映射假说”。“功能映射假说”是指小脑映射了大脑的功能,小脑内可以定位出与大脑内阅读区具有相似功能的区域,大、小脑的功能对应区协同合作以参与阅读。具体而言,1)与大脑类似,小脑内也存在多个参与阅读的区域,不同区域负责阅读过程中所涉及的不同认知过程。2)这些区域与大脑内负责同一认知过程的区域在空间分布上呈对侧对应的关系。如前所述,大脑内的阅读区主要在左半球,包含左侧的额下/额中回、颞顶区域和枕颞区域(Cao, 2016; Dehaene, 2009; Li, Zhang, & Ding, 2021);而小脑内的阅读区主要在右半球,包含右侧小脑 VI 区和 VIII 区(Li, Kepinska, et al., 2021; Stoodley & Schmahmann, 2018; Stoodley & Stein, 2013)。3)小脑内阅读区的功能实现依赖于与大脑的合作。这主要是因为小脑的皮层细胞构筑较为单一(Ramnani, 2006),单一的结构往往对应着单一功能。因此,小脑很可能是通过与大脑的不同区域进行连接,来参与不同的认知过程(Gatti, Vecchi, & Mazzoni, 2020; King et al., 2019; Schmahmann et al., 2019)。4)阅读障碍

与大、小脑合作异常有关。比如, 现有较多研究发现, 阅读障碍者大脑和小脑的功能连接异于正常读者(Ashburn et al., 2020; Feng et al., 2017; Greeley et al., 2020)。

“功能映射假说”与以往研究者提出的“大脑-小脑的闭合回路模型”一致(Ramnani, 2006)。闭合回路模型指出大脑与小脑间存在信息传输的通路, 而且大脑皮层作为回路输入的主要来源, 也是回路输出的主要目标(Bostan & Strick, 2018; Doya, 2000; Ramnani, 2006)。大脑输出的信号经脑桥传递至对侧的小脑皮层; 小脑皮层将信号传递至小脑内的齿状核, 之后被传递至丘脑, 并返回至同一大脑皮层(Bostan et al., 2013)。通过病毒示踪技术, 研究者进一步发现小脑与大脑的信息传输通路是分区式的。例如, 大脑运动皮层(M1)与小脑前部负责运动的区域相互传递信息, 而大脑前额叶皮层(BA46)与小脑后外侧负责高级认知加工的区域相互传递信息(Buckner, 2013)。

未来的研究可以通过验证“功能映射”的相关假设来检验此假说。例如, 可以考察小脑内是否存在负责不同认知加工(比如负责形、音、义)的区域, 以及这些区域是否与大脑内负责相同功能的区域呈对侧对应关系。其次, 也可以考察小脑与大脑的合作关系, 即两者在结构或者功能水平的连接情况。如果映射关系成立, 未来的研究也可扩展映射假说的内容。比如, 可以深入探讨为什么小脑与大脑内各存在一套阅读系统? 再次, 可以关注小脑与大脑映射关系的对应方式, 是一一对应、多对一、还是一对多的关系。最后可以关注小脑与大脑究竟是如何协同合作的。

总体而言, 以往关于阅读障碍与小脑异常的研究或假说(比如小脑缺陷假说)更多的从单一功能或单一因果关系的视角思考小脑异常与阅读障碍的关系。在本综述中, 我们提出了小脑与大脑的“功能映射假说”。此假说从多功能和多因果关系的视角出发, 既解释了现有的研究发现, 也更系统地总结了小脑与阅读的关系, 对小脑参与其他高级认知加工的方式也具有重要的启示作用。但未来还需要更多的研究, 来检验“功能映射假说”是否成立, 以及探讨此假说的相关延伸问题, 以期全面揭示小脑参与词汇阅读的机制以及阅读中小脑与大脑的关系。

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Developmental dyslexia and cerebellar abnormalities: Multiple roles of the cerebellum and causal relationships between the two

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Abstract: Developmental dyslexia (hereinafter referred to as “dyslexia”) will not only affect the life-long development of individuals, but also impose a heavy economic burden on society. Digging into the relevant neural mechanisms contributes to the early prediction and intervention of dyslexia. Established models of the neural bases of dyslexia primarily focused on the cerebrum. In recent years, extensive studies have shown that dyslexia is also associated with cerebellar abnormalities. However, it remains unclear about the relationships between the two. By summarizing recent findings, we found that the cerebellum could play multiple roles in reading and a bi-directional causal relationship may explain the associations between cerebellar abnormalities and dyslexia. Based on these findings, we proposed the “cerebro-cerebellar mapping hypothesis of word reading”. This new framework aims to reveal the relationship between reading, the cerebellum, and the cerebrum from a new perspective. This review offers important insights into the neural mechanism of dyslexia and the role of the cerebellum in high-level cognitive processing.

Key words: developmental dyslexia, cerebellar abnormalities, the neural mechanisms of reading, mapping hypothesis, causal relationship