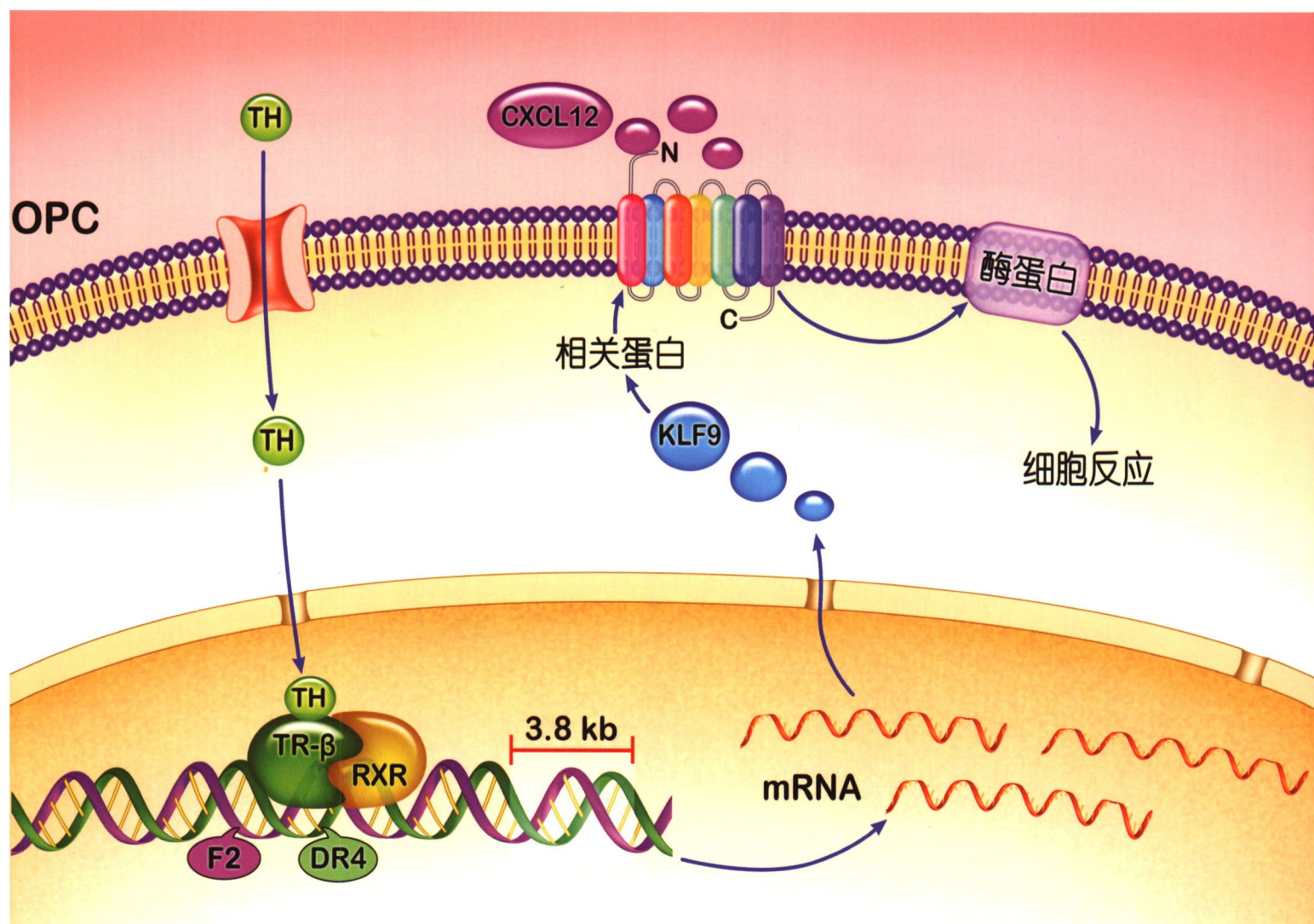




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封面故事:陆军军医大学姚忠祥教授课题组研究发现:甲状腺激素(thyroid hormone, TH)通过上调少突胶质前体细胞(oligodendrocyte precursor cells, OPCs)内kruppel样因子9(kruppel-like factor 9, KLF9)的转录水平而促进OPCs增殖分化为成熟少突胶质细胞(oligodendrocytes, OLs)。相关作用机制模式图如封面所示:TH和甲状腺激素 β 受体(thyroid hormone beta receptor, TR- β)参与调节KLF9基因的转录过程;KLF9基因序列从启动子到翻译区的长度是3.8 kb,包括倒置回文(inverted palindrome, F2)和同向重复序列4(direct repeat 4, DR4)的启动子序列;F2和DR4是甲状腺激素受体作用元件(thyroid hormone receptor elements, TREs),可以被TR- β 和视黄醇类X受体(retinoid X receptor, RXR)识别,RXR可以作为协同激活剂调节TR- β 与TREs的结合;TH、TR- β 、RXR和TREs组成的转录复合物激活了KLF9基因的转录,从而上调了KLF9的表达水平;KLF9可以促进相关蛋白分子的产生,进一步激活CXCL12/CXCR4信号通路;CXCL12/CXCR4信号通路活化后促进了OPCs向成熟OLs的增殖分化。“TH→KLF9→CXCL12/CXCR4→OPCs→OLs”这一信号通路阐明了TH促进OPCs增殖分化为成熟OLs的机制,也为TH促进髓鞘再生修复提供了新证据。该研究内容详见:Thyroid hormone potentially benefits multiple sclerosis via facilitating remyelination[J]. Molecular Neurobiology, 2016,53(7):4406-4416. DOI: 10.1007/s12035-015-9375-z。



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