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子宫平滑肌收缩相关机制

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摘　要

子宫收缩是子宫平滑肌在神经、体液与分子信号网络共同调控下产生的节律性舒缩活动，也是维系妊娠稳态、启动分娩、推动产后子宫复旧的关键生理环节。一旦宫缩功能出现异常，便会成为产科最常见的病理诱因，极易诱发早产、产程阻滞、胎儿窘迫、产后出血、复旧不全等严重并发症，显著升高母婴不良妊娠结局的风险。近年来，分子生物学、生物医学工程与临床医学的快速进步，让宫缩相关研究持续推进，研究视角也从以往的宏观临床观察，逐步深入到细胞、离子通道、基因与信号通路等微观层面。不过，当前研究仍存在不少短板：机制探讨较为零散、临床研究不够规范、基础成果向临床转化效率偏低、针对特殊人群的研究相对匮乏，很多问题还没有形成统一、清晰的认识。本文通过系统检索与梳理国内外近年来相关文献，分类总结现有研究进展，剖析当前研究体系存在的不足，并结合临床实际，阐明深化宫缩研究的必要性与应用价值，对未来发展方向进行展望，以期为宫缩相关疾病的机制探索、临床诊疗优化以及新型干预策略研发提供可靠的理论依据。

关键词

子宫收缩，子宫平滑肌，妊娠，缝隙连接，离子通道，激素

Mechanisms of Uterine Smooth Muscle Contraction

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Abstract

Uterine contraction refers to the rhythmic contraction and relaxation of uterine smooth muscle

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regulated jointly by the neural, humoral and molecular signaling networks. It serves as a pivotal physiological process for maintaining gestational homeostasis, initiating labor, and promoting postpartum uterine involution. Abnormal uterine contraction function is one of the most common pathological incentives in obstetrics, which can easily induce severe complications such as preterm birth, labor arrest, fetal distress, postpartum hemorrhage and subinvolution of uterus, and markedly increase the risk of adverse maternal and fetal pregnancy outcomes. In recent years, with the rapid advances in molecular biology, biomedical engineering and clinical medicine, researches on uterine contraction have been continuously promoted. The research perspective has gradually extended from previous macroscopic clinical observation to microscopic levels including cells, ion channels, genes and signaling pathways. Nevertheless, there still exist many deficiencies in current studies: fragmented exploration of mechanisms, non-standard clinical research, low transformation efficiency of basic research findings into clinical practice, and insufficient researches on special populations, and a unified and clear consensus has not been reached on many issues. By systematically retrieving and reviewing relevant domestic and foreign literature in recent years, this paper categorically summarizes the current research progress, analyzes the shortcomings of the existing research system, clarifies the necessity and application value of further research on uterine contraction combined with clinical practice, and prospects the future research directions, so as to provide a reliable theoretical basis for the mechanism exploration of uterine contraction-related diseases, optimization of clinical diagnosis and treatment, and research and development of novel intervention strategies.

Keywords

Uterine Contraction, Uterine Smooth Muscle, Pregnancy, Gap Junction, Ion Channel, Hormone

1. 引言

子宫是女性特有的肌性器官。人类子宫肌层具有独特的结构与收缩特性，无典型平滑肌分层、无明显传出神经支配，且含起搏样细胞参与电活动调控；围产期子宫由孕期静息状态向分娩期节律性收缩转化，依赖收缩相关蛋白上调与电生理重构，需间隙连接介导同步化、多离子通道协同调控，是多信号参与的复杂生理过程，诸多基础调控机制仍有待明确。子宫平滑肌细胞间通讯是宫缩同步化的关键，以 Cx43 为主的间隙连接介导细胞电与代谢偶联，是分娩发动的重要结构基础，其表达受多重信号精准调控。免疫–肌细胞交互成为宫缩调控新方向，现有研究虽证实巨噬细胞铁死亡可参与分娩启动，但未关联免疫信号与 Cx43 介导的经典胞间通讯通路，机制研究链条尚不完善。近 15 年，子宫收缩相关离子通道研究不断突破，除经典钙、钾通道外，T 型钙通道、Kv7、BKCa 等多种离子通道被证实参与宫缩调控，研究范畴也拓展至子宫血供、病理应激、炎症通路及早产机制等领域，机械敏感离子通道的发现更开辟了全新研究方向，但各类离子通道协同调控网络仍需进一步深入探究。本文系统综述子宫肌层收缩调控、细胞间通讯及离子通道作用的研究进展，剖析现有研究不足，为阐明子宫收缩分子机制、防治分娩相关并发症提供理论依据。

2. 子宫肌层收缩调控机制研究

人类子宫肌层并非典型的纵行与环行平滑肌分层结构，而是以肌束与纤维束有序排列为主；子宫肌细胞并非完全均一，还存在类似 Cajal 间质细胞的起搏样细胞，参与电活动调控。子宫收缩兼具时相性收

缩与强直性收缩特点，无明显传出神经支配，区别于胃肠道、膀胱等其他内脏平滑肌，属于独特的内脏平滑肌收缩模式[1]。子宫收缩是妊娠晚期与分娩期子宫肌层的核心功能，是完成胎儿娩出、胎盘剥离及产后止血的关键。妊娠子宫在围产期发生显著功能转变：孕期以静息、容纳、肥大为主，分娩期则转为节律性、协调性收缩，由静息态向收缩态的转化依赖收缩相关蛋白的上调与电生理重构[1]。子宫收缩的同步协调依赖间隙连接(以 Cx43 为主)，它不仅介导电信号传递形成电合胞体，还通过代谢偶联(如 IP_3 扩散)调控局部兴奋性，单纯动作电位传导速度较慢，难以实现全子宫同步动员，流体力学－牵张激活可能是器官水平同步收缩的重要补充机制。电生理层面，钙通道、钾通道(BKCa)、氯通道共同调控子宫肌细胞兴奋性。总体而言，子宫收缩是结构特化、电信号－机械信号耦合、多通道协同、间隙连接介导同步化的复杂生理过程，受母体与胎儿信号共同调控，其基础生理机制仍存在诸多争议与待解问题。

3. 子宫收缩相关机制

3.1. 平滑肌细胞之间介导的收缩机制

间隙连接介导的细胞间电偶联与代谢偶联，使分散的子宫平滑肌细胞形成功能合胞体，保证宫缩同步化。Tabb 等较早提出，细胞间偶联是子宫肌层生理功能实现的关键，直接影响子宫收缩效率与节律[2]。Sheldon 等进一步证实，间隙连接参与子宫平滑肌网络兴奋性调控，是子宫由静息向收缩转化的结构基础[3]。Cx43 是介导子宫平滑肌细胞通讯的主要间隙连接蛋白，其表达水平与宫缩能力密切相关。Döring 等通过基因敲除实验证实，小鼠子宫平滑肌 Cx43 缺失可直接导致分娩延迟，从动物模型证实 Cx43 为分娩发动必需蛋白[4]。Yang 系统总结了 Cx43 在子宫肌层的表达规律与调控通路[5]。Khanam 等发现大鼠妊娠期子宫肌层 P2X1 受体与 Cx43 表达呈动态变化，提示 Cx43 在妊娠不同阶段受多重信号调控[6]。免疫－肌细胞交互是近年宫缩调控领域的新方向。Song 等首次报道，发生铁死亡的 M2 型巨噬细胞可调控子宫平滑肌收缩并促进分娩启动，将免疫细胞程序性死亡与分娩发动直接关联，为理解分娩启动提供了免疫调控新视角[7]。尽管 Song 等在其文中对于免疫细胞铁死亡调控分娩的论述有了新的突破口，但显而易见的是，该研究忽视了巨噬细胞信号对 Cx43 表达与缝隙连接功能的直接调控问题，也未意识到关于免疫信号－缝隙连接－宫缩同步化通路的重要性。从而，在他的观点存在着机制链条不完整、未关联经典胞间通讯通路的问题。

3.2. 离子通道对子宫收缩的作用

近 15 年间，关于子宫收缩相关离子通道的研究在子宫平滑肌兴奋性与收缩调控方面的论述有了新的突破，不再局限于原有的经典钙通道与钾通道基础功能描述，开始出现新研究的角度与方向。如 Lee 等通过 T 型钙通道的角度，以新颖视角诠释了钙信号启动妊娠大鼠子宫平滑肌自发性节律收缩的重要性，并提出 T 型钙通道功能增强是妊娠状态下子宫收缩能力上升的关键离子基础之一[8]。Rosenfeld 等的研究提到，大电导钙激活钾通道 BKCa 与 cGMP 信号通路共同参与子宫血管平滑肌舒张调控，妊娠可显著改变其通道功能与信号耦联，打开了离子通道与血管舒张、子宫血供研究的广度[9]。McCallum 等提出 Kv7 通道直接参与小鼠与人妊娠子宫肌层收缩调控，可作为抑制子宫过度兴奋的重要靶点，进一步扩充了电压依赖性钾通道在分娩启动中作用的研究深度[10]。Senadheera 等进一步证实，妊娠大鼠子宫动脉收缩增强与 TRPC3、L 型及 T 型钙通道功能上调密切相关，将钙通道家族的协同作用从肌层收缩延伸至子宫血管调控，完善了离子通道在子宫活动中的整体网络认识[11]。Zhu 等研究发现，妊娠期缺氧通过升高活性氧，抑制类固醇激素对子宫动脉钙激活钾通道功能的上调，首次将微环境应激、氧化应激与离子通道功能重塑相联系，拓展了病理妊娠下离子通道异常的研究维度[12]。Lorca 等系统总结 BKCa 通道活性调控可直接改变子宫肌层收缩特性，明确该通道为宫缩相关疾病的重要干预靶点，为后续机制研究提供清晰

框架[13]。Wakle-Prabagaran 等揭示 BKCa 通道调控人子宫平滑肌细胞中 α2-巨球蛋白诱导的钙振荡，将离子通道与炎症相关信号通路结合，丰富了宫缩调控的信号网络[14]。

Ferreira 等发现催产素可通过抑制 Na^+激活钾通道 Slo2.1 调节子宫肌层兴奋性，首次建立催产素－钠激活钾通道－子宫收缩的直接调控通路，完善了催产素促宫缩的离子机制[15]。Bi 等在 2024 年提出子宫 Piezo1 机械敏感通道过表达可促进子宫肌层收缩与炎症相关早产，将机械敏感离子通道引入早产机制，开辟了机械信号－离子通道－炎症－宫缩的全新研究方向[16]。同年 Bao 等证实 BKCa 通道参与妊娠晚期小鼠自发性及脂多糖诱导的子宫收缩，进一步夯实 BKCa 通道在生理性与炎症性宫缩中均发挥关键作用的结论[17]。尽管 Bi 等在其文中对于 Piezo1 介导炎症相关早产的论述有了新的突破口，但显而易见的是，该研究忽视了 Piezo1 与其他钙、钾通道在子宫肌层的交互调控问题，也未意识到机械通道与内分泌信号(如雌激素、催产素)协同驱动分娩启动的重要性。因而其观点存在机制相对单一、未构建多信号整合网络的问题，仍需后续研究完善机械敏感通道与经典离子通道的对话机制。

3.3. 妊娠相关激素与微环境对子宫平滑肌的调控

子宫肌层并非单一靠细胞缝隙链接或离子通道产生收缩效应，也是激素信号网络与局部微环境因子共同调控的动态结果，二者通过受体介导、信号通路交联、细胞表型转换等机制，协同维持妊娠期子宫静息状态与分娩期收缩激活的平衡转换。

雌激素和孕激素是调控子宫收缩最关键的两类甾体激素，它们通过受体介导的基因组与非基因组效应，双向塑造子宫肌层的收缩特性。KÜÇÜKYURT 等人的研究就直接证明，足月和过期妊娠时，雌、孕激素水平的动态波动，会直接改变宫缩的强度与频率，算得上是启动分娩的关键内分泌信号[18]。孕激素一向被看作孕期里的“子宫镇静剂”，能直接压制收缩相关蛋白的表达、抑制细胞内钙信号激活，帮子宫维持松弛状态。Shynlova 团队也进一步证实，孕激素能阻断多条促收缩通路，它的浓度下降和受体功能转变，正是发动分娩的重要节点[19]。Mesiano 等人则提出，甾体激素其实是在调控肌层细胞在“收缩型”和“静息型”之间切换，从而精准控制宫缩，这一过程高度依赖激素受体在肌细胞上的特异性分布[20]。雌激素的作用刚好相反，更偏向促收缩。它可以上调催产素受体、前列腺素受体等关键分子，让子宫对各种促收缩信号更敏感。Gittens 等人就发现，内皮素对子宫的激活作用必须依赖雌激素，说明雌激素是很多局部因子起效的“基础条件”[21]。除了经典受体，Anamthathmakula 等人还发现，雌激素受体 α 的一个亚型 ERΔ7 在子宫肌层里特异表达，反而有助于维持孕期子宫安静，这也让雌激素调控宫缩的机制更复杂[22]。另外，雄激素也参与其中，Makieva 等人系统梳理了雄激素在妊娠和分娩中的角色，补上了甾体激素调控网络里的重要一环[23]。

多肽激素和局部微环境里的活性物质，相当于宫缩的直接“启动开关”，和甾体激素配合形成完整调控网。HCG 不只是维持黄体功能，Ticconi 等人发现，它还能直接作用于子宫肌层和胎膜，稳定子宫、抑制异常收缩，对降低早产风险有帮助[24]。Ambrus 与 Rao 发现人绒毛膜促性腺激素可调控妊娠子宫平滑肌间隙连接功能，提示妊娠相关激素直接参与细胞间通讯调节[25]。催产素、前列腺素、内皮素-1、缓激肽这些局部因子，都是很强的宫缩激活剂。Nosálová 等人在体外实验里直接验证，这几种物质都能引发人子宫肌层收缩，只是效果强弱和模式不一样[26]。Challis 团队则重点阐明了前列腺素在早产中的核心地位，指出前列腺素合成与释放异常，是早产发生的重要机制[27]。前列腺素不仅能直接激活收缩通路，还能促进宫颈成熟，双管齐下启动分娩。子宫局部微环境里的一些分子，比如维生素 D、褪黑素，也会通过信号通路影响肌层收缩。Thota 等人发现，维生素 D 能通过 NF-κB 通路调整人子宫肌细胞的收缩状态，抑制异常收缩，说明营养相关的微环境因子也在参与调控[28]。Abd-Allah 等人的动物实验则显示，褪黑素可以改变雌、孕激素受体的表达，进而影响大鼠子宫收缩[29]，这也给通过微环境干预子宫功能提

供了新思路。

4. 总结与展望

总的来说，子宫收缩的生理调控是一个多因素、多通路共同参与的复杂网络。目前研究已经基本搭建起间隙连接、离子通道、激素与微环境三大核心调控框架：以 Cx43 为主的间隙连接是实现宫缩同步化的关键结构；T 型/L 型钙通道、BKCa、Kv7、Piezo1 等离子通道决定了肌层兴奋性与收缩节律，并且会随妊娠进程和微环境变化发生功能性重塑；妊娠相关激素与母体代谢状态，则通过调节间隙连接和收缩蛋白表达，从整体上控制子宫的收缩能力。

近几年，免疫－肌细胞相互作用(比如 M2 型巨噬细胞铁死亡)、机械敏感信号(如 Piezo1 通道)等新方向不断出现，大大拓宽了研究视野，也让人们意识到非经典通路在宫缩调控里同样重要。现有认识仍存在不少关键缺口：一方面，各通路之间的交叉调控还没说清楚，比如免疫信号究竟怎么影响 Cx43 表达、机械通道如何与离子通道、激素信号一起推动分娩，这些都需要更系统的研究；另一方面，很多研究还停留在单一维度，缺少整合性的网络模型，很难完整解释生理性和病理性宫缩的差异。未来的研究应该更注重多信号整合和临床转化，重点把“免疫/机械信号－离子通道－间隙连接－宫缩同步化”这条完整通路打通，阐明各环节之间的动态调控关系。同时，加快基础研究向临床应用的落地，围绕 Cx43、关键离子通道等靶点开发精准干预方案，为优化分娩管理、防治宫缩相关疾病提供更扎实的理论支撑和可行策略。

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