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基于细胞力学的得气研究

王睿[1] 盛雯茹[1] 薛玺情[1] 陆东宇[1] 曾以德[1,2]*
([1]山东中医药大学中医药创新研究院, 济南 250355; [2]山东中医药大学针灸研究院, 济南 250355)

摘要 目前对于针刺的基础科学研究多聚焦于神经系统, 对穴区发生的针刺启动机制较少涉略。针刺启动指机械刺激在穴区引发的初始细胞应答, 包括机械信号感知与转导; 得气则是针刺启动在临床层面的主观与客观体现。针刺产生的机械信号由机械力敏感的细胞转化为生化信号, 促使细胞释放生物活性因子, 改变细胞所在微环境; 进而影响邻近细胞的生物学响应, 产生迭代效应, 引发循经感传, 实现气至病所。在此过程中, 穴位敏化以局部免疫反应降低细胞对机械刺激感受的阈值, 进而放大针刺得气效应。据此, 该文提出“力学输入–细胞机械信号转化–针刺信号级联放大–针感传导”的假说框架, 将针刺信号由“点”(穴位)至“线”(经脉)乃至全身的传导过程阐述为“敏化得气–循经感传–系统调控”的逻辑链。在此基础上, 进一步构建了涵盖力学物理量、生化分子及组织功能反应的得气多维量化评估框架, 以期为假说的实验验证提供可测量的路径。该文从细胞力学视角阐释针刺得气的形质基础与信号转导机制, 探究穴位敏化与循经感传的内在联系, 为针刺本质提供现代科学的理论框架, 并为后续实验验证提供可检验的机制假说。

关键词 针刺; 细胞力学; 得气; 细胞机械信号转化; 循经感传; 穴位敏化

Research on Deqi Based on Cell Mechanics

WANG Rui[1], SHENG Wenru[1], XUE Xiqing[1], LU Dongyu[1], TSENG Yiider[1,2]*
([1]*Innovative Institute of Chinese Medicine and Pharmaceutical Science, Shandong University of Traditional Chinese Medicine, Jinan 250355, China*; [2]*Institute of Acupuncture and Moxibustion, Shandong University of Traditional Chinese Medicine, Jinan 250355, China*)

Abstract Current basic science research on acupuncture has predominantly focused on the nervous system, with limited exploration of the acupuncture initiation mechanism at acupoints. Acupuncture initiation refers to the initial cellular responses triggered by mechanical stimulation at acupoints, encompassing mechanical signal perception and transduction; Deqi (the arrival of qi) represents the clinical subjective and objective manifestation of this initiation. The mechanical signals generated by acupuncture are converted into biochemical signals by mechanically sensitive cells, which prompt the cells to release bioactive factors and alter the microenvironment where the cells are located. This, in turn, affects the biological responses of neighboring cells, creating an iterative effect, triggering meridian sensation transmission, and achieving Deqi at the affected area. During this process, acupoint sensitization reduces the threshold of cellular perception to mechanical stimulation via local immune responses, thereby amplifying the Deqi effect. Based on these observations, this paper proposes a hypothesis framework: “mechanical input-cellular mechanical signal conversion-acupuncture signal cascade amplification-needle sensation transmission”.

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*通信作者。Tel: 0531-89628012, E-mail: ytseng@sdutcm.edu.cn
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*Corresponding author. Tel: +86-531-89628012, E-mail: ytseng@sdutcm.edu.cn

The expansion of acupuncture signals from a “point” (acupoint) to a “line” (meridian) and the entire body is further conceptualized as a logical chain: “sensitized Deqi-meridian sensation propagation-systemic regulation”. Building on this, a multi-dimensional quantitative evaluation framework for Deqi is constructed, integrating mechanical physical quantities, biochemical molecules, and tissue functional responses, to provide a measurable pathway for experimental validation of the hypothesis. This paper elucidates the material basis and signal transduction mechanisms of acupuncture Deqi from a cellular mechanics perspective, establishes the intrinsic relationship between acupoint sensitization and meridian sensation propagation, and offers a modern scientific theoretical framework for the essence of acupuncture, along with a testable mechanistic hypothesis for subsequent experimental verification.

Keywords acupuncture; cell mechanics; Deqi; cellular mechanical signal conversion; propagated sensation along channels; acupoint sensitization

《黄帝内经》记载: “刺之要, 气至而有效。”在这里“气至”即是目前所称的“得气”(Deqi), 被认为是针刺取效的关键环节。在《标幽赋》中提到: “气速至而速效, 气迟至而不治”, 凸显了“得气”在针刺疗效中的核心地位。然而, 历代医家多从医者指下“沉紧”的阻力感以及患者受针处体验到的酸、麻、胀、重、痛等主观性感受加以描述。从现代科学的角度来看, 什么是得气? 它的形质基础是什么? 产生的信号如何转导? 这些仍然是需要进一步研究的课题。

现代对得气的研究多聚焦于得气后的神经生理变化, 对得气初始阶段时针刺穴区的作用, 也就是其启动机制则关注不足。随着细胞力学的发展, 细胞对机械刺激的反应及其在组织内信号转导中扮演的角色日益明晰, 这为得气的诠释提供了新的视角。因此, 从细胞力学角度对针刺启动与感传机制进行阐释, 是连接传统理论与现代科学的一个关键桥梁。

1 得气的内涵与针感辨析

得气的观念源于《黄帝内经》, 其中明确得气是针刺起效的核心。之后, 《标幽赋》以“鱼吞钩饵”来比喻针刺时施针者指下沉紧的感觉, 其与患者的酸胀感受相辅相成, 构成得气的双重表征。然而, 得气不仅是一种瞬间或静态的感觉, 更是医患之间通过提插捻转维持针感、产生气至病所效果的动态互动过程。

由于得气在针刺成效中的重要性, 现代医学采用如马萨诸塞州总医院针刺感觉量表(Massachusetts General Hospital Acupuncture Sensation Scale, MASS)等来对得气的状态提供客观化的指标, 希望能够对酸、麻、胀、重等患者的主观感受进行量化评分[1]。此类量表有助于得气的标准化研究, 然而却难以完全涵盖得气的整体内涵。得气不仅关乎局部的针感, 更涉及经气的运行以及脏腑的动态调节。因此, 在借鉴量化工具的同时, 现代研究仍需回归《黄帝内经》“气至而有效”这一根本原则。

2 针刺的细胞力学应答机制

从本质上看, 针刺操作的提插捻转向穴区组织施加了机械力。作为一种特定模式的机械力刺激, 针刺通过激活组织牵拉附着在胶原纤维上的细胞, 进而造成细胞骨架的重构和离子通道的开合等, 并以细胞的力学信号转导来产生生物学效应[2-3]。据此, 本文将从细胞力学与经气转导层面对得气的形质基础进行阐释。

2.1 经络–筋膜解剖关联与经隧假说

现代研究从解剖学角度描述中医经络时发现, 经络与筋膜的分布具有高度一致性[4], 也与《灵枢》中记载的经脉“伏行分肉之间”高度契合。但是这种相似性仅是分布上的相似, 对于筋膜是否就是经络循行的载体却没有定论。此外, 在穴区得气后拔针所需的力量较非穴区拔针所需的力量平均高18%[5]。同样在足三里穴区下针时, 得气时其组织的位移幅度与皮肤血流量均大于非得气时[6]。据此可知, 得气与其后的经络循行不能够单从组织的结构与分布来理解。从得气时组织的表现来看, 组织不仅被动承受针刺机械力, 还需协同其内部细胞对针刺机械力作出主动响应。

在传统理论中提到经气可“内属脏腑, 外络肢节”, 沿经隧运行。而现代研究试图以组织液在胶原纤维网络中流动来解释“经隧”: 针刺可激发组织液产生压力脉冲, 驱动透明质酸等黏弹性流体流动, 形成流体–固体耦合[7]。利用超声弹性成像观察到针刺

时结缔组织发生位移，可能影响局部结缔组织反应的空间分布[8]；利用荧光照相法对大鼠任脉低流阻通道进行活体显示，结果证实组织液可在结缔组织间隙中沿特定路线进行定向流动[9]；数学模型与离体实验则表明筋膜形变产生的压力梯度可驱动透明质酸发生定向流动[10]；结合活体示踪与流阻测量技术，在大鼠肢体深层结缔组织间隙中，观察到经胃经低电阻点注射的示踪剂发生长程迁移，且迁移路径与人体经穴针刺位置高度重合，为结缔组织中存在组织液流动的循经间质通道提供了直接的可视化证据[11]。然而，虽然援引“组织液流动假说”为针刺远端效应的力学传播提供了一种可能的解释框架，但是其完整内涵包括结缔组织内的血管、淋巴管及神经鞘膜间隙，目前的了解并不是对“经隧”本质的最终定论。

2.2 针刺力学表征与穴区细胞机械响应

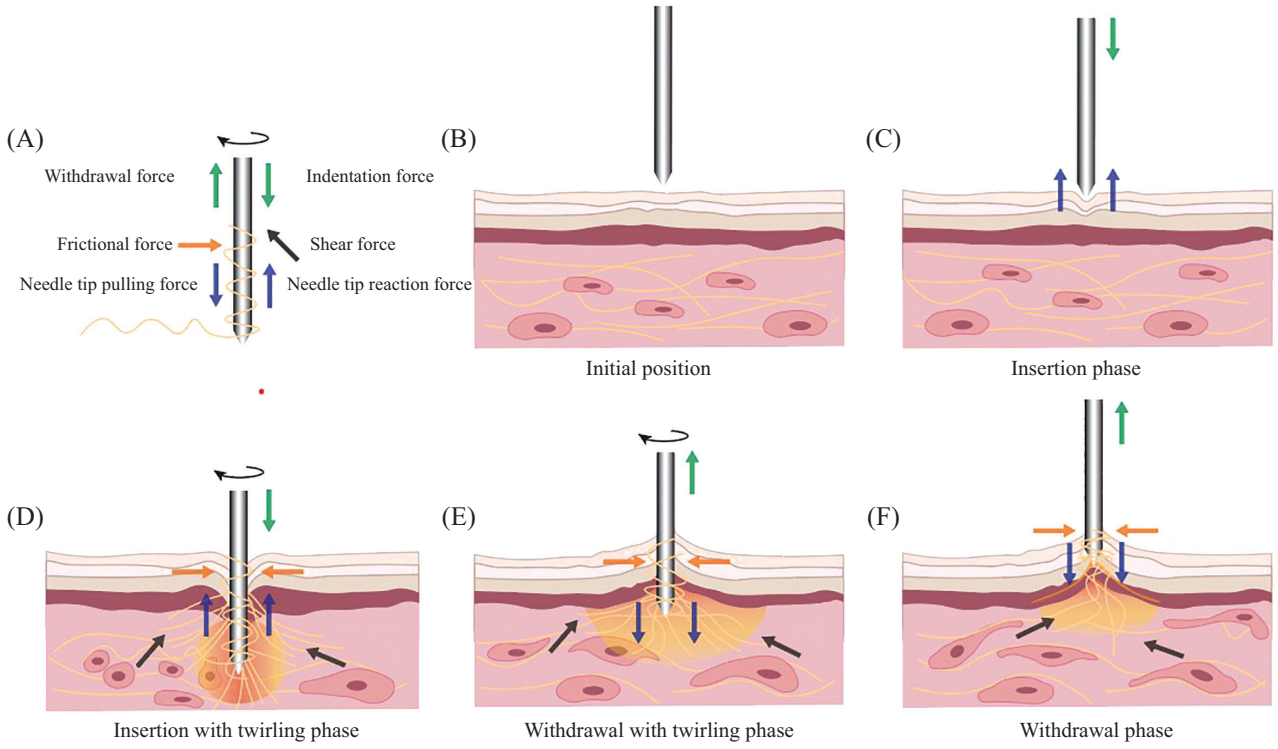
当针插入穴区时，针体对组织施加向下压力，组织则对针尖产生向上反力；拔针时，针体向上提拉，而组织对针尖产生向下拉力；而在捻转过程中，针体与胶原纤维缠绕形成“微绞盘”结构，并由缠绕在这个微绞盘结构中的胶原纤维片段将机械力延伸至网络中的所有与其连结的胶原纤维(也就是穴区的细胞外基质)。这些针体在提插捻转运动的过程中与穴区组织产生的机械耦合，使纤维网络被动地产生“振动”的同时，也在胶原纤维上产生大小不等的张力，对附着于胶原纤维上的细胞产生剪切力[12](图1)。

结缔组织内细胞因受细胞外基质的张力驱动而在胞内引发肌动蛋白(actin)的聚合以及应力纤维(stress fiber)的形成。这些变化会增强细胞的收缩能力和提高细胞骨架刚度，并通过黏着斑将细胞内张力传递至细胞外基质，进而使结缔组织的弹性模量发生改变，这种弹性模量的变化可在局部软组织中诱发低频机械扰动，低频机械扰动以剪切波的形式在结缔组织网络中传播[7,13]。在此过程中，细胞膜上的整合素(integrin)是细胞与其外基质产生黏附作用中最为关键的分子(图2)。细胞的整合素$\alpha_1\beta_1$和$\alpha_2\beta_1$可与皮肤中含量丰富的III型胶原结合[15]，经过一连串的生化反应，最终形成锚定于胞膜上的成熟黏着斑，使得外基质通过胞膜上整合素与以肌动蛋白为骨干的细胞骨架连接更为稳固，从而将细胞外基质上的机械张力更为及时地导入细胞骨架[16-17]。

此外，针刺后穴区细胞膜上的Piezo1表达显著上调[18]，Piezo1在针刺引起的组织拉伸、剪切或压力变化作用下迅速响应，介导胞外的Ca^{2+}内流，以毫秒级别启动钙依赖信号级联[19]。以角质细胞为例，其上的Piezo1通道在受力后被激活，可引发瞬时受体电位香草酸亚型4(transient receptor potential vanilloid 4, TRPV4)通道开放，导致胞内的Ca^{2+}水平持续升高[20-21]，还可促进三磷酸腺苷(adenosine triphosphate, ATP)释放，将针刺信号转化为生化信号[22]。针刺可激活肥大细胞膜上的机械敏感通道蛋白瞬时受体电位香草酸亚型2(transient receptor potential vanilloid 2, TRPV2)，在肥大细胞内引发Ca^{2+}流，触发颗粒胞吐，使得组胺(histamine, HA)释放量提升；释放的HA又可通过肥大细胞自分泌的方式与肥大细胞表面的H1或A1受体结合，进行级联放大，将机械力刺激高效地转导成生化信号，从而发挥针刺疗效[23]。这说明穴区内有多种能够对机械力产生感应的分子，它们在穴区构成“力学感知共同体”。

2.3 细胞骨架–核内转录的力学转导

细胞通过细胞膜上对机械力敏感的蛋白感知外界的机械刺激，并在胞内进行信号转导及级联放大(图2)。其中，整合素感知从细胞外基质传来的机械刺激后，细胞膜内侧的黏着斑激酶(focal adhesion kinase, FAK)和Src络氨酸激酶发生磷酸化，招募桩蛋白(paxillin)与纽蛋白(vinculin)，并激活RhoA/ROCK通路，驱动肌动蛋白骨架重组并增强细胞收缩力[24]。一方面，细胞骨架的张力变化降低对力学敏感的小GTP酶Ras相关蛋白Rap-2(Ras-related protein Rap-2, RAP2)的活性，导致Hippo通路核心激酶大肿瘤抑制激酶1/2(large tumor suppressor kinase 1/2, LATS1/2)的活性下降，阻断对Yes相关蛋白(Yes-associated protein, YAP)及包含PDZ结合基序的转录共激活因子(transcriptional co-activator with PDZ-binding motif, TAZ)的磷酸化，阻止YAP/TAZ的胞质滞留[25-26]，从而促使YAP/TAZ发生核转位[27]。另一方面，细胞骨架张力由Nesprin-1/2与SUN蛋白共同构成的核质连结器复合物[LINC(linker of nucleoskeleton and cytoskeleton)]传递至细胞核膜，引起核纤层蛋白lamin A/C表达的短暂下调，从而促进YAP1的核内定位，放大机械力引发的转录效应[28-29]。最终，YAP/TAZ与转录增强相关结构域蛋白(transcriptional enhanced associate domain, TEAD)等转录因子结合，激活可调控细胞生长与极



A: 整体示意(汇总各阶段受力方向)。绿色: 压缩力(向下)/提拉力(向上); 蓝色: 针尖反力(向上)/针尖拉力(向下); 黑色: 剪切力; 橙色: 摩擦力。B: 在初始位置, 针尖未接触皮肤。C: 在刺入阶段, 针体向下推进, 对组织产生向下的压入力; 组织对针尖产生向上的针尖反作用力。D: 在插入捻转阶段, 针体边插入边捻转, 针体与胶原纤维缠绕形成“微绞盘”结构; 针与组织界面产生摩擦力与剪切力, 纤维网络被牵拉。E: 在提出捻转阶段, 针体边捻转边上提, “微绞盘”结构逐渐松解, 但针体与纤维之间仍存摩擦力与剪切力, 组织继续受到牵拉。F: 在拔出阶段, 针体向上提拉, 对组织产生向上的拔出力; 组织则对针尖产生向下的针尖拉力。

A: overall schematic summarizing force directions across stages. Green indicates compressive force (downward) or lifting-pulling force (upward); blue indicates needle tip reaction force (upward) or needle tip pulling force (downward); black indicates shear force; orange indicates frictional force. B: at the initial position, the needle tip is not in contact with the skin. C: in penetration stage, the needle shaft is advanced downward, exerting a downward indentation force on the tissue, while the tissue generates an upward reaction force against the needle tip. D: in insertion-twisting stage, the needle shaft is inserted while being twisted, wrapping around collagen fibers to form a “micro-winch” structure; frictional and shear forces are generated at the needle-tissue interface, and the fiber network is subjected to traction. E: in withdrawal-twisting stage, the needle shaft is twisted while being lifted upward; the “micro-winch” structure gradually loosens, but frictional and shear forces persist between the shaft and fibers, and the tissue continues to be pulled. F: in extraction stage, the needle shaft is lifted upward, exerting an upward extraction force on the tissue, while the tissue exerts a downward pulling force on the needle tip.

图1 针刺不同阶段的力学表征

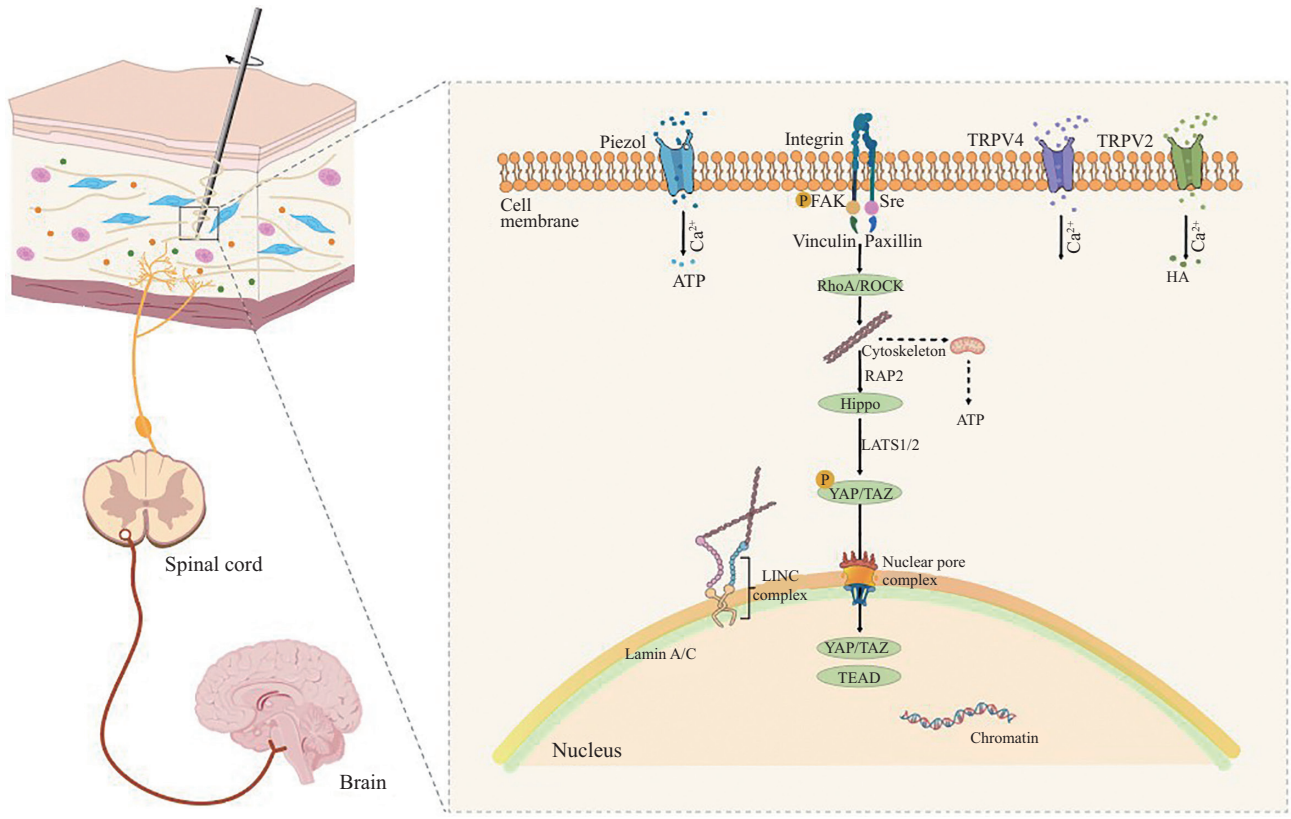
Fig.1 Mechanical characterization of acupuncture at different stages

化的靶基因转录[30], 实现力学信号向生物学效应的转化。此外, RhoA/ROCK通路的激活诱发细胞骨架重构, 进一步影响线粒体膜电位及ATP生成[31-32], 此“细胞骨架–线粒体轴”或可为针刺“调气”提供生理层面的支持。

2.4 多尺度力学信号整合

胶原纤维网络是结缔组织内一种主要的组成结构, 而附着在胶原纤维上的细胞并不是一成不变地仅附着在一根胶原纤维上, 于是针刺时细胞感受到的力学环境可能包含多种物理模式, 包括剪切力、拉伸力和压缩力[14,33]。综合近年研究, 不同力学模式优先激活的信号通路各有侧重: 剪切力主要激活Piezo1和整合素-FAK通路[34]; 拉伸力更易调动YAP/TAZ与RhoA/ROCK信号[35]; 而压缩力则对TRPV4等机械敏感通道具有较强刺激作用[36]。然而, 这些通路并非孤立运行, 而是通过共同的下游丝裂原活化蛋白激酶(mitogen-activated protein kinase, MAPK)等实现信号整合, 从而使细胞对力学微环境的机械力刺激作出协同响应[2]。

在细胞层面, 穴区不同细胞类型在力学信号转导中呈现清晰的层级关系。第一层(感受层): 角质细胞与成纤维细胞直接感知针刺机械力, 释放ATP等初始介质。其中角质细胞的Piezo1和外核苷三磷酸二磷酸水解酶-1(ectonucleoside triphosphate



针刺产生的机械力依次激活细胞膜上的机械敏感通道(Piezo1、TRPV2、TRPV4)和整合素(integrin)。整合素激活下游FAK/Src信号, 募集桩蛋白(paxillin)和纽蛋白(vinculin), 形成成熟黏着斑。这些信号进一步激活RhoA/ROCK通路, 引发细胞骨架重构; 并通过抑制RAP2激活Hippo通路, 导致LATS1/2失活, 从而促进YAP/TAZ去磷酸化。细胞骨架张力经LINC复合物传递至核膜, 引起lamin A/C表达下调, 促进YAP/TAZ通过核孔复合体入核。在核内, YAP/TAZ与TEAD转录因子结合, 调控靶基因表达。最终, 信号经脊髓上传至大脑, 完成从力学输入到中枢响应的全过程。

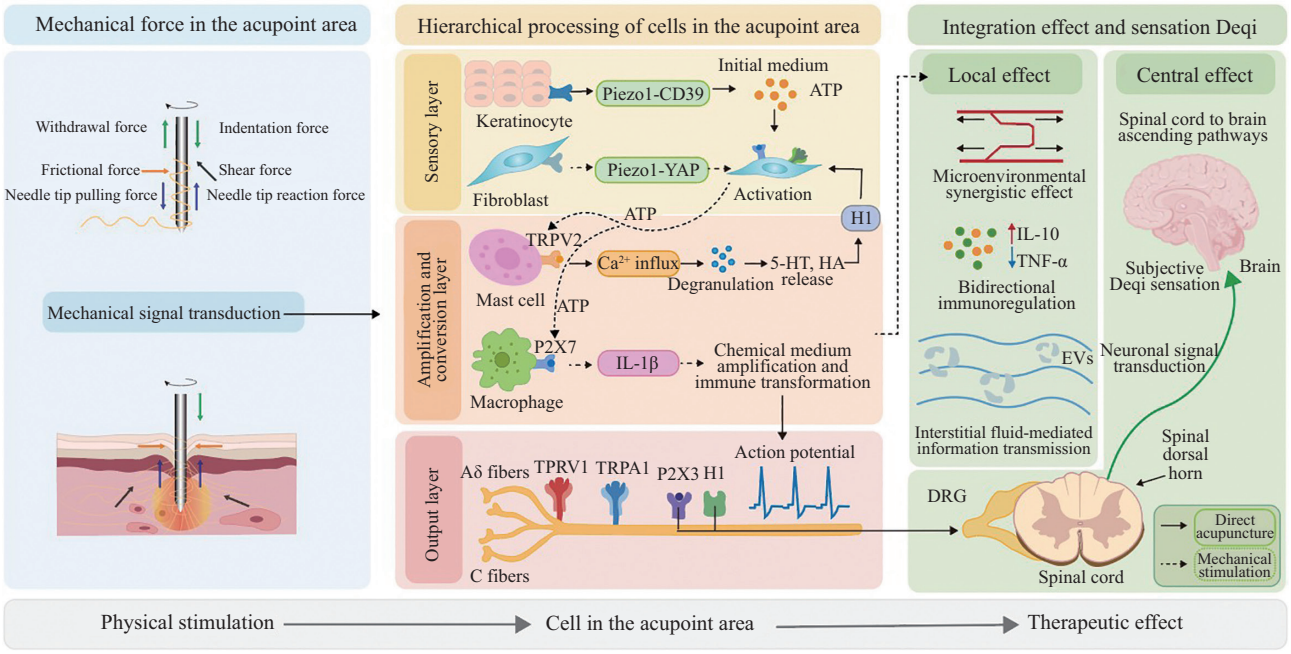
Acupuncture-induced mechanical stimulation sequentially activates mechanosensitive ion channels (Piezo1, TRPV2, and TRPV4) as well as integrin receptors on the plasma membrane. Integrin engagement triggers downstream FAK/Src signaling, leading to the recruitment of adaptor proteins paxillin and vinculin and the assembly of mature focal adhesions. This cascade subsequently activates the RhoA/ROCK pathway, driving actomyosin-mediated cytoskeletal remodeling. Inhibition of RAP2 activates the Hippo pathway, leading to inactivation of LATS1/2, thereby promoting dephosphorylation of YAP/TAZ. Cytoskeletal tension is transmitted to the nuclear envelope via the LINC complex, inducing downregulation of lamin A/C and facilitating YAP/TAZ nuclear import through the nuclear pore complex. Within the nucleus, YAP/TAZ heterodimerize with TEAD family transcription factors to regulate target gene expression. Finally, afferent signals are relayed via dorsal root ganglia and spinal cord circuits to higher brain centers, establishing a complete mechano-neural axis from peripheral mechanical input to central nervous system response.

图2 针刺机械信号转导示意图

Fig.2 Schematic diagram of mechanical signal transduction in acupuncture

diphosphohydrolase-1, ENTPD1; 又称CD39)共同调控胞外ATP水平, 释放的ATP经酶解触发腺苷信号, 起到针刺镇痛的效果[22]; 同时, 成纤维细胞在机械力刺激下, 其Piezo1-YAP通路也被激活[37]。第二层(放大与转换层): 肥大细胞被ATP激活, 通过TRPV2通道引发Ca^{2+}内流与脱颗粒, 释放5-羟色胺(5-hydroxytryptamine, 5-HT)、HA等化学信号, 其中HA通过H1受体增强成纤维细胞的力学敏感性[23]。巨噬细胞则通过P2X7受体响应ATP, 分泌白细胞介素-1β(interleukin-1 beta, IL-1β)等促炎细胞因子, 参与免疫网络激活[38]。肥大细胞与巨噬细胞协同配合, 共同完成从“力学”到“化学”的放大转换。第三层(输出层): 感觉神经末梢(Aδ/C纤维)通过其上的TRPV1、TRPA1等通道直接感受力学刺激, 或通过嘌呤能受体P2X3(P2X purinoceptor 3)、H1等响应化学介质, 产生动作电位[18,39]。

针刺电信号经脊神经节传入脊髓背角, 再经脊髓–丘脑束上传至大脑皮层(S1、岛叶、前扣带回等脑区), 形成酸、麻、胀、重等主观得气体验[40]。同时, 细胞膜P2X7受体在感知ATP或剪切力后, 诱导释放携带生物活性分子的细胞外囊泡, 这些囊泡经组织液流动, 实现细胞间信息交换[41]; 针刺可以上调抗炎细胞因子白细胞介素-10(interleukin-10, IL-10)、下调



左侧展示针体作用于穴区组织的力学种类, 绿色: 压缩力(向下)/提拉力(向上); 蓝色: 针尖反力(向上)/针尖拉力(向下); 黑色: 剪切力; 橙色: 摩擦力。中心板块为穴区细胞的三层功能架构: 第一层感受层(角质细胞、成纤维细胞)通过Piezo1-CD39、Piezo1-YAP等机械敏感通道感知力学刺激, 引发ATP等初始因子释放; 第二层放大与转换层细胞(肥大细胞、成纤维细胞), 肥大细胞TRPV2通道被ATP激活, 引发Ca^{2+}内流, 进而通过脱颗粒释放5-HT和HA, 这些物质经H1受体反作用于成纤维细胞; 第三层输出层(Aδ纤维、C纤维), TRPV1、TRPA1通道打开, 嘌呤能受体P2X3、H1受体响应化学介质, 将整合后的化学信号转化为动作电位。右侧为IL-10、TNF-α等细胞因子产生的化学信号使穴区微环境发生协同效应, 并通过细胞外囊泡(EVs)及组织液介导的信息传递实现信号转导; 神经信号经背根节(DRG)及脊髓上传至中枢, 形成得气的主观感觉。实线箭头表示已发表研究中针刺刺激直接证实的效果; 虚线箭头表示使用非针刺机械刺激所报告的机械效应。

The left panel illustrates mechanical force types exerted by the acupuncture needle on acupoint tissue, green indicates compressive force (downward) or lifting-pulling force (upward); blue indicates needle tip reaction force (upward) or needle tip pulling force (downward); black indicates shear force; orange indicates frictional force. The central module outlines a three-tiered cellular functional architecture within the acupoint. Sensing layer (keratinocytes, fibroblasts): mechanosensitive channels (e.g., Piezo1-CD39, Piezo1-YAP) transduce mechanical stimuli, triggering the release of initial signaling factors such as ATP. Amplification and conversion layer (mast cells, fibroblasts): ATP activates TRPV2 channels in mast cells, inducing Ca^{2+} influx; subsequent degranulation releases 5-HT and HA, which act on fibroblasts via H1 receptors. Output layer (Aδ fibers, C fibers): activation of TRPV1/TRPA1 channels and engagement of purinergic receptors P2X3 and H1 convert integrated chemical signals into action potentials. The right panel depicts that chemical signals derived from cytokines (e.g., IL-10, TNF-α) drive synergistic effects in the acupoint microenvironment, with information propagation mediated by EVs (extracellular vesicles) and interstitial fluid. Neural signals are relayed to the central nervous system via the DRG (dorsal root ganglion) and spinal cord, generating the subjective sensation of Deqi. Solid arrows indicate effects directly demonstrated by acupuncture stimulation in published studies; dashed arrows indicate mechanical effects reported using non-acupuncture mechanical stimuli.

图3 多尺度力学信号整合图

Fig.3 Multiscale mechanical signal integration diagram

促炎细胞因子肿瘤坏死因子-α(tumor necrosis factor-alpha, TNF-α)等细胞因子的表达, 从而发挥双向免疫调节作用[42]。“力–化学–电”级联实现了从物理刺激到得气与治疗效应的完整闭环(图3)。

3 穴区敏化、得气与循经感传的三级放大

当机体处于疾病状态时, 相应的穴位会发生敏化现象, 表现为感受野扩大以及痛觉敏感性及热敏感性增加等, 这些变化与神经炎症密切相关[43]。穴区敏化后, 在穴区内与神经激发相关的肥大细胞数量增多、脱颗粒的阈值降低, 因此在承受同等针刺机械力刺激时, 肥大细胞更易于脱颗粒而释放胰蛋白酶(trypsin)、5-HT和HA等改变细胞微环境的生物介质[44]。

基于现有证据推测, 穴位敏化的程度可能与细胞微环境改变的程度有关。在穴区敏化状态下(出现各种免疫因子), 细胞可能表现出更强的机械响应能力(也就是更低的激活阈值), 从而改变得气效应的强度与范围, 并可能产生不同程度的循经感传[45]。在此, 针刺启动或许扮演着得气效应的“初级放大器”。针刺启动就是针刺产生的机械刺激通过针体引起细胞外基质的形变与张力变化, 从而对组织内

细胞产生剪切力、拉伸力和压缩力。而细胞上密集的机械感受器(Piezo1通道、TRPV2通道、TRPV4通道以及整合素家族等)激活，促使细胞释放ATP、P物质(substance P, SP)、降钙素基因相关肽(calcitonin gene-related peptide, CGRP)等生物分子[46]。这种机械信号向生物化学信号转化的机能，为后续发生的生理调节提供了充足的初始动力。

循经感传为针刺效应在空间内的扩展，是针感沿经脉传导的“远程效应”，实现针刺信号从“点”(穴位)到“线”(经脉)乃至“面”(脑及全身)的跨越过程[47]。《素问》中记载: “五脏之道，皆出于经隧，以行血气”。其中，“隧”的本义为通道，强调经气运行的空间性与流动性。现代研究如超声成像显示针刺时结缔组织发生位移[8]、经络线具有低阻抗特性[48]等，提示针感产生的信号可能沿结缔组织网络传播。进一步支持这一推论的研究包括: 在离体筋膜组织中，机械振动可沿胶原纤维束进行长程传播[49]; 在动物实验中，穴位注射荧光染料后，针刺可诱发荧光染料沿经络线的定向传播，循经方向上迁移长宽比接近3倍，而在非经脉部位注射后荧光信号则呈各向同性扩散[9]; 在人体研究中，沿经络线的组织液示踪剂迁移速度快于非经络对照区域[50]。需要指出的是，上述证据多来自离体或间接测量，更详细的机制研究必须依托更精准的在体成像技术和细胞力学理论。

4 得气的量化评估与验证路径

4.1 主观针感的量表评估

针刺得气的主观感受，已通过多套标准化量表工具得到评估与验证(表1)。除了前面提到的MASS外，还有主观针感量表(Subjective Acupuncture Sensation Scale, SASS)[58]、南安普顿针感问卷(Southampton Needle Sensation Questionaire, SNSQ)等[51,59]。量表法操作简便，但本质属主观评价，易受个体差异干扰，且无法用于动物实验。因此，必须发展客观、可跨物种转化的量化指标。

4.2 客观表征: 生物力学与生化功能指标

得气的客观表征可划分为三个可量化层级: 力学物理量，如应力、应变、扭矩等; 组织微环境生化量，如ATP、Ca^{2+}、HA等; 组织功能响应量，如肌电、血流、温度、脑功能信号等(表1)。

4.2.1 *力学物理量* 得气时施针者手下的“沉紧感”本质上是一种力学触觉，且针刺本质是一个生物力学过程，针体刺入皮肤后，通过提插捻转产生机械性刺激，使组织发生位移，在局部形成应力场。有学者根据这个现象，研制了针刺专用力传感器，用于该应力场的定量表征[52,60-61]，此外，还可借助现代技术观测针刺过程中穴区结缔组织的变化[62-63]。行针手法的核心要素包括提插幅度、捻转角度与频率，均可定量控制。目前剪切波弹性成像(shear wave elastography, SWE)可无创测量穴位局部组织的弹性模量(kPa)，且得气后肌肉层弹性模量显著升高[14]。有限元模型(finite element model, FEM)则显示在针体穿刺皮肤时，最大主应力集中于针尖前方，其大小与针具半径、穿刺速度相关[53]。综上，拔出力(N)、捻转扭矩(N·m)、组织位移(mm)及弹性模量变化率(%)等可以一起构成表征得气力学效应的可测量指标组。

4.2.2 *生化信号分子* 得气的发生不仅涉及针刺启动的力学过程，还伴随穴区微环境的生化级联反应，进而触发沿神经通路的信号转导与中枢整合效应。力学信号向生物效应的转化受局部微环境生化分子的影响，其精细机制主要通过离体细胞力学加载实验进行分析。关键生化指标包括: 机械刺激诱导释放的ATP及其介导的嘌呤能信号[22]、细胞内Ca^{2+}信号与钙波传播速度[2,64-65]，以及肥大细胞脱颗粒所释放的HA[23]等。

4.2.3 *组织功能响应* 生化级联最终表现为组织器官的功能状态变化，主要通过在体实验与神经影像技术评估。核心功能指标包括: 反映局部肌纤维微弱收缩的肌电信号、反映微循环灌注与代谢的局部血流与温度变化，如激光多普勒血流成像、红外热成像[54-55]，以及反映中枢整合效应的脑功能信号，如功能性磁共振成像(functional magnetic resonance imaging, fMRI)、脑电图(electroencephalogram, EEG)、功能性近红外光谱(functional near-infrared spectroscopy, fNIRS)等[56-57,68]。这些指标为得气提供了多维的生理层面输出证据。

综上，三个可量化层级构成了从力学交互、生化涌现到功能重塑的客观量化框架。在体实验是功能测量的主阵地，而力学测量可在体或离体条件下进行; 离体实验则是解析力学–生化转导的核心平台，未来研究需推动它们的深度整合以构建全链条量化标准。

针刺启动的过程不仅可产生局部的穿刺力和摩擦阻力，还可在组织内部建立一个随针体运动而

表1 得气量化指标的综合框架(测量与验证体系)

Table 1 A comprehensive framework for the quantification of Deqi (measurement and validation system)

指标类型 Indicator type	具体指标 Specific indicator	测量/验证方式 Measurement/validation system	验证目的 Validation purpose	参考文献 References
Subjective score	MASS	Visual analog scale (0-10), questionnaire (13 sensations)	Quantify subjective needle sensation intensity and quality	[1]
	SASS	10 cm visual analog scale (9 sensations+anxiety score)	Evaluate composite needle sensation, distinguish Deqi from pain	[58]
	SNSQ	None, mild, moderate, severe rating (17 sensations)	Multi-dimensional assessment of needle sensation quality	[59]
Mechanical	Needle pull-out force, twisting torque	Force sensor (dedicated needle/custom sensor)	Quantify the practitioner's Deqi hand sensation	[60-61]
	Tissue elastic modulus	Shear wave elastography	Characterize local stiffness changes at acupoints	[14]
	Stress field distribution	Finite element model simulation	Reveal force transmission pathways	[53]
Morphology	Connective tissue winding angle, displacement	Ultrasound/tissue section; polarized light microscopy	Observe deformation and winding of connective tissue fibers	[5,62-63]
Molecular	ATP concentration in acupoint area	Microdialysis+luciferase	Quantify the initial signal activated by acupuncture	[22]
	Ca^{2+} wave velocity	*In vivo* calcium imaging (spinal cord/DRG); microfluidic chip co-culture	Detect communication efficiency of acupuncture signals	[64-65]
EMG (electromyography)	EMG amplitude, firing frequency; integrated EMG value	Surface EMG/implantable EMG electrodes; isolated nerve-muscle preparation	Reflect spinal reflex excitability, quantify Deqi initiation	[54]
Blood flow	Local blood perfusion	Laser Doppler flowmetry, ultrasound	Vasodilation and acupuncture maintenance-phase response	[62,66-67]
Temperature	Acupoint skin temperature	Infrared thermography	Auxiliary monitoring of local energy metabolism	[55]
Brain function	BOLD (blood oxygen level-dependent) signal	fMRI (functional magnetic resonance imaging)	Characterize central integration effects	[56]
	EEG band power, functional connectivity, and microstates	EEG (electroencephalogram)	Track real-time EEG dynamics	[57]
	Concentrations of HbO (oxyhemoglobin) and HbR (deoxyhemoglobin)	fNIRS (functional near-infrared spectroscopy)	Human studies of acupuncture central mechanisms in natural environments	[68]

动态变化的应力场。这一应力场的空间分布和时序演化规律, 与得气感的产生、穴位敏化及针刺疗效的启动密切相关。应进一步将应力场分布与得气主观评分、穴区组织响应及临床疗效进行关联分析, 明确不同针刺手法对应的最优应力场模式, 为针刺手法的定量优化和针刺机器人的力学控制提供理论依据。

5 讨论与展望

《素问·六微旨大论》指出: "出入废则神机化灭, 升降息则气立孤危", 揭示生命以物质为基础、运动为表象、信息调控为枢纽的普遍规律。针刺"调气"即通过物理刺激调控机体经络的升降出入。本文从细胞力学视角, 整合了得气传统理论与现代基础科学研究, 系统性地阐释了针刺启动的形质基础与经气转导机制, 为针刺得气提供了一个可能的、多尺度的科学诠释, 涵盖形质基础与信号转导机制。

细胞力学研究揭示了得气现象的物理化学本质: 机械刺激通过细胞力学转导启动生物应答, 在"力学输入–细胞机械信号转化–针刺信号级联放大–针感传导"的假说框架下, 本文尝试阐明"针刺启动–敏化得气–循经感传–系统调控"的逻辑链, 为针刺从经验描

述向精准力学调控的范式转变提供理论探索，并对已有细胞力学技术在针刺研究中的应用及得气量化指标进行系统梳理。

上述假说旨在与现有的以神经通路为核心的针刺研究形成互补。其核心观点在于：针刺机械刺激可被穴区多种细胞上的机械敏感分子直接感知，并引发局部生化信号释放，这一过程发生在感觉神经末梢兴奋之前，构成针刺信号启动的早期环节。

值得关注的潜在机制包括：反复针刺或穴位敏化状态下细胞外基质刚度的变化，可能通过YAP/TAZ信号通路影响穴区细胞的转录活性，进而调节其对力学刺激的转录适应性。这一现象在体外细胞力学研究中已有报道，在体内条件下进行针刺时该现象的存在与功能意义需进一步验证。此外，部分研究提示细胞外囊泡可能参与细胞间通信，其在结缔组织间隙中的迁移是否与循经感传相关，需通过特异性阻断实验予以检验。

未来研究可优先关注以下方向：建立不同针刺手法参数(如提插幅度、捻转角度、频率)与穴区细胞力学响应(如ATP释放量、胞内钙信号)之间的量效关系；利用基因敲除或药理学干预手段，在在体条件下验证特定机械敏感分子对得气感及针刺效应的影响；探索细胞力学转导通路与神经通路在针刺信号传递中的时序协同关系。

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