·综述·

运动训练与骨生长代谢的研究进展

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摘要: 采用文献研究法从运动训练对运动员(包括力量性、耐力性、速度性、柔韧性运动项目)骨形态结构、骨量、骨密度的影响及其相互间的差异等方面进行综述。提出目前不同运动项目对运动员骨代谢影响的研究不够全面,大多只集中于骨形态结构、骨量和骨密度阶段,没有深入探讨其机制,涉及到干细胞层面的研究尚未全面展开。

关键词: 运动训练;骨生长代谢;进展

Research progress of exercise training and bone metabolism

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Abstract: The effect of exercise training on bone morphological structure, bone mass, bone mineral density of athletes and the difference among them, including physical strength resistance, endurance resistance, speed resistance, and flexibility resistance, is reviewed using literature research method. At present, researches about different projects on bone metabolism of athletes are not comprehensive enough. They mainly focuse on bone morphological structure, bone mass, and bone mineral density. Few studies discuss the underlying mechanism at the level of stem cells.

Key words: Exercise training; Bone metabolism; Progress

运动训练学作为一门真正意义上的学科自本世纪六十年代于莱比锡问世以来,至今已经经历了50余年的历史。我国有文献将运动训练界定为"在教练员与运动员的参与之下的,旨在提高或保持运动员运动成绩而进行的一种教育过程"[1]。业已证实,长期系统的运动训练对人体各器官、系统的形态、结构和机能水平都会产生影响,从而形成独特的运动员形态和机能特征[2]。近年来,运动训练对骨量、骨密度、骨生物力学性能的改变以及骨代谢的影响引起了体育界的广泛关注,是运动医学工作研究的重要课题之一。本文主要对运动训练影响骨生长代谢的研究成果作一简要综述。

1 不同运动项目对运动员骨形态结构、骨量、骨密度的影响

长期的运动训练对于运动员来说是一种刺激,必然机体自身出现反应,包括组织、器官的形态、结构和分子水平等发生变化。运动训练方式不同对骨的生长、结构以及生物力学特性影响也不相同。而骨代谢的过程往往能反映破骨细胞和成骨细胞的活动,骨基质与骨矿的变化以及各种疾病状态,且更及时、更灵敏[3]。

1.1 力量性运动项目

前苏联普利维斯、库拉琴柯夫、阿斯丹宁等曾报道过,在青少年举重运动员表现出骨密质层变厚,骨松质改建。王小燕[4]通过对举重、中长跑和不从事任何专项运动的男青年的前臂桡骨、尺骨骨皮质、骨髓腔、骨矿含量和骨密度等进行统计分析。结果显示,举重组的桡、尺骨骨皮质厚度显著高于中长跑组和对照组,且中长跑组略高于对照组,但无显著性差异;骨髓腔径未见随骨皮质增厚而缩小;三组之间的桡、尺骨骨矿物含量和骨密度存在着显著性差异,即举重组、中长跑组、对照组。Nilsson[5]的早期研究也表明,举重运动员的骨密度高于其它项目运动员。研究运动训练起始年龄对网球和壁球运动员惯用手骨量的影响,选择芬兰国家级女运动员105名和50

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名健康女性(对照组),运动组根据其训练年份分为4组:0~2年组;3~5年组;6~15年组和大于15年组。测量肱骨近端、肱骨长骨和横截面以及桡骨远端的骨矿含量,得出运动组每组的骨矿含量与对照组相比呈高显著性差异;各组之间骨矿含量也表现出显著差异[6]。

1.2 耐力性运动项目

魏亚茹等[7]对黑龙江省冰上项目运动员运动训练强度与骨密度进行相关研究。结果显示,冰上项目运动员跟骨超声骨量参数显著高于体育类大学生。研究男子赛艇运动员急性负能量平衡中的骨骼指标,9名男子运动员参加了耗时2小时的长期低强度单手摇桨划船恒定负荷量的实验,实验前后对比,骨钙素呈显著差异[8]。研究女子优秀耐力跑运动员的骨密度,34.2%的运动员腰椎骨密度低,且得出运动员腰椎骨密度低与训练量的大小有一定的关系[9]。Aboia 等分析30名马拉松运动员体内总钙量,发现比同年龄配对的书案工作者要高出11%。据 Dalon 等人报道,越野跑运动员肱骨头、桡骨、尺骨、跟骨无机盐含量增加20%;腰椎、股骨颈无机盐含量增加8%-9%[10]。

1.3 速度性运动项目

杨浩然[11]研究得出乒乓球运动员的跟骨骨密度与普通大学生相比有显著性差异。赵杰修等[12]发现短跑运动员的骨密度显著高于对照组。

1.4 柔韧性运动项目

用双能X线骨密度法研究青春期和退役女子体操运动员的骨密度差异,选择45名青春期女子体操运动员(每周训练15-36小时)、36名退役女子体操运动员和50名健康女性。在横断面分析,青春期女子体操运动员的骨密度高于同等负荷量对照组0.7-1.9个标准差,呈显著性差异;退役女子体操运动员除头骨以外的骨密度高于同等负荷量对照组0.5-1.5个标准差;在12个月训练过程中,青春期女子体操运动员的骨密度比对照组增加了30-85%,得出青春期前的运动可以提高骨密度并维持到成年[13]。

1.5 不同运动项目对运动员骨生长代谢影响的差异

极端的静止(如卧床)和活动(如运动员)对于骨量的影响显著不同。研究表明,运动员的骨密度比久坐的受试者更大[5,14]。长期从事专项运动训练的男大学生左右髋关节及其股骨颈,L2、L3、L4,腰椎的骨密度高于非体育类男大学生[15]。Ari 等[16]对女子定向越野、自行车、滑雪和举重运动员的研究得出,举重运动员的骨密度高于其它项目运动员和正常对照者。比较女子优秀足球运动员和女子优秀游泳运动员骨密度之间的差异。选取身高、体重无差异的26名女子游泳运动员(15.9 ± 2年)和32名女子足球运动员(16.2 ± 0.7年)进行身体成分和骨密度检测,得出女子优秀足球运动员的骨密度显著高于游泳组[17]。比较26名男子运动员(包括跑步、摔跤和高尔夫球)的骨密度,得出摔跤运动员所测定骨骼的密度高于跑步组和高尔夫球组[18]。回顾性研究绝经后女子优秀运动员的骨密度、骨矿含量和身体成分的差异。选择在体重、身高、身体指数和活动时间无明显差异的24名绝经女子优秀运动员,根据其专项训练历史平均分为跑步组和游泳组,选取无年龄差异的24名女性作为对照组。研究表明,运动组的骨密度和骨矿含量显著高于对照组;运动组之间的骨矿含量无明显差异,但游泳组运动员的左右臂骨密度显著高于跑步组[19]。Morel 等[20]研究发现,划艇、游泳运动员的全身骨密度和下肢骨密度较低,而橄榄球、足球、拳击运动爱好者的全身骨密度以及下肢骨密度较高;足球、田径运动员的下肢骨密度占全身百分比较高,从事健美、拳击、攀岩和游泳的则是上肢骨密度比率大,橄榄球运动员的脊柱骨密度比率较大。魏亚茹等[21]对哈尔滨体育学院11个不同运动专项男大学生跟骨超声骨量进行研究。不同运动专项男大学生跟骨超声骨量 STI 值由高到低的顺序依次为田径、羽毛球、网球、游泳、跆拳道、足球、篮球、排球、体操、乒乓球和柔道。田径、游泳、羽毛球、网球专项男大学生的跟骨超声骨量 T 值高于同性别青年组平均值,而舞蹈、篮球、乒乓球、排球、足球、跆拳道专项的跟骨超声骨量 T 值低于同性别青年组平均值。不同运动专项、运动强度对男大学生跟骨超声骨量的影响不同。邓道善等[22]对我国不同项目运动员右前臂骨矿含量进行了测试,少年男子和成年男子运动员中以投掷、摔跤运动员最高,拳击次之,其它各专项相对较低;少年女子和成年女子运动员中以摔交项目最高,其次为投掷、游泳,其它各专项(速滑、跳跃、径赛、排球、艺术体操)较低。

综合以上材料可知,运动员的骨形态结构、骨量、骨密度不同于非运动训练的普通人,即使是运动员之间由于其专项和训练强度不同骨形态结构、骨量、骨密度也会出现差异。

2 运动训练对骨生长代谢的基础研究

2.1 动物实验研究

高强度的运动对于骨的影响短期内很明显[23]，动物模型在研究运动模式和骨之间的关系时很有帮助[24]，较大的骨密度往往出现在高强度活动组[25-28]。动物实验研究表明，运动训练影响细胞活动[2]。以往研究中高强度的动物实验多选用大鼠，运动形式主要集中于踏步、跑步、游泳、悬吊、跳跃，多见于高强度运动，往往使动物达到疲劳的状态，对骨形态结构、矿物质含量、骨密度、骨生物力学等方面产生影响。

在动物实验中，Raab 等对踏步训练 5 个月的母猪股骨作形态学测量，发现皮质骨骨单位增加 23%，骨单位平均骨壁厚度也明显增加，但其横截面积和骨矿物质含量无变化。王人卫等[29]研究在递增运动负荷下，骨密度的动态变化，得出中长期的大强度运动训练可导致大鼠骨量减少，引起雌性大鼠骨密度的改变。研究不同强度的间歇跑步训练对生长期大鼠骨代谢的影响，按体重随机分为 7 组，包括对照组和运动组。根据其最大摄氧量将运动组分为 65%、70%、75%、80%、85% 和 90% 6 组。研究表明，最大摄氧量为 70% 运动组的大鼠骨密度明显高于其他对照组；最大摄氧量为 70% 运动组的大鼠碱性磷酸酶显著高于其他对照组[30]。Bourrin[31]以 5 月龄雄性大鼠进行大强度跑台运动（80% VO_2max）11 周，大鼠造成骨丢失，研究显示这种骨丢失机理可能是成骨细胞活性降低所致。为了研究不同的运动训练模式对骨生长的影响，T. H. Huang[24]取 7 周 29 只雄性大鼠随机分为跑步组、游泳组和对照组。测得跑步组大鼠的胫骨近端骨密度显著高于游泳组，跑步组和游泳组大鼠的股骨湿重显著高于对照组；采用三点弯曲试验测得跑步组和游泳组大鼠的胫骨和股骨骨机械性能显著高于对照组。李建设等[32]研究长期不同强度游泳运动对生长期大鼠骨代谢生化指标及骨量的影响，得出负重 6% 游泳组右侧股骨骨密度、骨矿含量均显著高于安静对照组、实验对照组和无负重游泳组，碱性磷酸酶、骨钙素非常显著高于安静对照组、实验对照组、无负重游泳组和负重 9% 游泳组，骨钙素水平显著高于负重 3% 游泳组。大鼠经过尾部悬吊，其股骨和胫骨的重量显著低于对照组，得出骨的生长受到心血管改变和尾部悬吊影响[33]。对于动物实验中的尾部悬吊常常和模拟失重结合起来，导致股骨和软骨基质钙化障碍，原有钙化基质内钙盐脱钙明显。佟海英等[34]观察尾部悬吊模拟失重及解悬吊后大鼠骨密度的变化。大鼠尾部悬吊 14 天即可引起骨代谢的紊乱，承重骨骨矿盐大量丢失；即使解悬吊 14 天后承重骨骨密度及力学强度也明显降低，骨代谢紊乱短期内不能恢复正常。对不同月龄的大鼠尾吊 4 周后，测得大鼠骨形成指标均显著下降；除弹性负荷外，大鼠负重骨各力学指标均显著下降；年幼鼠骨矿盐含量减少高于成年鼠，得出尾吊模拟失重对不同发育阶段大鼠骨代谢和负重骨生物力学性能变化的影响程度不同[35]。虽说上述尾部悬吊实验都使大鼠骨矿质含量丢失、骨密度及力学强度下降，骨代谢发生紊乱，但就其悬吊的方法和时间是否能对大鼠构成高强度训练还有待进一步商榷。杨国敏、魏文仪[36,37]对生长期大鼠进行 8 周大强度跳跃训练，并对不同运动量进行比较研究，测定其股骨的长度和横截面参数及骨盐含量、骨密度。结果表明，大鼠股骨长轴方向成长受到了明显抑制，骨盐含量明显下降，且运动量越大表现越明显。

目前，动物实验中骨代谢的测定方法包括骨生化指标（包括骨钙素、血清钙、磷、碱性磷酸酶等）、组织形态学、骨密度测定、骨生物力学测试等等。在上述的几种测定方法中由于生物力学测试因具有一定的破坏性，尤其是在临床试验以前，往往将其设计为最后一种测试手段。

2.2 人体机制研究

运动训练对骨生长代谢的影响主要包括对骨细胞系和骨内环境的影响，其中骨细胞系的主要成分包括破骨细胞、成骨细胞、骨细胞、骨衬细胞核它们的前体细胞，还有骨髓中免疫系统中的相关细胞[38]；骨内环境主要因子包括骨形态发生蛋白、骨钙素、碱性磷酸酶、胰岛素样生长因子-I、转化生长因子-B、骨桥蛋白、骨涎蛋白等。

研究一个赛季（4～6 个月）不同运动强度女子运动员的骨形成和吸收波动，分为运动组（29 人）和对照组（4 人），其中运动组分为 3 组：篮球组（6 人）、足球组（12 人）和游泳组（11 人）。研究表明，篮球组和足球组运动员的碱性磷酸酶高于游泳组和对照组，并且在赛季中保持稳定，无明显改变[39]。在研究少年女子花样游泳运动员的骨代谢时，选取 14～17 岁花样游泳女运动员 20 名（每周训练至少 6 小时），对照组是年龄相仿的 20 名少女（不参加运动竞赛或者除学校组织外的运动），排除任何骨疾病、口服糖皮质激素、口服避孕药、无月经或延迟，得

出两组少女的血清骨钙素浓度无明显差异[40]。研究维生素D受体多态性和运动训练对骨代谢的影响,选取年青男性FF基因组10人、Ff或ff基因组10人、对照组14人,两基因组通过六个周期的运动训练,对照组不训练,测得两基因组的骨矿含量、骨密度、碱性磷酸酶训练后明显高于训练前,与对照组相比骨钙素呈显著增长[41]。研究女子滑雪运动员在不同时期骨代谢标志物的变化,对14名顶级女子滑雪运动员在相对静止期、赛前期、赛季期进行碱性磷酸酶、酸性磷酸酶5b、骨钙素进行检测,得出在赛前期和赛季期碱性磷酸酶、酸性磷酸酶5b和骨钙素显著增加;在赛前期和赛季期四个骨骼标记物之间呈两两相关,但是在相对静止期和赛前期之间则无显著相关性[42]。

3 展望

综上所述,目前骨代谢方面的研究主要集中于骨生化指标的测试、组织形态学测量、骨密度测定和骨生物力学测试。运动训练对骨生长代谢有影响;运动员的骨代谢不同于非运动训练的普通人,即使是运动员之间由于其专项和运动强度的不同骨代谢也会出现差异。骨代谢指标适用于监测运动员的生理机能状态,对于提高运动成绩等方面发挥着重要的作用。

儿童、青少年在早期运动过程中训练强度、训练起始年龄都需要进一步研究,因为儿童时期的骨骼发育达到理想水平,获得最佳峰值骨量,是预防骨质疏松症的一个关键因素。临床上也将骨代谢指标用于特发性身材矮小儿童骨生长发育评价体系中。

不同专项运动员由于长期从事运动训练强度、项目特点不同使骨生长代谢发生变化,但是目前对于运动员骨代谢的研究大多只集中在骨形态结构、骨量和骨密度阶段,仅仅只是骨代谢的最终结果,没有进一步深入探讨其机制;同时不同运动项目运动员骨代谢研究不全面,特别是高危运动项目研究缺失。

运动员骨生长代谢出现紊乱的时候,外源性摄入物质(如食物、药物等)、治疗(如针灸、推拿、整理活动等)对其骨代谢紊乱是否加以缓解这一部分还有待进一步研究。

在骨代谢中,激素和许多细胞因子影响着骨形成和吸收过程。目前骨代谢过程中存在WNT/β-连环蛋白通路、NFAT通路、胰岛素信号通路、Sema4D-Plexin-B1通路、PI3K-AKT及MAPK通路等已然对于研究骨代谢有很好的帮助。骨代谢涉及到干细胞层面的研究尚未全面展开。随着科研技术的飞速发展以及细胞分子生物学研究的不断进行,相信对骨代谢的研究会步步深入,为运动训练提供理论依据。

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运动训练与骨生长代谢的研究进展

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