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EXPLORING A NEW CONCEPT OF BONE HEALTH

The role of collagen, magnesium, zinc, manganese and copper in bone toughness

探索骨骼健康新概念

骨胶原和镁锌锰铜在骨韧性中的作用

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MAINTAINING BONE HEALTH OVER THE LONG TERM IS MORE THAN JUST A MATTER OF DENSITY OR STRENGTH — IT'S ALSO ABOUT TOUGHNESS.

Bone toughness is the ability of bones to absorb impact from falls or hard knocks. Recent research has revealed that bone toughness is crucial in preventing fractures and ensuring long-term bone health.

Scientists have been exploring how this toughness comes about and are finding the mechanisms originate in a complex interplay of organic and inorganic components, influenced by collagen networks and mineral interactions.

Over the past decades, advances in biomechanics and nutritional science have shed light on how collagen — the most abundant protein in the organic bone matrix — boosts toughness through its unusual triple helix structure.

But it's not only collagen that holds the key to bone toughness. Minerals such as magnesium, zinc, manganese and copper — often overlooked in traditional bone health strategies — are gaining attention for their newly discovered roles in collagen synthesis and maintaining bone integrity. Research is continuing to unravel these connections.

The following pages feature highlights of some significant findings about bone toughness and resilience, made by researchers globally, as well as the role of collagen and micro-nutrients.

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长期维持骨骼健康不仅依赖骨密度或骨强度，还取决于骨韧性。

骨韧性即骨骼承受因跌倒或重击产生的冲击的能力。近期研究显示，骨韧性是预防骨折和保证长期骨骼健康的关键。

科学家一直在探索骨韧性的形成机制，他们发现这种机制源于骨骼中有机质与无机质的复杂作用，受到骨胶原网络和矿物质相互作用的影响。

过去几十年里，生物力学和营养学的进展发现了骨胶原——骨有机质中最丰富的蛋白质——如何通过其独特的三螺旋结构促进骨韧性。

不过，决定骨韧性的不只是骨胶原。镁锌锰铜这类矿物质在维持骨骼健康的传统策略中常被忽视，但最新研究发现了它们对于骨胶原形成以及维持骨完整性的作用，使得这些元素受到越来越多的关注。科学研究正在不断揭示这些关联。

接下来的内容阐释了全球科研人员关于骨韧性的部分重要发现，以及骨胶原与微量营养素的作用。

The hidden factor in bone health: bone toughness

Toughness helps bones to resist damage and fractures caused by deformation.

In 2019, approximately 178 million new cases of fractures were recorded worldwide. And, 455 million people were living with fracture-related symptoms, such as limited mobility, chronic pain and nerve damage.

Bone health is not just defined by density or strength, but also by toughness. Strength is needed for bones to endure impacts, while toughness helps bones to resist damage and fractures caused by deformation.

Both the toughness and strength of bone are largely attributed to the nature of bone matrix — the framework that gives shape and form to bone tissue and provides mechanical support. Bone matrix is mainly composed of collagen fibres and hydroxyapatite (calcium-phosphate) crystals, along with other minerals, arranged in a hierarchical structure consisting of distinct structural features from the nano- to macro levels.

A better understanding of what contributes to bone toughness can help minimize the risk of fractures.

This unique structural arrangement is key to the toughness of bone, according to a team of researchers from Beihang University in Beijing, China[1]. In their 2022 review published in the journal *Bone Research*, the researchers highlighted how this "special multistage structure of the bone matrix gives it a unique toughening mechanism."

For instance, the triple helix structure of collagen fibres, and the way they are cross-linked, enable them to deform through stretching and sliding, rather than breaking when force is applied — helping prevent fractures. Similarly, the staggered pattern of hydroxyapatite crystals contributes to bone's exceptional toughness[2].

Beyond structure, the properties of the matrix play an important role in bone toughness. About two-thirds of the matrix is made up of hydroxyapatite which directly influences bone strength. The remaining one-third of the bone matrix is collagen, which plays a pivotal role in toughness. For example, when a crack forms, collagen fibres spread across its surface, preventing further propagation.

In one experiment highlighted in the review, researchers heated bone specimens to temperatures ranging from 37°C to 200°C. As temperatures increased, the progressive denaturing of collagen led to a corresponding decline in bone toughness.

"These results showed that the collagen network plays an important role in the toughness of bone," the authors concluded.

Another significant factor impacting bone toughness is the process of mineralization. During bone formation, collagen molecules assemble into fibrils, attracting calcium and phosphate ions, which in turn leads to the formation of hydroxyapatite crystals. This process transforms the soft collagen matrix into a strong, mineralized tissue capable of withstanding high mechanical loads — a hallmark of healthy bones.

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骨骼健康的奥秘——骨韧性

骨韧性能帮助骨骼抵抗损伤，以及形变导致的骨折。

2019 年全球约有 1.78 亿例新增骨折病例。4.55 亿人有骨折相关症状，如活动能力受限、慢性疼痛和神经损伤。

骨骼健康不仅要看密度或强度，还要看韧性。骨骼需要强度来承受冲击，而韧性能帮助骨骼抵抗损伤以及形变导致的骨折。

骨骼的韧性和强度主要取决于骨基质的性质——骨基质是赋予骨组织外形和形态并提供机械支撑的框架。骨基质的主要成分包括骨胶原纤维和羟基磷灰石（磷酸钙）结晶，还有一些其他矿物质，这个结构呈层状排列，由从微观到宏观的不同结构特征组成。

北京航空航天大学的一个研究团队指出[1]，独特的结构排列是骨韧性的关键。该研究团队 2022 年在《骨研究》（Bone Research）上发表了一篇综述，强调了这种"骨基质的特殊多层结构赋予骨骼一种独特的增韧机制"。

比如，骨胶原纤维的三螺旋结构以及它们的交联方式，使它们能通过拉伸和滑动变形，从而不会在受力后断裂——这有助于预防骨折。类似地，羟基磷灰石结晶的交错排列模式也促进了骨骼的超强韧性[2]。

除了结构，骨基质的性质对于骨韧性也很重要。约 2/3 的骨基质由羟基磷灰石组成，这会直接影响骨强度。剩下 1/3 的骨基质是骨胶原，而骨胶原对韧性起到了关键作用。比如，当裂缝形成时，骨胶原纤维会在表面扩散，防止裂缝进一步扩大。

在这篇综述提到的一项研究中，研究人员将骨样本从 37° C 加热至 200° C。随着温度上升，骨胶原逐渐变性导致骨韧性相应下降。作者总结道，"这些结果表明，骨胶原网络在骨韧性中起到了重要作用。"

另一个影响骨韧性的重要因素是矿化过程。在骨形成期间，骨胶原分子聚集成纤维丝，吸引钙和磷酸根离子，这会反过来促进羟基磷灰石结晶的形成。这个过程将柔软的骨胶原基质转换成一种能承受高机械负荷的坚硬矿化组织——这也是健康骨骼的一个标志。

更好地理解促进骨韧性的因素，或帮助将骨折风险降至最低。

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How collagen contributes to bone toughness

The protein makes bones flexible, allowing them to absorb shocks and resist fractures.

The adult skeleton, consisting of 206 bones, serves multiple vital functions: it protects organs, stores minerals, and houses the marrow where blood cells are produced. One of its most important roles, however, is to be a scaffold to provide structure to the soft tissues of the body and to facilitate movement.

Central to this locomotive function is a high quality of bone, which helps prevent fractures. However, bone quality is a complex property influenced by many interrelated factors, as highlighted by a team of researchers from the French National Institute of Health and Medical Research (INSERM) in Paris, and Claude Bernard University in Lyon, France. In a review published in *Osteoporosis International*, they explain that bone quality is based on the amount of bone, its architecture, its turnover, and also the intrinsic properties of the mineral and organic matrix[1].

According to the authors, collagen is the most abundant protein in the organic matrix of bone. Previous studies revealed that collagen plays a substantial role in bone toughness — the ability of bone to absorb energy without breaking. The complex structure of collagen is central to this role. Collagen molecules assemble into thread-like fibrils, which in turn are cross-linked by the lysyl oxidase enzyme to create a stable, three-dimensional structure. This configuration contributes to the overall mechanical strength and elasticity of bone.

The importance of this complex structure is evident in conditions where it is disrupted. For instance, people with brittle bone disease have genetic mutations that cause collagen molecules to form branched, rather than long, rope-like, fibres. This results in thin, fragile bones prone to fractures.

Collagen in bones changes with age. In one study highlighted in the review, 40 healthy male subjects of a wide series of ages were analysed. Researchers found that bone collagen content increased with age, but started to decrease after they entered their 30s[2]. In osteoporosis too, collagen fibres undergo several abnormal changes — such as thinning, a reduction in quantity, and the adoption of a disorganized arrangement with altered cross-links — which lead to weakened bones and an increased risk of fractures.

Collagen may also undergo modifications after initial synthesis from mRNA — dynamic processes that can regulate the activity and function of proteins. One such modification is glycation, in which sugars such as fructose are attached to collagen molecules to form advanced glycation end products (AGEs).

According to the review authors, these compounds can accumulate with age, causing a decrease in bone toughness. They cite a study showing that higher levels of AGEs were associated with a 29% increase in bone stiffness, and a 79% higher likelihood of deformation under stress.

Furthermore, collagen's interaction with inorganic molecules, including hydroxyapatite — a calcium-rich mineral that is another major component of bone — plays a significant role in health. The integration of hydroxyapatite into collagen fibrils enables bones to achieve the balance between toughness and strength.

As the authors explain, bone strength refers to how much load bones can carry before breaking, whereas toughness — rooted in collagen's ability to absorb energy — reflects flexibility under stress. Achieving this balance depends on a complex interplay of factors.

骨胶原示意图。骨胶原是骨有机质中含量最多的蛋白质。

A conceptual illustration of bone collagen, the most abundant protein in the ogranic matrix of bone.

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骨胶原如何促进骨韧性

骨胶原蛋白能维持骨骼的柔韧性，使骨骼能承受冲击，不易发生骨折。

成人骨骼由 206 块骨头组成，承担着多种重要功能：保护器官，储存矿物质，为血细胞的生成提供骨髓环境。然而，骨骼最重要的作用之一是作为支架，支撑身体结构并支持身体运动。

良好的骨质量是实现运动功能的关键，还能预防骨折。不过，骨质量属性复杂，受许多相互关联的因素影响。由法国国家健康与医学研究院（INSERM）和里昂第一大学（Claude Bernard University）的研究人员组成的团队在《国际骨质疏松症》（Osteoporosis International）上发表了一篇综述，解释了骨质量取决于骨量、骨结构、骨转换，以及矿物质和有机基质的固有特性[1]。

作者指出，骨胶原是骨有机质中含量最多的蛋白质。既往研究表明，骨胶原对于维持骨韧性——骨骼吸收能量而不发生断裂的能力——很重要，而骨胶原的复杂结构是发挥这一重要作用的关键。骨胶原分子聚合成丝状的原纤维，随后在赖氨酸氧化酶的作用下发生交联，形成稳定的三维结构。这种结构能增强骨骼的整体机械强度和弹性。

这一复杂结构的重要性在其断裂时尤为凸显。例如，脆骨症（成骨不全症）患者体内的基因突变会使胶原分子形成分枝纤维而不是长绳状纤维。这会使骨骼变薄变脆，易发生骨折。

骨胶原会随年龄增长而改变。这篇综述提及的一项研究分析了 40 名不同年龄段的健康男性受试者。该研究发现，骨胶原含量会随年龄而上升，但在 30 岁后开始减少[2]。骨质疏松症患者的胶原纤维也会出现多种异常变化，如纤维变薄、数量减少、排列紊乱以及交联结构异常，这些变化会增加骨骼脆性和骨折风险。

骨胶原在最初由 mRNA 合成后也可能发生了修饰——这些动态过程会调节骨胶原蛋白的活性和功能。其中一个修饰是糖基化，在糖基化过程中，果糖等糖类附着在胶原分子上，形成晚期糖基化终产物（AGEs）。

综述作者指出，这些化合物会随年龄增长而积累，从而降低骨韧性。他们引用的一项研究表明，AGEs 水平较高与骨刚度增加 29% 有关，还与压力下骨骼变形的概率提高 79% 有关。

此外，骨胶原与无机分子（包括羟基磷灰石）的相互作用也对维持健康至关重要。羟基磷灰石是一种富含钙的矿物质，是骨骼的另一个主要成分。羟基磷灰石与胶原纤维的结合，使骨骼能在韧性和强度之间取得平衡。

作者解释道，骨强度指的是骨骼在断裂前能承受多大的负荷，而骨韧性取决于骨胶原吸收能量的能力，反映了骨骼在压力下的柔韧灵活性。实现这一平衡依赖各因素之间复杂的相互作用。

The minerals behind collagen formation

Magnesium, zinc, manganese and copper are among minerals that are crucial in collagen formation, increasing toughness and helping to prevent fractures.

Bone health is influenced by a variety of factors, with minerals playing a fundamental role in maintaining the strength and resilience of bone. Among them, calcium and phosphorus are often the first minerals that come to mind when you think of bone health. Together they form hydroxyapatite crystals that make bones strong.

However, recent research has revealed that small amounts of other elements, such as magnesium, zinc, manganese and copper, are just as essential in collagen synthesis that gives bones its toughness.

Magnesium and collagen synthesis

Although magnesium is found throughout our bodies, 60% of our magnesium is stored in bones. It plays a pivotal role, with experts from the European Food Safety Authority (EFSA) concluding in studies in 2009[1] and 2013[2], that it contributes to the development and maintenance of healthy bone in adults, infants and young children.

Magnesium is required for maintaining high bone density, which helps prevent fractures.

But when the authors looked at 12 studies concerning dietary intake of magnesium, they found that about 20% of adults and teenagers consumed less than the recommended amount, which in turn was associated with lower bone mineral density and a higher fracture risk.

This could be due to changes in collagen, the major organic component of bone. Magnesium may affect the secretion of so called 'propeptides' such as P1NP, a marker of collagen formation during bone formation. Elevated levels of P1NP indicate increased collagen synthesis. A 2014 study of 142 post-menopausal women in New Zealand found that magnesium intake increased levels of P1NP[3]. Meanwhile, another study of 144 women in South Africa found that those with low magnesium intakes had higher levels of CTX, a peptide fragment derived from collagen breakdown[4]. They noted that high levels of CTX occur when bone is being lost faster than it is being replaced, leading to osteoporosis.

While the best way to get sufficient magnesium is through a balanced diet, a small clinical trial led by researchers at the Yale University School of Medicine in the United States found that magnesium supplements could also bring benefits. The authors found an increase in the mineral content of hip bones in a study of 120 teenage girls who took magnesium as a dietary supplement after a year[5].

Vink Fan/shutterstock

多重矿物质促进骨胶原形成

镁、锌、锰、铜等矿物质对于骨胶原形成、增强骨韧性、预防骨折至关重要。

骨健康受多种因素影响，而矿物质对于维持骨强度和骨韧性必不可少。提到骨骼健康，人们首先想到的往往是钙和磷，它们能联合形成羟基磷灰石结晶，维持骨骼强度。

但近期研究表明，镁锌锰铜和其他微量元素对于促进骨韧性的骨胶原形成同样不可或缺。

镁与骨胶原合成

虽然镁遍布全身，但人体内 60% 的镁都贮存在骨骼中。根据欧洲食品安全局（EFSA）在 2009 年[1] 和 2013 年[2] 发表的研究，镁具有非常关键的作用，能促进成人和婴幼儿的健康骨骼发育和维持。

镁是维持骨密度高的关键，而骨密度高有助于预防骨折。

然而，研究人员在分析关于膳食镁摄入的 12 项研究后发现，约 20% 的成人和青少年的镁摄入量低于推荐标准，而这被发现与骨密度偏低以及骨折风险偏高相关。

这背后的原因可能是骨胶原的变化——骨胶原是骨骼的主要有机成分。镁或许会影响前肽的分泌，如 I 型原胶原氨基端前肽（P1NP），它是骨形成过程中骨胶原形成的标志物。P1NP 水平高意味着骨胶原合成增加。2014 年对新西兰 142 名绝经后女性开展的一项研究发现，摄入镁可提高 P1NP 水平[3]。与此同时，另一项针对南非 144 名女性的研究发现，镁摄入不足者体内的 CTX 水平较高——CTX 是骨胶原形成分解后产生的肽链片段[4]。研究团队指出，当骨骼流失速度快于再生速度时，CTX 水平就会升高，进而导致骨质疏松症。

虽然均衡饮食是获取足够镁的最佳方式，但美国耶鲁大学医学院的一项小型临床试验发现，镁补充剂同样有益。研究人员让 120 名青少年女性将镁作为膳食补充剂服用一年后，发现她们髋骨的镁含量有所增加[5]。

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Copper and collagen cross links

Copper is another important mineral for bone health, with roughly half of the body's copper stored in the bones and bone marrow. Copper is essential for the activity of lysyl oxidase, an enzyme that helps cross-link collagen fibres. The cross-linking process would strengthen the collagen matrix, which is crucial for bone integrity and toughness.

Both abnormally high- and low levels can lead to complications. A 2024 review conducted by researchers at China Medical University in Shenyang highlighted that a diet low in copper has been linked with age-related osteoporosis and low bone density[6]. The authors noted that copper deficiency can reduce bone strength by disrupting the cross-linking between collagen fibres. Copper deficiency can also increase abnormal activation of cells that resorb bone called osteoclasts, leading to weaker bones.

Though less common than copper deficiency, an excess of copper can promote harmful oxidation and interfere with bone metabolism, potentially damaging the outer layer of the bone, influencing strength. For instance, people who have Wilson's disease — a rare genetic order in which excess copper accumulates in various organs — are more prone to bone fractures.

Zinc and collagen matrix

Zinc is a critical trace element, essential for bone development and bone homeostasis — a delicate balance between bone resorption and bone formation. In a literature review on the topic of zinc and bone health, researchers at the Medical University of Warsaw, in Poland, highlighted that zinc plays an important role in the synthesis of collagen matrix, mineralization and bone metabolism[7].

The authors noted how zinc can enhance differentiation of bone-forming cells known as osteoblasts, potentially leading to greater bone tissue formation. Zinc was also found to help protect osteoblasts from oxidative stress caused by free radicals.

In the review, the authors reported that zinc is also known to increase the activity of alkaline phosphatase (ALP), an enzyme involved in bone metabolism. Increased ALP activity is associated with osteoblastic activity, mineralization and bone remodeling. Zinc further contributes to bone strength by enhancing collagen synthesis and cross-linking.

These processes are crucial for maintaining bone integrity, as well as the toughness and strength required to resist fractures. It is noted in the review that women with osteoporosis had lower zinc content in their bones compared with other women.

Manganese and type I collagen

Another trace mineral, manganese, has recently emerged as a key element affecting bone integrity and function. As with magnesium, most of the body's manganese is stored in the bones.

A 2024 review conducted by researchers at West Kazakhstan Marat Ospanov Medical University in Aktobe underscored the critical role that manganese plays in bone mineralization, and the formation of cartilage and collagen especially by promoting secretion of type I collagen, the most abundant collagen in bone matrix[8]. They also highlighted the dual impact of manganese on osteoblasts and osteoclasts. Manganese stimulates the growth of osteoblasts, promoting bone growth. Conversely, it inhibits the formation of osteoclasts — which break down old or damaged bone — reducing the rate of bone resorption.

Recent research detailed in the review also suggests that manganese ions can help to enhance bone regeneration while also preserving bone mass. Manganese may also help to promote bone growth by increasing both the activity of genes linked to bone formation, and the rate at which collagen is produced and deposited. "Manganese affects hormones involved in bone regeneration and the activity of enzymes involved in bone metabolism," the authors write.

All these studies indicate that magnesium, zinc, manganese, copper and other minerals present in trace amounts play crucial roles in collagen formation, bone mineralization and bone metabolism.

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铜与骨胶原交联

铜是维持骨骼健康的另一种重要矿物质，体内约 50% 的铜贮存在骨骼和骨髓中。铜对于赖氨酸氧化酶活性至关重要，而赖氨酸氧化酶能催化骨胶原纤维的交联。这个交联过程可增强骨胶原基质的稳定性，对骨骼完整性和骨韧性都很关键。

铜含量过高或过低都会影响骨骼健康。2024 年，位于沈阳的中国医科大学发表的一项研究指出，低铜饮食与年龄相关性骨质疏松症以及低骨密度相关[6]。作者指出，铜缺乏会破坏骨胶原纤维之间的交联，降低骨强度。铜缺乏还会导致负责骨吸收的破骨细胞异常活化，加速骨质流失，增加骨骼脆性。

尽管铜过量相对少见，但其过度积累也许会引发氧化应激，干扰骨代谢，进而可能损害骨骼外层结构，影响骨强度。例如，在患有威尔逊氏病（Wilson's disease）这种罕见遗传病的人群中，铜会在多个器官中过度沉积，使他们更易发生骨折。

锌与骨胶原基质

锌是维持骨发育和骨稳态的一种关键微量元素——骨稳态是指骨吸收和骨形成之间的微妙平衡。波兰华沙医科大学在一篇关于锌与骨骼健康的综述中指出，锌在骨胶原基质合成、矿化、骨代谢中都发挥了重要作用[7]。

作者指出，锌可以增强成骨细胞的分化，从而促进骨组织形成。研究还发现，锌有助于保护成骨细胞免受自由基引起的氧化应激。

作者在该综述中进一步指出，锌能增强碱性磷酸酶（ALP）的活性。ALP 是一种参与骨代谢的酶，其活性增强与成骨细胞活跃度、矿化以及骨重塑相关。此外，锌还能促进骨胶原形成和交联，进一步增进骨强度。

这些过程对于维持骨完整性以及抵抗骨折所需的骨韧性和骨强度都很重要。该综述指出，患有骨质疏松症的女性的骨骼锌含量比其他女性更低。

锰与 I 型骨胶原

另一种微量矿物质锰近年来也被发现是影响骨骼完整性和功能的重要元素。与镁一样，人体中的大部分锰也储存在骨骼中。

2024 年，哈萨克斯坦西部医科大学的研究人员发表的一篇综述强调了锰在骨矿化、软骨形成和骨胶原形成中的关键作用，尤其是锰能促进 I 型骨胶原的分泌——I 型骨胶原是骨基质中最丰富的胶原[8]。他们还解释了锰对成骨细胞和破骨细胞的双重影响。锰能刺激成骨细胞的生长，促进骨骼发育，反过来，锰也能抑制破骨细胞的形成——破骨细胞负责分解旧的或受损的骨骼——从而降低骨吸收速率。

该综述中的最新研究还表明，锰离子能够增强骨再生，同时维持骨量。此外，锰还可以通过提高骨形成相关基因的活性，以及加快骨胶原的形成和沉积速度，进一步促进骨骼生长。文章指出，"锰会影响参与骨再生的激素及骨代谢相关酶的活性。"

以上研究表明，镁锌锰铜等潜在矿物质在骨胶原形成、骨矿化和骨代谢中发挥了不容忽视的作用。

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