nature insight

bone and cartilage

Cover illustration

Inset, lumbar vertebrae of a newborn mouse stained with alcian blue (cartilage) and alizarin red (bone). Background, chondrocytes in the phalange of a newborn mouse; blue coloration signals expression of 'digit genes' of the HoxD gene cluster. (Courtesy of Marie Kmita, Jozsef Zakany and Denis Duboule.)

Bone and cartilage comprise the skeleton — a finely patterned structure that provides mobility, protection of vital organs, and housing of the bone marrow. Because bones are the most inert and enduring of our mortal remains, it is surprising what dynamic and highly tuned organs they are during life, as they must simultaneously balance growth to achieve strength and resilience, and repair without overgrowth.

This balance is achieved by bone remodelling — an interplay of mineral deposition and resorption by specialized bone cells, which is constantly occurring on a microscopic scale throughout our bodies. An imbalance in remodelling is what leads to the debilitating loss of bone mass called osteoporosis, a condition that is estimated to afflict 200 million women world wide, and often leads to immobility and death. This and other diseases of bone and cartilage are responsible for a large portion of healthcare expenditures in developed countries — US\$14 billion is spent annually on treating osteoporotic fractures in the United States alone.

These statistics emphasize the need for a better understanding of both the fundamentals of skeletal biology and the pathologies associated with it, which in addition to osteoporosis, include birth defects, genetic diseases, and rheumatoid and osteoarthritis. Indeed, as human life is prolonged by biomedical advances, the weak link in our health and longevity may become the strength of our bones and mobility of our joints.

This month's Insight presents a collection of articles that explore some key elements of skeletal biology, including how skeletal structures are patterned and bones and cartilage develop, the influence of genetics over human skeletal biology, the signalling pathways and transcription factors that control bone mass, and the pathogenic mechanisms of rheumatoid arthritis.

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overview:

- 316 The complexities of skeletal biology**
G. Karsenty

review articles:

- 319 Deciphering skeletal patterning: clues from the limb**
F. V. Mariani & G. R. Martin
- 326 Cranial skeletal biology**
J. A. Helms & R. A. Schneider
- 332 Developmental regulation of the growth plate**
H. M. Kronenberg
- 337 Osteoclast differentiation and activation**
W. J. Boyle, W. S. Simonet & D. L. Lacey
- 343 The genetic basis for skeletal diseases**
E. Zelzer & B. R. Olsen
- 349 Control of osteoblast function and regulation of bone mass**
S. Harada & G. A. Rodan
- 356 Evolving concepts of rheumatoid arthritis**
G. S. Firestein

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