

Invited Speaker Abstract

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Building new bone - does the osteoblast do all the work?

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New bone can be built by two mechanisms. Intramembranous bone formation involves the condensation of mesenchymal progenitor cells, their differentiation into osteoblasts, deposition of collagen matrix and ultimately woven bone formation. This woven bone is then remodelled into lamellar bone by sequential osteoclastic resorption and further osteoblastic bone deposition. The second mechanism is endochondral ossification that occurs through a cartilaginous intermediate, laid down by chondrocytes, it is subsequent remodelled by osteoclasts/chondroclasts and osteoblasts to form lamellar bone. It is apparent from this brief overview that osteoblasts, while clearly the workhorse of new bone formation, require assistance. The paradigm of osteoclast to osteoblast coupling in bone remodelling has strong in vitro and in vivo experimental support, but the specific coupling molecule(s) have been elusive. This coupling paradigm provides a logical mechanism for achieving balanced site-directed bone remodelling and maintenance of adult bone mass. However it does not explain the initial phase of intramembranous bone deposition or how bone modelling is coordinated. Several animal models have displayed discordance in osteoclast-osteoblast coupling, indicating that there maybe another cellular participant in the regulation of new bone formation. We have identified a population of resident tissues macrophages within osteal tissues (OsteoMacs), which form a striking, canopy structure over both sites of bone modelling and bone remodelling. We propose that OsteoMacs, like their related but distinct cousins osteoclasts, are also able to modulate osteoblast function and that they direct osteoblast activity during bone modelling and perhaps also participate in the late stages of bone remodelling.