

Mammalian target of rapamycin (mTOR) functions mainly in the form of two complexes, namely mTORC1 and mTORC2, which are distinct in their unique components, raptor and rictor. Here, we focused on bone phenotypes in mice with a specific deletion of *rictor* using a *Cre* recombinase gene whose expression was driven by the promoter of *osteocalcin*. All procedures involving mice were approved by the Institutional Animal Care and Use Committee of the local admin. DXA analysis showed a significant reduction in BMD of the *Rictor<sub>ob</sub><sup>-/-</sup>* mice (53.5mg/cm<sup>2</sup> vs 59.3mg/cm<sup>2</sup>, P<0.001). Furthermore, micro-computed tomography, histomorphometric, and molecular biological analyses revealed a marked impairment of the cortical bone growth, as well as minor changes in trabecular bone, of the *Rictor<sub>ob</sub><sup>-/-</sup>* mice. Cortical tissue mass (1138.17mg/ccmHA vs 1179.52 mg/ccmHA, P<0.01) and thickness (162.4  $\mu$ m vs 193.6  $\mu$ m, P<0.001) of the femoral mid-shaft were dramatically reduced, with unusual increases in porosity (0.69% vs 0.36%, P<0.01) and marrow area (0.98mm<sup>2</sup> vs 0.91mm<sup>2</sup>, P<0.05) by micro-CT. These changes were associated with significantly decreased bone mechanical properties of the femurs as reflected by reduced peak load (15.03N vs 20.54N, P<0.001) and stiffness (43.47N/mm vs 58.98N/mm, P<0.001). Thinner trabeculae were found in the lumbar spine (25.81  $\mu$ m vs 33.22  $\mu$ m, P<0.001) with relatively normal structural indices of trabecular numbers and separation by histomorphometry. However, there were no significant changes in the trabecular bone of the distal femur by micro-CT. A lower rate of bone turnover was observed, as the consequence of the decreased individual osteoblast activity and osteoclast number and activity. Furthermore, osteoblast differentiation was reduced, with down-regulation of mTORC2 signaling activity as shown in primary cultures of osteoblasts that did not contain *rictor*. In conclusion, expression of *rictor* in osteoblasts is essential for the maintenance of normal bone mass, especially for the normal accrument of cortical bone.