Bone mass and strength achieved at the end of the growth period, simply designated as "Peak Bone Mass (PBM)", plays an essential role in the risk of osteoporotic fractures occurring in adulthood. It is considered that an increase of PBM by one standard deviation would reduce the fracture risk by 50%. As estimated from twin studies, genetics is the major determinant of PBM, accounting for about 60 to 80% of its variance. During pubertal maturation, the size of the bone increases whereas the volumetric bone mineral density remains constant in both genders. At the end of puberty, the sex difference is essentially due to a greater bone size in male than female subjects. This is achieved by larger periosteal deposition in boys, thus conferring at PBM a better resistance to mechanical forces in men than in women. Sex hormones and the IGF-1 system are implicated in the bone sexual dimorphism occurring during pubertal maturation. The genetically determined trajectory of bone mass development can be modulated to a certain extent by modifiable environmental factors, particularly physical activity, calcium and protein intakes. Prepuberty appears to be an opportune time to modify environmental factors that impinge on bone mineral mass acquisition.

Keywords
Bone growth, peak bone mass, genetic determinants, sex hormones, IGF-1, physical activity, calcium intake, protein intake.