T2DM is characterized by hyperglycaemia, which results from a progressive deterioration of insulin secre–tory ?– cell function, typically combined with varying degrees of insulin resistance.Evidence suggests that a relationship exists between ageing and T2DM at a biological level: a number of studies in humans have shown that both diabetes melli–tus and ageing shorten telomere length38 (Box 1) and that T2DM induces premature cellular senescence(39) However, the nature of this relationship requires fur–ther study to understand if the biological processes involved in ageing drive T2DM pathology or if diabetes increases the rate of biological ageing.Excess visceral and ectopic (intramuscular and hepatic) adiposity decreases insulin sensitivity by producing the adipokines and cytokines that impede the pathways of insulin action downstream of the insulin receptor, such as tumour necrosis factor, and low–grade inflamma–tory factors such as C– reactive protein29.Impaired nutrient metabolism and an age–related decline in mito–chondrial function also link ageing and insulin resis–tance, although the mechanistic details remain to be clarified (Box 1).Age– associated blunting of insulin– mediated glucose uptake is linked with the progressive deterioration of the structure and function of skeletal muscles.In addition, both ageing and obesity are associated with an increased population of macrophages within adipose tissue, a decreased num–ber of regulatory T cells and a reduced self– renewal of mesenchymal progenitor stem cells, thereby promoting metabolic dysregulation and inflammation31.Underlying mechanisms include mitochondrial dys–function, increased low–grade inflammation, intramyo–cellular lipid accumulation and oxidative stress as well as the accumulation of senescent cells and decreases in autophagic capacity and enzymatic activity During skeletal muscle ageing, pro– inflammatory path–ways become activated.For example, ?– cell senescence and reduced ?– cell sensitivity to glucose dur–ing ageing increase the susceptibility to T2DM through inadequate compensation for insulin resistance(18,19.) The detrimental effects of ageing on cellular path–ways of insulin action and glucose metabolism are modest when age–related changes in body composition are considered(20,21).Furthermore, the number of mitochondria is reduced and their oxidative capacity is decreased due to the reduced activity of antioxidant enzymes, which leads to the intracellular accumulation of reactive oxygen species and increased levels of oxi–dative stress in skeletal muscle.1)14,15.1)(16,17).1)17,22,23.