FEATURES

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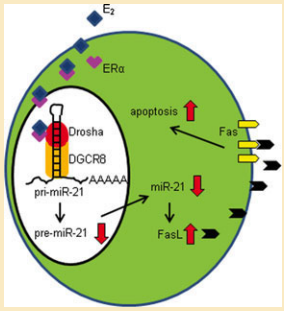

Estrogen inhibits osteoclastogenesis and induces osteoclastic apoptosis; however, the molecular mechanisms remain controversial. Recently, a group has demonstrated that osteoclasts are a direct target for estrogen because estrogen stimulates transcription of the Fas Ligand (FasL) gene in osteoclasts, which in turn causes cell death through an autocrine mechanism. In contrast, other groups have shown that the cells are an indirect target for estrogen because estrogen fails to stimulate the transcription of that in osteoclasts. Thus, two quite different molecular mechanisms have been suggested to explain the effects of estrogen in osteoclastic apoptosis. Here it is shown that the proapoptotic effect of estrogen during osteoclastogenesis is regulated by a posttranscriptional increase in FasL production by down-regulated microRNA-21 (miR-21) biogenesis. Previously, it was reported that miR-21 is highly expressed in osteoclastogenesis. It was found that estrogen down-regulates miR-21biogenesis so that FasL, the targets of miR-21, protein levels are posttranscriptionally increased that induce osteoclastic apoptosis. Moreover, the gain-of-function of miR-21 rescued the apoptosis. In addition, there was failure to detect estrogen-enhanced FasL levels at mRNA levels. Thus, osteoclastic survival is controlled by autocrine actions of FasL regulated by estrogen and miR-21 plays a central role during estrogencontrolled osteoclastogenesis.

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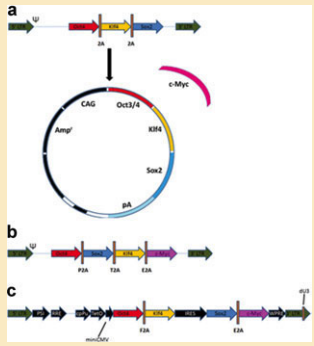

The first successful attempt to reprogram somatic cell into embryonic-like stem cell was achieved on 2006. Since then, it had sparked a race against time to bring this wonderful invention from bench to bedside but it is not easily achieved due to severe problems in term of epigenetic and genomic. With each problem arise, new technique and protocol will be constructed to try to overcome it. This review addresses the various techniques made available to create iPSC with problems hogging down the technique.

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