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核孤儿受体TR3/nur77与一种新细胞凋亡机制

Orphan Receptor TR3/nur77 and a New Apoptosis Mechanism

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英文关键词：TR3/nur77　orphan receptor　immediate-early gene　mitochondria　apoptosis　translocation

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中文摘要：

核孤儿受体TR3/nur77是一种立刻早期基因（immediate-early gene）的产物，与固醇类激素受体结构相似，是核受体超家族的重要成员之一，可被多种生长因子或凋亡诱导剂诱导表达，具有复杂的生物学功能，涉及细胞增殖、分化发育和凋亡过程.最近对其诱导凋亡机制的研究取得了重大进展，发现当细胞受到凋亡诱导剂刺激后，TR3基因表达升高，其产物从细胞核移位至线粒体膜，引起细胞色素c释放，从而导致细胞凋亡.即TR3的转录激活功能和诱导凋亡功能是由其不同的亚细胞定位结合所决定的，其诱导凋亡过程与其对基因的反式激活功能无关.核转录因子p53也具有类似情况.这种核转录因子由细胞核移位至细胞浆并发挥生物学功能的调控方式是一种新模式，可能具有重要的生物学意义.

英文摘要：

Orphan receptor TR3/nur77, a member of nuclear receptor superfamily, is a product of immediate-early gene which is expressed rapidly after induced by several proliferation factors such as EGF, FGF, NGF, PDGF or phorbol ester, and different apoptosis inducers. It is similar in structure with the members of steroid/retinoid receptor superfamily that all have a DNA-binding region of two zinc-finger and a ligand-binding region. As a transcription factor its complex functions are involved in proliferation, differentiation and apoptosis of cells. Recently there is an important development about the TR3 receptor in the mechanism of inducing apoptosis. After treated by different apoptosis inducers TR3 expressed increasingly, and it is found surprisingly that TR3 translocates from the nucleus to mitochondria to induce cytochrome c release and apoptosis. That is, mitochondria targeting of TR3 but not its DNA binding and transactivation is essential for its proapoptotic effect. This is a new model may be a very significant pathway in the regulation of cell signal transduction especially in the apoptosis.

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