## Introduction

Apoptosis is a form of programmed call death in multicellular organisms. It involves a series of biochemical events that leed to a varriety of morphological changes including blebbing, loss of cell membrane asymmetry and attachment, cell shrinkage, nuclear fragmentation chromatin condensation, and chromosomal DNA fragmentation (*Leist*, 2001).

Multiple triggers e.g viral infection, cell damage, starvation, ionizing radiation, or toxic chemicals can lead to a cell undergoing apoptosis by different pathways, namely death receptors [ fas ( CD 45) , TNFR 1, TRAIL], mitochondrial (cellular stress) causes Bax, Bak and / or Bid to bind to mitochondria, cytotoxic cells (T cell and NK cells).inject granzyme B which activates caspase 3 and DNA damage is detected by p53, resulting in activation of apoptosis (*Hengartner et al.*, 1999).

Apoptosis is essential for maintaining stable cell population by ensuring that the rate of new cell production is balanced by an equal rate of cell death. This is particularly true in the haemopoietic system (*Fadeel and Orrenius*, 2005).

Cellular suicide also has a defensive function: cells that are infected by a virus or other intracellular pathogens may kill themselves, which help limit spread of infection (*Fadeel and Orrenius*, 2005).

Apoptosis also plays a pivotel role in immune tolerance by inducing B and T cell apoptosis of autoraactive cells (*Bouillet et al.*, 2002).



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Apoptosis plays a role in preventing cancer, if a cell is unable to undergo apoptosis, it can continue dividing and develop into a tumor (*Johnstone et al.*, 2002).

Defective apoptosis is incriminated in the pathogenesis of many diseases e.g tumors, autoimmune diseases, infections, diabetes rnellitus and many neurological diseases including Alzheimer's disease Huntington's disease, Parkinsonism, infantile spinal muscular atrophy and amyotrophic lateral sclerosis (*Thompson*, 1995).