The structural stability of the adult human is maintained by balancing the growth of new cells (mitosis) with the death (apoptosis) of others. In diseases such as AIDS and cancer, this fine regulation is lost, often as a result of abnormal apoptosis. In cancer, to make things worse, the effectiveness of chemotherapy is limited by the intrinsic resistance of cancer cells to apoptosis. The molecular mechanisms of apoptosis are therefore of extreme importance to identify new therapeutic targets for the treatment of these diseases.

A vast degree of information is now available to identify all the molecular components involved in apoptosis. Very recently this has been further increased by the identification of the human genome. However, there are limitations to a purely genomic approach. Proteins are sociable, and their effects are not produced in isolation, but through interaction with other proteins. In clinical situations where excess apoptosis occurs, such as in AIDS and neurodegeneration, understanding of caspase-IAP interactions may open new therapeutic opportunities for manipulating the apoptotic process. Genomics may have provided the notes on the piano keyboard: it is up to proteomics to show us how biological symphonies are constructed.

Cellular effector molecules, whose nature depends on the death stimulus, can accumulate to cause the permeabilization of the mitochondrial membrane. Once the stimulus has been delivered to the mitochondrial membrane, the permeabilization occurs through a limited set of mechanisms, the fate of the cell has been decided and leakage of proteins normally confined to mitochondria determines the loss of vital functions and/or activation of degrading enzymes such as nucleases and proteases.

Thus, tissue-protective strategies that regulate apoptosis will minimise cell loss with a corresponding reduction in disease severity in cases such as in stroke patients. Understanding the molecular mechanisms that regulate apoptosis in cells and tissues is therefore essential for the design of new treatments.

These and other mechanisms will be discussed by outstanding researchers from different European top-ranking institutes in view of their importance in our understanding, as well as the development of novel therapeutic strategies for cell demise or death resistance. For example, Professor Pierluigi NICOTERA, Director of the MRC Toxicology Center in Leicester (GB), will present his work on nitric oxide in regulating neuronal death. Doctor Henning WALCZAK, Head of the Apoptosis Laboratory at the DFFZ in Heidelberg (D), will introduce the Death Receptors and their regulation of apoptosis.

University of Calabria
DEPARTMENT OF PHARMACOBIOLOGY

V Workshop on

Apoptosis in Biology and Medicine

Arcavacata di Rende
May 29th, 2001
Aula Magna

Under the auspices of The

Calabria Region
University of Calabria, Cosenza
University of Catanzaro "Magna Græcia"

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Programme

Welcome address by G. Latorre and S. Venuta

Chairman
A. Quattrone (Italy)

8.45-9.00 G. Rotilio (Italy)
Funding biomedical research in Italy: the role of the CNR

Chairpersons
D. Boraschi (Italy) & C. Riccardi (Italy)
Invited Lecture on
IL-1 in pain and sickness behaviour
S. Poole (UK)

Chairmen
S. Andò (Italy) & P. Nicotera (UK)

9.30-9.55 P. Nicotera (UK)
Mitochondria as a control for apoptosis

9.55-10.20 G. Melino (Italy)
Apoptosis mechanisms by p73 and possible mitochondrial involvement

10.20-10.45 G. Bagetta (Italy)
Mitochondrial IL-1β: role in neuronal death

10.45-11.10 C. Riccardi (Italy)
Glucocorticoid hormones and regulation of cell death

11.10-11.25 Coffee Break

Chairmen
G.F. Di Renzo (Italy) & D. Borgese (Italy)

11.25-11.50 G. Rotilio (Italy)
Redox and mitochondrial regulation in apoptosis

11.50-12.15 M. Piacentini (Italy)
Transglutaminase and regulation of mitochondria

12.15-12.35 B. Bruene (Germany)
NO and apoptosis: Upstream and downstream of mitochondria

12.35-13.00 H. Mehmet (UK)
Induction of apoptosis or necrosis in neuronal PC12 cells depending on the redox state of nitric oxide

13.00-13.30 General Discussion

13.30-15.30 Lunch break

Chairmen
R.A. Knight (UK) & H. Walczak (Germany)

15.30-15.55 H. Walczak (Germany)
TRAIL-indiced apoptosis: biochemical pathway, physiological role and therapeutic potential

15.55-16.20 C. Turco (Italy)
A.I.R., a novel gene that regulates apoptosis

16.20-16.45 C. Indiveri (Italy)
Switch from carrier to channel properties of mitochondrial transport systems

16.45-17.10 V. De Laurenzi (Italy)
Function of p73 family

17.10-17.35 A. Spinedi (Italy)
Reversal of drug resistance in HepG2 cells by PDMP

17.35-18.00 E. Clementi (Italy)
Cross talk between NO and ceramide in the control of apoptosis

18.00-18.30 General Discussion