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.

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DEPARTMENT OF PHARMACOBIOLOGY

VII Workshop on

Apoptosis in Biology and Medicine

Basic and Therapeutic Aspects of Brain Ischemia

Aula Magna, University of Calabria

29th - 30th April, 2003

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Programme

Tuesday 29th April

17.15 Welcome address

17.30 Opening Lecture

Martin Raff (UK)
Self-destruct programs in neurons

18.30 Welcome cocktail and Poster viewing

Funding biomedical research in Italy: the role of the CNR

Wednesday 30 April

Chairpersons:
G. Bagetta (Rende) & D. Knight (London)

9.00-9.30 *G. Melino (Leicester, UK)*
Thirsty of knowledge in cell death research

9.30-10.00 *P. Nicotera (Leicester, UK)*
Apoptosis and necrosis: new vistas

10.00-10.30 *F. Cattabeni (Milan)*
Synaptic signals for neuronal damage/protection

10.30-11.00 *N. B. Mercuri (Rome)*
Synaptic plasticity under brain ischemia

11.00-11.30 *Coffee Break*

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

11.30-12.00 *M. Marcoli (Genoa)*
Vasicular-like glutamate release from human and rat cerebral cortex slices during ischemia

12.00-12.30 *L. Annunziato (Naples)*
Oxidative stress and brain ischemia

12.30-13.00 *R. G. Knowles (Stevenage)*
Inhibitors of NOSs in brain ischaemia

13.00-13.30 *F. Moroni (Florence)*
Poly(ADP)ribose polymerase and neuronal damage

13.30-13.45 *General Discussion*

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