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Pancreatic Disorders & Therapy



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Editor for
Pancreatic Disorders & Therapy



BIOGRAPHY

Dr. Jiazhi Sun has been trained in the field of basic medical science and molecular biology and received M.D. degree from Peking University in 1986. After completing his residency and fellowship training at Peking University in 1990 and graduate studies on targeting Q-binding protein into yeast mitochondria at West Virginia University in 1994, he received two-terms postdoc training at the University of Pittsburgh and H. Lee Moffitt Cancer Center in the field of Pharmacology, Biochemistry and Drug Discovery from 1994 to 2003.

RESEARCH INTERESTS

Dr. Jiazhi Sun focuses on Ras and ER signaling study, HDL-Receptor RNA alternative processing, and K-Ras, GPCR, BH3 protein and VDR/ER drug discovery targeting by utilizing Target-Guided Synthesis (TGS) approach and conventional intervention. Dr. Sun has more than 20-year professional experience in academic field including Drug Discovery, Signal Transduction, Apoptosis, Cancer Biology and Cardiovascular field, also leading collaborative Institutional and Pharmaceutical Industry rational design/targeted-therapeutic approach and development of FTI, GGTI and Kinase inhibitors (tyrosine, serine/threonine and sphingosine) and nuclear hormone receptor mimics as well as BH3-mimics as drug candidates and “Chemical-Genetics” tool/model. Recently Dr. Sun’s lab has been focus on scFv and ADC development in terms of targeting EGFR/K-Ras in Cre-activated K-rasG12D transgenic mouse models.

RECENT PUBLICATIONS

- Prediction of the likelihood of drug interactions with kinase inhibitors based on in vitro and computational studies.
- Prediction of drug-target interactions for drug repositioning only based on genomic expression similarity.
- The BH3 alpha-helical mimic BH3-M6 disrupts Bcl-X(L), Bcl-2, and MCL-1 protein-protein interactions with Bax, Bak, Bad, or Bim and induces apoptosis in a Bax- and Bim-dependent manner.
- Apogossypol derivatives as antagonists of antiapoptotic Bcl-2 family proteins.
- Bcl-XL-templated assembly of its own protein-protein interaction modulator from fragments decorated with thio acids and sulfonyl azides.
- Structure-based design of imidazole-containing peptidomimetic inhibitors of protein farnesyltransferase.
- Cucurbitacin Q: a selective STAT3 activation inhibitor with potent antitumor activity.

What is apoptosis?

There are two ways that a cell can die: necrosis and apoptosis. Necrosis occurs when a cell is damaged by an external force, such as poison, a bodily injury, an infection or getting cut off from the blood supply (which might occur during a heart attack or stroke). When cells die from necrosis, it's a rather messy affair. The death causes inflammation that can cause further distress or injury within the body.

Apoptosis, on the other hand, is relatively civil, even though it may not sound so at first -- it's when a cell commits suicide. How is that better than necrosis? For one thing, the cleanup is much easier. It's sometimes referred to as programmed cell death, and indeed, the process of apoptosis follows a controlled, predictable routine.

When a cell is compelled to commit suicide (we'll get to the triggers for apoptosis in just a minute), proteins called caspases go into action. They break down the cellular components needed for survival, and they spur production of enzymes known as DNases, which destroy the DNA in the nucleus of the cell.

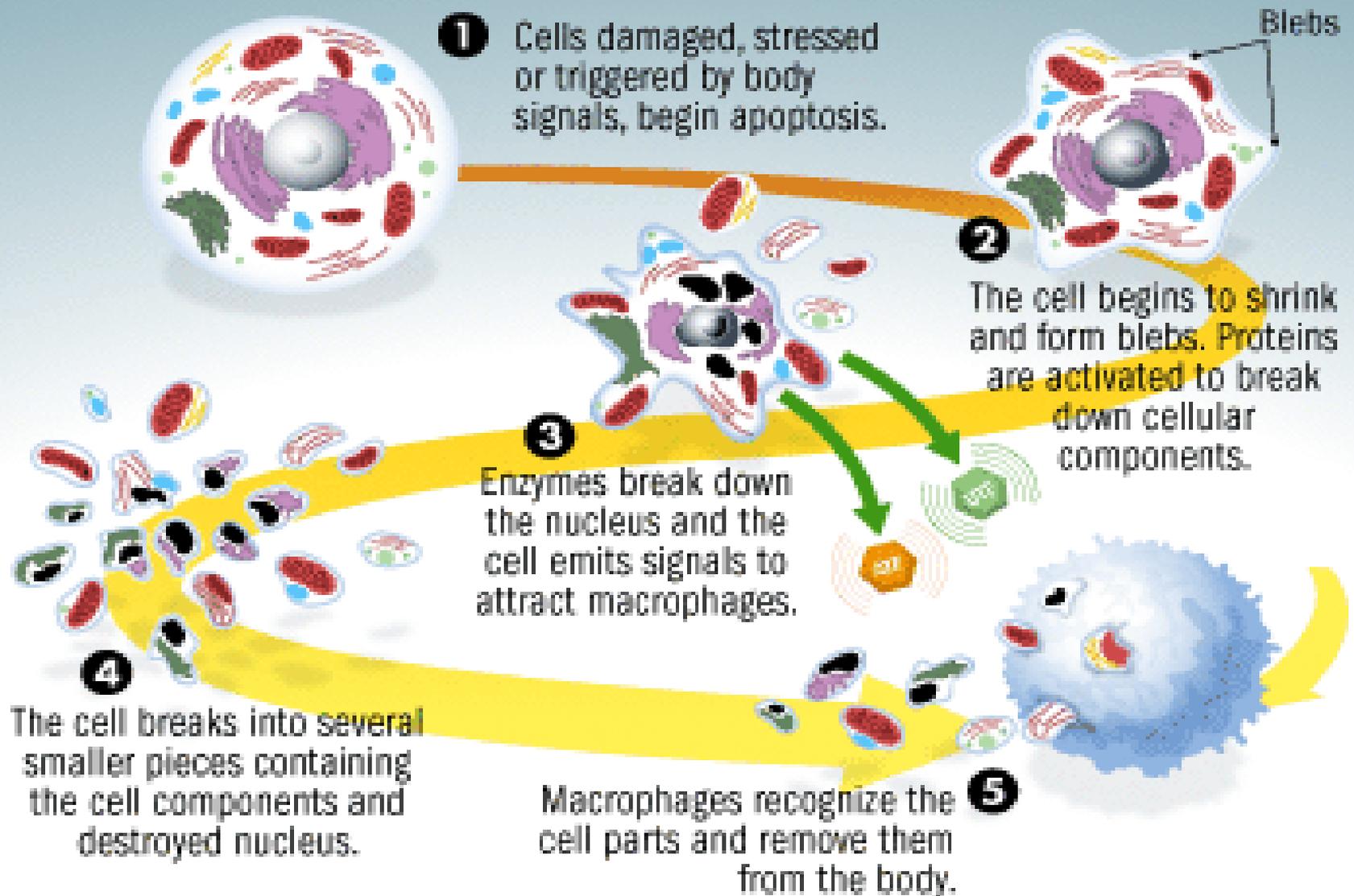
It's like roadies breaking down the stage in an arena after a major band has been through town. The cell shrinks and sends out distress signals, which are answered by vacuum cleaners known as macrophages. The macrophages clean away the shrunken cells, leaving no trace, so these cells have no chance to cause the damage that necrotic cells do.

Apoptosis also differs from necrosis in that it's essential to human development. For example, in the womb, our fingers and toes are connected to one another by a sort of webbing. Apoptosis is what causes that webbing to disappear, leaving us with 10 separate digits. As our brains develop, the body creates millions more cells than it needs; the ones that don't form synaptic connections undergo apoptosis so that the remaining cells function well. Programmed cell death is also necessary to start the process of menstruation.

Scientists are trying to learn how they can modulate apoptosis, so that they can control which cells live and which undergo programmed cell death. Anti-cancer drugs and radiation, for example, work by triggering apoptosis in diseased cells. Many diseases and disorders are linked with the life and death of cells -- increased apoptosis is a characteristic of AIDS, Alzheimer's and Parkinson's disease, while decreased apoptosis can signal lupus or cancer. Understanding how to regulate apoptosis could be the first step to treating these conditions.

Apoptosis Process

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- **Journal of the Pancreas**
- **Journal of Gastrointestinal & Digestive System**

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