

COSMETISCOPE



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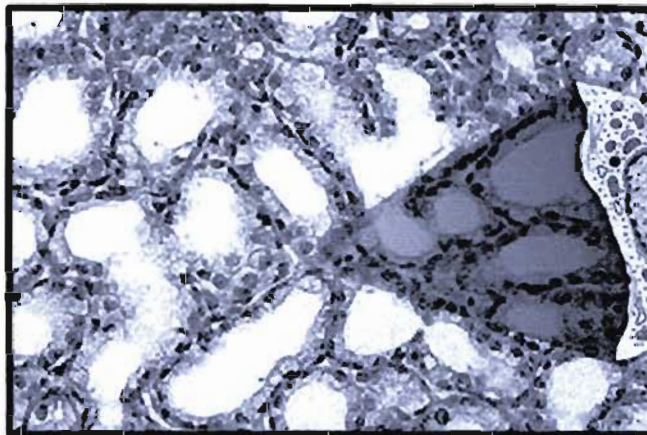
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Natural Aging, Suicide or Terminal Illness - The Three Possible Paths for Cell Death

By: Nava Dayan Ph.D., Lipo Chemicals Inc.

The human perception of death was fascinating to me for as long as I can remember. As a high school major in literature and arts the title I chose for my graduation thesis was: "The Description of the Devil in Christian Art in the Middle Ages". In preparation for writing the thesis I read the Old and New Testaments of the Bible and external books and



tried to filter the events that led to trends in artistic creations of heaven and hell. After months of work I concluded what was already known but seemed to be a discovery for me as a teenager: "there is nothing new under the sun". "Memento mori" (from Latin: 'remember that you are mortal') is not typical to the Middle Ages. The modern phrase: "life is a lethal disease," means exactly the same.

Later on at the School of Pharmaceutics I tried to understand the connection between the ways our body gets old and our perception of life. I am still exploring...

Every cell in our body has properties that can be reflected in the entire organ and hence can affect the function of our entire body. One of the most deadly diseases, cancer, begins with a mutation in a single cell.

There are three possible ways for a cell to die:

senescence (natural aging), apoptosis (suicide) or necrosis (terminal illness). All three processes will come to a stage where they will be irreversible.

Cell senescence is a proof that we are "programmed to die". Normal cells cannot divide indefinitely because they are genetically programmed for a set proliferative life span. After going through a finite number of cell divisions the

cell enters a state of irreversible growth. This is a dominant, genetically controlled process that constitutes a tumor suppressive mechanism. Recent findings however suggest that specific extrinsic factors, including ones that are considered to be "normal" can prematurely induce the transition to a senescent cell phenotype. How do we identify a cell that has reached old age?

Senescent cells are not capable of dividing and can exhibit changes in form and function. They

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Natural Aging, Suicide or Terminal Illness

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can still allow metabolic processes. As we age we accumulate senescent cells. This process actually controls the induction and progression of cancer. Some of the genes that trigger uncontrolled proliferation in cancer are turned off when the cell is senescent. Compounds that induce or delay senescence can have anti-tumor or anti-aging properties. It is yet unclear if delaying senescence will directly induce cancer.

Apoptosis is the process by which a cell "decides" to die. It is also called: "programmed cell death" because the pattern of events that leads to it is very methodical. Induction of apoptosis can occur when it is needed for proper development of the tissue, such as the sloughing off of the inner lining of the uterus in menstruation. In the skin, keratinocytes "commit suicide" by losing their nucleus, becoming corneocytes and then being exfoliated. Ceramides are known to play a role in keratinocyte apoptosis and in the renewal of the tissue. Apoptosis can also happen when a cell presents a threat to the tissue, for example when it is infected by virus or has a damaged DNA. The cell will induce apoptosis either when there is a decline in the positive outside signaling or when it is receiving negative signals. Normal cells receive stimulation from growth factors or cytokines. When those are changing, declining or absent, the cell can enter into apoptosis. Negative signals can be the accumulation of reactive oxygen species that can come from UV light or toxic compounds.

When a cell enters apoptosis it shrinks, its surface becomes uneven and the chromatin (DNA and protein in the nucleus) is degraded. These processes are followed by the breakdown of the cell's "engine", the mitochondria that releases cytochrome C. The cell then breaks down to small fragments in which phosphatidylserine (a phospholipid that is normally hidden in the cell membrane) is exposed. The exposure of phosphatidylserine triggers phagocytosis by macrophages that in response secrete cytokines to inhibit further inflammation.

The third trigger for cell death, injury, can be either mechanical damage or exposure to toxic compounds. When it happens the cell's outside membrane and the membrane of its organelles lose their integrity. This allows water to penetrate the cell and as a result it swells. When the disrupted membrane can no longer hold its content, it leaks to the surroundings and causes inflammation in the tissue. Here again, our immune system is on call to engulf the inflammation triggering cell fragments and release mediators that will terminate the inflammation and induce restoration of the tissue.

Although senescence can inhibit apoptosis these processes are related and possibly synergistic. It was shown for example that epidermal keratinocytes taken from humans at ages 45 and up had

increased levels of apoptotic mediators and decreased levels of anti-apoptotic mediators. As we age the number of keratinocytes and corneocytes in the skin are reduced and their size is enlarged. As a result, the surrounding intercellular area is reduced. Since the intercellular lipids represent the major route for skin penetration and also for transepidermal water loss, these changes can lead to significant alterations in aged skin barrier properties.

One of the interesting findings about skin cells aging is the telomere-shortening phenomenon. Telomere is a region on the chromosome that has highly repetitive DNA. It functions as a disposable buffer by preventing the loss of useful genetic information and allows the DNA replication to be completed. Both intrinsic and photo related skin aging involve progressive telomere shortening. This is as a result of accumulation of reactive oxygen species.

As we all look for the best complete path to prevent skin aging and restore it we often tend to forget that the aging program is controlled from the DNA in the nucleus at the cellular level. We moisturize the skin, condition it, try and affect different enzymes and lipids, but the way to controlling it at its core program, the nucleus, is long. The mysteries of cellular aging are only beginning to disentangle and we should focus our efforts in understanding them.

Although some of us strive for perfection, accepting the fact that we

are designed with imperfections may make us realize that reaching the goal is impossible. Cells can "decide" to either die or live. If they decide to live they need to proliferate, differentiate or simply continue with their metabolic and signaling paths. All these processes require energy. Cell energy is provided by the mitochondria that also release reactive oxygen species that may accumulate and trigger apoptosis. Furthermore, when a cell divides it can accumulate replicative DNA damage.

If the cell does not die its chances to be defective are elevated with time. Defective cells may become malignant. If the cell does not die from aging, the body will suffer from cancer. Any way we look at it, living is making mistakes and those buy us a one-way ticket.

Recognizing the above and knowing the horror and fear from death that was seeded in the population of the Middle Ages, it was not difficult to understand why after this period in history we entered into the Renaissance, Baroque and Rococo. Enjoy the beauty and life while you can. 🌸

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