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請用中文敘述下列英文短文的大意。本試卷一共有三題，只能用規定內的字數(註明於每題前)來寫。專用名詞(有下標線者)可用原文書寫。

第一題: (50 字) (25%)

A new study published earlier this year showed dramatic reversal of aging in mice. The mice had been genetically modified to age rapidly by "knocking out" the gene that makes telomerase, the enzyme responsible for maintaining the telomeres (位於染色體尾端之端粒點). The genetically manipulated mice had short telomeres which caused premature aging with atrophy of the brain and spleen (脾臟), loss of sense of smell, and loss of fertility with testicular (睪丸的) atrophy.

The next step of the experiment was to give back the missing gene for telemerase and see if that would reverse all these signs of aging in the mice. For this next step, the aged mice were treated with a drug which served to "turn on" production of telomerase and lengthen the telomeres. This dramatically reversed the signs of aging with the aged mice surprisingly rejuvenated. Their shrunken brains, spleens and testes (睪丸) resumed normal size, and they regained their sense of smell. The aged infertile males once again became fertile, and fathered large litters.

第二題: (100 字) (35%)

A classic metaphor (比喻) in biology pictures an embryonic cell at the top of a hill. As the embryo develops, the cell rolls downhill into a series of branching valleys. Once a cell enters, say, the valley that leads to becoming a skin cell, it cannot suddenly change course and become a neuron. When developmental biologist came up with the image in the 1950s, the message was clear: Development is a one-way trip.

Not so. In the past decade, scientists have figured out how to push differentiated cells back up the hill and, perhaps even more surprising, directly from one valley to the next. By prompting a cell to overexpress a few genes, researchers can turn a skin or blood cell into a pluripotent cell: one that has regained the potential to become any number of cells in the body. In 2006, Dr. Shinya Yamanaka stunned the world when he showed that simply by adding extra copies of four genes to adult mouse cells, he could prompt them to become pluripotent. He called the resulting cells "induced pluripotent stem" cells (iPSCs).

Scientists are already using the technique to make cell lines from patients with hard-to-study diseases, and ultimately they hope to grow genetically matched replacement cells and tissues—perhaps even entire organs. The breakthrough offered a way around some of the sticky ethical and political issues that have dogged research with human embryonic stem cells, which are taken from early embryos. Suddenly, scientists had a source of human pluripotent cells free of special rules and regulations.

However, early reprogramming techniques did have several drawbacks. First, they permanently inserted the extra genes into the reprogrammed cell's genome. Although the genes seemed to turn back off once the cells were pluripotent, it wasn't clear how they might influence the cells' later behavior. Second, at least one of the genes Dr. Yamanaka used was known to trigger cancer, and indeed it soon became clear that mice grown from iPSCs frequently developed tumors. Finally, the process was inefficient, reprogramming only about one in 5000 of the treated cells.

(背面仍有題目,請繼續作答)

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第三題: (150 字) (40%)

When a kitchen knife slips while you're chopping vegetables, the body reacts swiftly (迅速的). White blood cells swoop (進攻)in and sterilize the injury, and the tissue-repair effort begins. This inflammatory response does have its downsides, causing swelling, redness, and pain. But there's no question that acute inflammation is a net positive, a response to trauma that evolved millennia (千年) ago to keep us alive and healthy.

A darker story began to emerge in the 1990s. Researchers studying at apparently unrelated diseases noticed that immune cells congregate (聚集) at disease sites. Atherosclerosis (動脈硬化), in which fatty plaques build up in the arteries, was among the first to make the list. In the 1980s, Dr. Russell Ross of the University of Washington, Seattle, saw macrophages in atherosclerotic tissue; these white blood cells are a hallmark (特徵) of inflammation. Slowly, as more people parsed arterial tissue, more came to agree that an inflammatory response was under way. There were T cells. There was interferon-γ, which the immune system produces as part of its inflammatory efforts.

Other conditions unrolled parallel story lines. In 1993, a group at Harvard University found that fat tissue in obese (肥胖) mice was churning (翻騰) out a classic inflammatory protein. Ten years later, back-to-back papers showed a correlation between macrophage infiltration of fat tissue in rodents and people and how obese they were. Newcomers to the inflammatory story include neurodegenerative diseases such as Alzheimer's (阿茲海默氏症-失智症的一種) and Parkinson's (巴金森氏症-神經退化疾病的一種). Here, it's murkier (混淆) whether inflammation is perpetuating disease or just along for the ride.

In most chronic illnesses for which inflammation has been fingered, it appears to drive ill health but not initiate it. In cancer, for example, papers published over the past decade suggest that tumors and inflammation dance together toward disaster: Tumors distort (扭曲)healthy tissue, setting off tissue repair, which in turn promotes cell proliferation and blood vessel growth, helping cancers expand. And although it's genetic mutations in tumor cells that initiate cancer, there's evidence that inflammation in surrounding tissue helps coax (哄) those cells along.

The surest way to prove that inflammation is driving any disease is by blocking it and testing whether that helps, and experiments are under way. In 2007, seventy patients received either a placebo or anakinra, a drug used occasionally to treat rheumatoid arthritis (類風濕關節炎) that blocks interleukin-1. Interleukin-1 is a proinflammatory cytokine, a protein that promotes inflammation; it's been found in beta cells from people with type 2 diabetes. The drug helped control the disease. Anakinra is not a good option for long-term diabetes treatment, so several companies are racing to develop alternatives.

Mediating inflammation in chronic diseases is a new frontier, its success still uncertain. But after inflammation eluded them for so long, researchers are chasing lead after lead, trying to stay a step ahead and discern when its fires need putting out.