For nearly all of human existence, people died young. Life expectancy improved as we overcame early death—in particular, deaths from childbirth, infection, and serious injury. By the nineteen-seventies, just four out of every hundred people born in industrialized countries died before the age of thirty. It was an extraordinary achievement, but one that seemed to leave little room for further gain; even *eliminating* deaths before thirty would not raise over-all life expectancy significantly. Efforts shifted, therefore, to reducing deaths during middle and old age, and, in the decades since, the average life span has continued upward. Improvements in the treatment and prevention of heart disease, cancer, and the like mean that the average sixty-five-year-old can expect to live another nineteen years—almost four years longer than was the case in 1970. (By contrast, from the nineteenth century to 1970, sixty-five-year-olds gained just three years of life expectancy.)

The result has been called the "rectangularization" of survival. Throughout most of human history, a society's population formed a sort of pyramid: young children represented the largest portion—the base of the pyramid—and each successively older age unit represented a smaller and smaller group. In 1950, children under the age of five were eleven percent of the U.S. population, adults aged forty-five to forty-nine were six percent, and those over eighty were one percent. Today, we have as many fifty-year-olds as five-year-olds. In thirty years, there will be as many people over eighty as there are under five.

Americans haven't understood the implications of these statistics. We keep maintaining the notion of retirement at sixty-five—a reasonable notion when those over sixty-five were a tiny percentage of the population, but completely unrealistic as they approach twenty percent. People are putting aside less in savings for old age now than they have in any decade since the 1930s. More than half of the very old now live without anyone else, and we have fewer

children than ever before — yet we give almost no thought to how we will live out our later years alone.

[設 問]

- (1) In 45 to 60 words, write a summary of the above in English.
- (2) In an English essay of 80 to 100 words, describe how you intend to face the situation discussed above.

Mosquitoes have been targeted by some of the world's most intense pesticide*1 programs and, as a result, have come up with many strategies for avoiding control. In 1989, 114 different species of mosquitoes resisted at least one insecticide*2. Many had developed resistance to more than one insecticide, especially to the powerful nerve poisons. Resistance comes from a number of different cell mechanisms that prevent the attachment of the insecticide to the insect's nerves. In some cases, an enzyme*3 called esterase*4 attaches to the insecticide before it gets to the nerve ending*5, blocking insecticidal action.

When there is a lot of insecticide around, this strategy works like shoveling the snow in front of your house during a heavy snowstorm—it's successful only if you have a lot of shovels. But these mosquitoes have a lot of chemical shovels (the esterase enzymes) to remove the insecticidal snow because, not so long ago, they invented a way to increase the production of their chemical shovel many times over.

Sometime, somewhere, while large quantities of nerve-poison insecticides were first being used, the esterase gene was duplicated*6 many times inside the cells of a mosquito. Many copies of a gene produce much more protein*7 than a single gene. This overproduction increases the amount of protective esterase, which can make even massive quantities of the insecticide harmless. The new strategy was extremely successful, and from its first recorded appearance in 1986 quickly spread around the world.

How did this mutation*8—i.e., the gene duplication—appear so suddenly and so widely? Two possibilities exist—that the mutation appeared several times independently or that it spread like lightning from one place. Careful research on DNA from resistant individuals of the mosquito Culex pipiens*9 from around the world tells us that the DNA surrounding the duplicated genes is identical in mosquitoes from California, Pakistan, Texas, and Egypt. Because we

expect such duplications rarely to happen independently in four different parts of the world, it seems that just one single mutation—a drastic one—caused esterase duplication in this species. Live adult mosquitoes fly internationally, hidden on airplanes, and easily leap across continents. So duplicated esterase genes soon went on a global tour, leaving offspring everywhere they stopped.

But evolutionary changes such as those observed in the mosquito, like contracts with the devil, carry a cost. A great amount of raw materials is needed to make overproduced esterase, and insects that needlessly make so much of the extra protein are selected against. Field studies show that, in the absence of insecticides, mosquitoes without the overproduction of esterases grow faster, survive longer, and reproduce better than the resistant types, and that areas without heavy pesticide use have fewer mosquitoes with the duplicated esterase genes. Natural selection against resistant individuals reduces their frequency in following generations—as long as the insecticides are not used.

Nevertheless evolution can run subtly, driven by selection to reduce the costs of insecticide resistance — and mosquitoes that pay the devil a discount price can thrive. For example, most mosquitoes make too much esterase and wastefully spread it throughout their bodies into tissues where insecticides have no effect. Other mosquitoes produce the protective protein only in their nerve cells, the tissue that needs protection most. This second group does much better than the first in growth rate, survival, and pesticide resistance.

Other kinds of mosquitoes also produce esterases in the intestine and the cuticle*10, where the insecticides enter the insect body. These may act like tiny organic robots attaching to the insecticide, making it harmless before it gets close enough to nerve cells to do any damage. This difference in the position in the body from which the esterase begins its defense is a continuing experiment in the evolution of increased pesticide resistance.

(注)

- * 1 , * 2 $\,$ pesticide, insecticide : a chemical used to kill insects
- *3 enzyme:酵素(触媒作用のあるタンパク質)
- *4 esterase:エステラーゼ(酵素の一種)
- * 5 nerve ending:神経終末
- * 6 duplicate(d): to make copies of
- *7 protein:タンパク質
- *8 mutation:突然変異
- *9 the mosquito Culex pipiens:アカイエカ
- *10 the intestine and the cuticle:腸と角皮

[設 問]

- (1) 下線部(1)を和訳しなさい。
- (2) 下線部(2)を和訳しなさい。
- (3) 下線部(3)を和訳しなさい。
- (4) 下線部(4)を和訳しなさい。
- (5) 最後の3段落(But evolutionary changes...から終わりまで)を120~150字 の日本語で要約しなさい。句読点も1字に数える。