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the telomerase RNA in the HeLa cells and impede its work, resulting in a shortening of the cells' telomeres. Indeed, after 23 to 28 doublings, more than half of the HeLa cells injected with the antisense went into crisis and died, the collaboration reports in the Sept. 1 SCIENCE.

Few investigators expect that the antisense approach will develop into a practical method of inhibiting telomerase. Instead, they hope to find or create small molecules that also bind to the enzyme and would be easier to develop into drugs.

But, researchers caution, inhibiting telomerase throughout the human body may not be safe. If normal cells depend on the enzyme, the treatment could cause as many problems as the cancer it targets.

In the first studies of telomerase activity in normal human tissues, researchers found that the only tissues that tested clearly positive for the enzyme were the ovaries and testes. That didn't surprise investigators, because those sites presumably contain a reservoir of stem cells that provide the progenitors of sperm and egg cells. These stem cells are long-lived and divide relatively frequently, Shay explains, so they must have found a way to keep making telomerase after most other human cells turn off the necessary genes.

If those so-called germline cells were the only human cells that needed telomerase, says Calvin Harley of Geron, telomerase inhibitors could be an effective cancer treatment. Most people, he argues, would sacrifice their reproductive ability to rid themselves of cancer. Moreover, cancer tends to strike after an individual's reproductive years, he adds.

Recent work by Harley and others, however, establishes that some nongermline cells do in fact contain telomerase, though in much less abundance than tumor cells or germline cells.

"The most simple paradigm—yes in malignant cells and no in normal [nongermline] cells—is not absolutely true," says Richard J. Hodes, director of the National Institute on Aging in Bethesda, Md., "That doesn't mean that there is not an important biological difference between normal and malignant cells that couldn't be capitalized on. But it certainly calls for great caution in presuming one could, in a perfectly specific way, influence malignant cells, but not normal cells, by influencing telomerase activity."

The nongermline cells that appear to use the enzyme come from so-called renewal tissues—blood, skin, and intestinal lining. The body must constantly supply new cells in these tissues, which means the tissues may have evolved means of avoiding the normal limits on cell division.

The renewal tissues probably contain a small reservoir of stem cells that can produce telomerase intermittently, suggests Shay. He believes that telomerase inhibitors, though they could affect those stem cells, may still offer a cancer treatment option that is far less dangerous than traditional chemotherapy, which kills all dividing cells.

Studies in mice over the next year or so should resolve many of the questions about the importance of telomerase in normal mammalian cells. Now that investigators have the gene for the RNA component of mouse telomerase, they can knock it out in mouse embryos and observe whether the animals are born healthy and survive without the enzyme.

"That's a very important experiment. This will settle the whole issue," says Titia de Lange of Rockefeller University in New York City.

Researchers also plan to test whether tumor growth differs between mice that can't make telomerase and those that can, says Greider.

If these efforts do provide a new strategy for combating tumors, or if they simply explain why a cell stops dividing in its old age, it will be a stunning example of the value of basic research, telomere investigators say.

"Telomere research started out as a novelty, with people focusing on these strange organisms you find in ponds," says Morin. "Now, all of a sudden, this backwater field of research is offering a potential cure for cancer."

Environment

Janet Raloff reports from Vancouver, British Columbia, at the annual meeting of the Society of Toxicology and Environmental Chemistry

Recycling pesticide bottles: A risk?

Manufacturers bottle many toxic pesticides in high-density polyethylene (HDPE). Because such contents can permeate this plastic, labels instruct consumers not to reuse the bottles. Yet people dispose of so many plastic pesticide containers—1 million a year in Canada alone—that waste managers have begun recycling pesticide-impregnated HDPE into building materials, such as fence posts. Researchers at the University of Guelph in Ontario now report that a technique adopted recently to limit pesticide leaching from this plastic "is not as effective as it could be in limiting pesticide penetration."

Manufacturers usually dissolve oily pesticides in a solvent to ease their dispersal. However, the solvent degrades HDPE, helping pesticides enter and leach out of the plastic. To stem the resulting losses from evaporation, pesticide makers have turned to fluorination—where fluorine atoms swap places with hydrogen in the plastic's molecules, forming a solvent barrier.

However, notes Graham M. O'Brien, "the containers we looked at, the industry standard for packaging these pesticide formulations, didn't form a complete barrier." These plastics still incorporated pesticides at "quite high levels"—up to 1 gram of solvent-based herbicide, such as trifluralin or 2,4-D, per 350 grams of plastic. By contrast, HDPE produced by a more stringent, but expensive, fluorination technique allowed one-thousandth or less of that amount to permeate—yielding truly negligible levels.

O'Brien

Orange stains illustrate extensive pesticide (trifluralin) penetration of HDPE container.

Few companies use this more expensive technique for pesticide bottles, he noted, because the old type "appeared to be doing its job"—cutting product losses from evaporation.

Guelph studies published earlier this year found that plastic from recycled pesticide containers can leach detectable, albeit insignificant amounts, of the toxic compounds. However, O'Brien observes, pesticides that are more mobile or more highly concentrated than those studied may still present problems. That's why he argues that only the better-fluorinated pesticide containers should be recycled if any resulting products will be used in watery environments or where extensive human contact can occur.

Another source of lead in kids

Potentially toxic amounts of lead can leach from good crystal into any drink it holds (SN: 1/26/91, p. 54). But the heavy metal poses its biggest risk to children, who don't tend to sip their milk, juice, and sodas from crystal goblets. So Charles V. Shorten and Mary L. Glowacki of West Chester (Pa.) University asked whether some other use of crystal, such as vinegar stored in crystal cruets, might add lead to children's diets.

Their 42-day study of 13 different cruets showed that lead's passage into the acidic liquid started quite rapidly, reaching an average of 162 micrograms per liter (µg/l) within the first hour. By the end of the trial, lead concentrations in the vinegar had climbed to an average of 730 µg/l, or 8 times the level achieved in distilled water. These data suggest that a 16-kilogram child could thus acquire 15 percent of the federal government's provisional daily tolerable intake from all sources.