



WHAT'S

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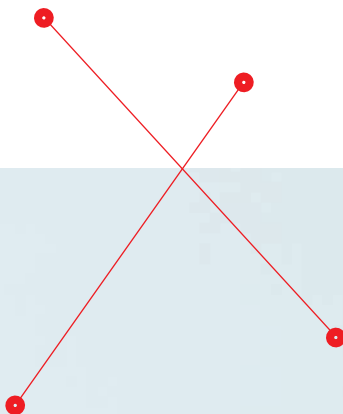


Legions of scientists are hunting for better, faster ways to keep us feeling younger. Here's a look at some of the research on the horizon

by ALICE LESCH KELLY

/ YOU ARE (NOT EXACTLY) WHAT YOU EAT /

If you've ever wondered why a vegetarian who consumes no saturated fat can have a cholesterol level of 240 while an overweight meat lover who lives on cheeseburgers and whole milk clocks in at 160, you'll be interested in the work of Katherine Tucker, PhD, an epidemiologist at Tufts University's Friedman School of Nutrition Science and Policy. Tucker is a researcher in the field of nutrigenomics, the study of how our genetic dispositions affect the way our bodies use the nutrients in what we eat. She believes that nutrigenomics researchers will be able to unravel some of the paradoxes that have emerged from major studies, such as why some people need more folate in their diet to process homocysteine, an amino acid that can damage arteries. "Current population-based studies often have conflicting and confusing results regarding specific nutrients and health outcomes," Tucker says. She thinks genetic predisposition, which is



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not taken into account in large-scale epidemiological studies, will probably explain the perplexing results.

“The nutritional advice we’re giving right now is based on the average person,” Tucker says. Nutrigenomics research should make it possible for health care providers to give more customized advice in many areas, including weight loss.

Although nutrigenomics is still in its infancy, Tucker expects it to become a major player in the science of aging. “In 10 years,” she says, “individuals will go to their doctors, have their genetics analyzed and be told, for example, that fish oil can help reduce their risk of heart disease and Alzheimer’s disease”—while that same advice may not be useful to people with a different genotype.

/ CLEANING UP THE BRAIN /

An amazing thing happened when Kim Finley, PhD, managed to maintain a cellular cleanup mechanism in the brains of fruit flies as they aged: They lived longer—by 55 percent, on average. “It was one of the most dramatic life span extensions ever found in flies,” says Finley, staff scientist at the Salk Institute for Biological Studies, in La Jolla, California. “Plus, those flies were extremely healthy.”

Now Finley wants to know whether the same process will work in humans. If it does, it could have a major impact on the preventive treatment of Alzheimer’s disease and other neurodegenerative disorders in which an excessive amount of damaged proteins in brain cells contributes to the death of the cells. “What I need to be able to do is see if this is doing the same thing in people, and all indications are that it is,” Finley says.

The mechanism Finley kept functioning is called autophagy, the process by which cells remove damaged protein molecules and other “trash” that accumulates as we age. As each birthday goes by, our autophagy likely slows down, and as cellular trash



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According to research from the National Institutes of Health, American women who ate a Mediterranean-style diet were 20 percent less likely to die within five years than women who didn’t follow one. Loaded with whole grains and fibrous fruits, nuts, veggies and

legumes, the diet is also high in the monounsaturated fats found in olive oil and rich in the omega-3 fatty acids in fish. (For examples of Mediterranean-style dishes and recipes, see “The Antiaging Dinner Party,” page 130.) Eating these kinds of nutrient-dense meals regularly can help protect your heart and strengthen your immune system. What’s more, you can lower cholesterol and fend off weight gain, reducing your chances of developing obesity, diabetes, heart disease and possibly cancer.

Fish is a healthy part of the Japanese diet too. “The Japanese eat 50 times more fish than Americans,” says Richard J. Flanigan, MD, codirector of the Heart and Health Prevention Center, in Denver. Don’t let concerns about mercury deter you from eating fish. Although long-living species such as swordfish and tilefish, which may contain high levels of mercury, shouldn’t be consumed more than once or twice a week, salmon and trout are considered safe and can be eaten often.

builds up, it damages and kills cells. Finley hopes a drug will be developed that will keep the autophagy process running smoothly. People may not necessarily live longer, as the flies did, she says, but they may experience a slower cognitive decline.

/ FIGHTING CANCER FROM THE INSIDE /

The latest mantra of Susan Love, MD, long a warrior against breast cancer, is “Go to the source.” In this case, that would be the half dozen or so milk ducts in the nipple. “All breast cancer starts in the lining of these milk ducts,” says Love, of the Dr. Susan Love Research Foundation, in Pacific

Palisades, California. “It makes sense that if you can get to where it starts, you can prevent it.” (A 40-year-old woman has a one in 70 chance of developing breast cancer; a 50-year-old woman has a one in 40 chance, according to the American Cancer Society.) In a new study, Love and her colleagues will look at whether introducing a low dose of chemotherapy into the milk ducts under local anesthesia can destroy precancerous cells or keep them from progressing to invasive cancer (which can spread to the rest of the breast and beyond). If intraductal chemotherapy works, it may spare women disfiguring surgery and radiation. “Rather than doing

NEW TREATMENTS MAY SPARE WOMEN SURGERY.

preventive mastectomies,” Love says, “wouldn’t it be great if we could just squirt something down the duct and clean it out?”

In another study, Love and her colleagues are attempting to develop an adhesive test strip that will signal whether a woman is at risk for developing breast cancer. A premenopausal woman could cover her nipple with the strip. If the strip picks up biological clues that the woman is at risk, it would change visibly, alerting her and her doctor to the need for further testing and follow-up.

/ SOLID AND STRONG BONES /

In 2006, Yongwon Choi, PhD, professor of pathology and laboratory medicine at the University of Pennsylvania, discovered that targeting a specific gene inhibited bone decay and stimulated bone growth in mice. Within 10 years, he’d like to find out whether altering the action of that gene in humans could prevent or delay the onset of osteoporosis, a disease that affects 10 million Americans—eight million of whom are women, says the National Osteoporosis Foundation.

It appears that inactivating the gene in question (Atp6v0d2) increases bone mass by immobilizing the action of osteoclasts (cells that break down and help resorb bone) and boosting the brawn of osteoblasts (cells that form bone). Current medications do one or the other but not both, Choi says. “If we can find a

way to get to our target gene with a drug, we may be able to help the millions of people with osteoporosis.”

/ HAVING YOUR GENES TAILORED /

Scientists are sure that genes influence the risk of type 2 diabetes, but they don’t know which ones. If they did, they believe, they could develop drugs that would alter the action of those genes and help rein in the soaring prevalence of the condition.

Leading this needle-in-a-haystack search is Joanne Curran, PhD, a genetic researcher at the Southwest Foundation for Biomedical Research, in San Antonio. Curran and her colleagues will spend the next five years analyzing 100 genes believed to play important roles in diabetes to see how they influence blood glucose, insulin production and body fat.

“Right now, the best prevention we have for diabetes is lifestyle change,” Curran says. “But what if you have a genetic predisposition that’s causing your high levels of blood glucose? Then lifestyle changes alone may not help. That’s why we need to find the genes involved, and the DNA variations that affect those genes’ function.

“In 10 years’ time, we’re still not going to know everything, but we’re going to be a lot closer than we are,” Curran adds. “There will likely be many more useful treatments on the market that will help prevent or treat diabetes.” **M**