tighten and loosen, they have lacked a comprehensive theory to explain how and why this happens.

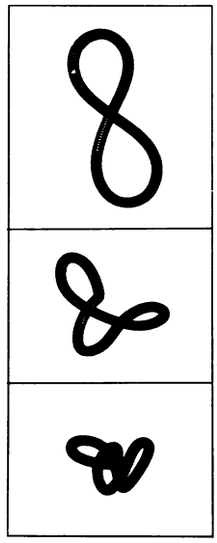
Using a detailed computer model, says Schlick, her group has demonstrated clear differences in DNA shape and behavior depending on the amount of salt in a simulated molecule's environment. The team also posits a mechanism for these changes.

Examining how simulated DNA strands, each with 1,000 base pairs, interact with charged salt molecules at varying concentrations, the researchers found that the energies, shape, and motion of supercoiled DNA all "change dramatically as a function of salt." The DNA's form became "highly compact, bent, rigid, and interwound" as the amount of nearby salts rose, while in the presence of lesser amounts, the coils became "open, loose, and flowing in shape."

Moreover, the researchers found that the amount of salts surrounding DNA strongly affects the "buckling transition," in which a loop of DNA twists into a figure eight. Indeed, Schlick noticed that by raising the amount of salt, pieces of the loop would "slither" past one another and then undergo a "collapse," crunching up into a highly compressed form.

"These observations suggest a potential regulatory role for salts on DNA processes," Schlick says. In the presence of a solution rich in salts, the tightly coiled molecule "brings into contact segments of DNA that are far away in the linear sequence." These actions, she believes, could "potentially play important regulatory roles in [gene] transcription and recombination."

The behavior of DNA in the computer model, adds Schlick, shows a striking similarity to results culled from recent electron microscopy studies and laboratory experiments. Yet "the structural information that can be extracted from the simulations is far richer in detail than that offered by low-resolution measurements" in a laboratory, she asserts. — *R. Lipkin*



Constantine Kreatsoulas, Gomathi Ramachandran

A computer simulation shows that, over time, increasing concentrations of salt can cause a loop of DNA to coil more tightly.

Vitamin C helps cigarette-smoking hamsters

There's good news for antioxidant supporters: A new study in the Aug. 2 PROCEEDINGS of the NATIONAL ACADEMY of SCIENCES shows that vitamin C protects hamsters from some of the harmful effects of cigarette smoke.

Previous studies suggested that the antioxidants vitamin C, vitamin E, and the drug probucol offer a shield against atherosclerosis — one smoking-induced health problem — because of their ability to disarm highly reactive free radicals in the blood (SN: 8/26/89, p.133). Data from two recent cancer prevention trials, however, have tarnished the image of antioxidants as the body's premier scavengers of harmful free radicals (SN: 7/23/94, p.54).

Certain smoking-related diseases, including atherosclerosis and emphysema, share a common trait: aggregations of leukocytes, or white blood cells, that adhere to endothelium, the smooth tissue that lines blood vessels.

Balz Frei of the Boston University School of Medicine, a coauthor of the new report, explains that "cigarette smoke is full of oxidants and free radicals," which bombard healthy DNA, wreaking havoc on normal cellular functions.

The researchers set out to see how antioxidants "can counteract cigarette-smoke-induced leukocyte activation-adhesion in the hamster." They discovered that water-soluble vitamin C, given as part of the diet or intravenously, significantly reduced white cell adhesion. Fat-soluble vitamin E and probucol had no effect on adhesion.

According to Frei, the mechanism of how cigarette smoke induces leukocyte adhesion is not well understood, though it "seems to involve a water-soluble free radical, most probably superoxide." A fat-soluble antioxidant like vitamin E may not come in contact with superoxide and therefore would not be able to detoxify this free radical.

The researchers also found that vitamin C injected just 5 minutes prior to cigarette-smoke exposure offered protection, suggesting, the authors write, "that vitamin C does not need to be incorporated into the cells in order to be effective."

William A. Pryor at Louisiana State University in Baton Rouge says he "wouldn't have predicted" vitamin E's lack of effect in these animals. Even so, both Pryor and Frei believe that antioxidants, including vitamin E, provide disease protection.

What's most important about this study, Pryor says, is the suggestion of "a biological plausibility, a mechanism" to explain how antioxidants might work to prevent leukocyte adhesion. "I think this is an important paper, really provocative, very intriguing." — *G. Marino*

Cancer: Pare protein to spare the kidneys

As lethal malignancies go, kidney cancer does not make the Top 10 list. Even in incidence, it ranks about 12th in the United States, below such cancers as uterine, ovarian, oral, bladder, melanoma, and pancreatic. However, over the past 25 years, cases of kidney cancer have been climbing steadily — by about 2 percent annually. This year, it's expected to strike more than 27,600 individuals in the United States and to claim some 11,300 lives.

A new study now suggests that a penchant for protein may be fueling the cancer's ascent.

Over the years, few clear-cut risk factors other than cigarette smoking and obesity have emerged for kidney cancer. However, several previous studies have suggested that eating patterns — principally, diets high in animal fat, meat, and milk — might be linked to the disease. Hoping to resolve the role of diet, Wong-Ho Chow of the National Cancer Institute in Bethesda, Md., and his coworkers administered a detailed questionnaire to 690 kidney cancer patients in Minnesota (or their next of kin) and to 707 demographically matched, cancerfree volunteers.

In the Aug. 3 JOURNAL OF THE NATIONAL CANCER INSTITUTE, Chow's team reports that after accounting for each subject's age, sex, smoking, weight, and average calorie intake, only diets high in protein — from *all* sources, including plants — increased an individual's risk of developing kidney cancer.

The researchers divided their study population into four groups, or quartiles, on the basis of how much of any analyzed nutrient each subject consumed. Those in the highest quartile of total protein consumption faced almost twice the kidney cancer risk of those in the lowest quartile. What's more, the increase in risk with protein consumption occurred independent of calories, the researchers observe — "particularly when caloric consumption was above the median intake."

The absence of any elevation in risk with increased consumption of fat or carbohydrates suggests that kidney cancer is not spurred simply by the number of calories in a diet.

Linda D. Youngman, a nutritional biochemist with the Imperial Cancer Research Fund in Oxford, England, says the new findings don't surprise her. She has observed that kidney cancer is one of many malignancies whose incidence diminishes dramatically in rodents that she raises on very-low-protein diets.

Data from her studies suggest that such anticancer effects may trace to the ability of low-protein diets to reduce the assault

on the body by biologically damaging free radicals (SN: 11/21/92, p.346).

Arginine – an amino acid building block of many proteins – is a precursor of nitric oxide (NO), a free radical, explains cell biologist Erkki Ruoslahti of the La Jolla (Calif.) Cancer Research Foundation. So if dietary protein fosters kidney cancer via the production of free radicals, Ruoslahti says, "It's possible that arginine – as a source of NO – might be the constituent of protein causing this effect."

However, there are other pathways by which protein may affect kidney cancer risk, notes nephrologist Wayne A. Border of the University of Utah School of Medicine in Salt Lake City. For instance, transforming growth factor beta (TGF-beta), an immune system messenger produced by the body, can be a tumor promoter. And experiments that Border's lab conducted together with Ruoslahti indicate that in rats, diets low in protein suppress the kidney's production of TGF-beta.

Moreover, Ruoslahti notes, kidney disease is almost invariably associated with increased TGF-beta production. As such, he says, protein's TGF-beta connection may also explain why a history of certain kidney diseases increases an individual's risk of developing kidney cancer.

– J. Raloff

Extreme fatigue hard on repaired hearts

In the days or weeks before a heart attack or sudden death from heart disease, many people feel drained and devoid of their usual energy. A new study suggests that psychological factors help to produce this kind of exhaustion in some individuals after surgical repair of narrowed heart arteries; the fatigue then markedly boosts their chances of suffering further serious cardiac problems.

Fatigue, heightened irritability, and a sense of demoralization make up what Willem J. Kop, a psychologist at the Uniformed Services University of the Health Sciences in Bethesda, Md., and his colleagues call "vital exhaustion."

"It may be that psychological stress reduction, resulting in a reduction of feelings of vital exhaustion, will reduce the risk of new cardiac events in patients [after the artery procedure]," the scientists conclude in the July/August PSYCHOSOMATIC MEDICINE.

Kop's team recruited 127 adults, none more than 70 years old, who had undergone angioplasty to widen at least one significantly constricted cardiac artery. About 2 weeks after successful treatment, each participant completed a questionnaire designed to tap into the three components of vital exhaustion.

A previous study employing this questionnaire, which followed healthy men for more than 4 years, found that the rate of initial heart attacks doubled for those who had cited vital exhaustion.

The current project, which monitored volunteers for 1½ years after their discharge from the hospital, reveals a similar pattern. About one-third of the 43 patients who noted vital exhaustion after treatment suffered severe chest pains, heart attacks, or sudden death due to cardiac complications; only 17 percent of the remaining 84 experienced these reactions.

This disparity remained after controlling for the initial severity of each volunteer's heart disease, as indicated by the number of narrowed arteries.

Vital exhaustion may overlap with severe depression, the researchers say. Other evidence suggests that depression markedly elevates the death rate of heart attack survivors (SN: 10/23/93, p.263).

However, depression includes intense sadness, low self-esteem, and guilt feelings that usually do not show up in cases of vital exhaustion, they argue.

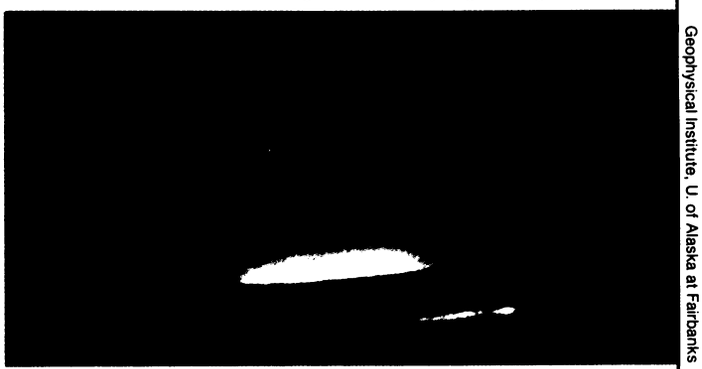
Vital exhaustion may render patients more prone to plaque formation or blood clotting in heart arteries, thus leading to narrowing or blockage of the vessels, Kop and his coworkers theorize.

In an accompanying editorial, Stewart Wolf, a psychiatrist at Totts Gap Laboratories in Bangor, Pa., compares vital exhaustion to a feeling of "carrying a heavy load and never quite getting to the resting place." He has found that this fatigue raises the likelihood of further complications in heart attack survivors. Brain areas that send impulses to the heart and blood vessels may trigger fatal reactions after particularly stressful experiences, Wolf maintains. *– B. Bower*

Enigmatic bursts show their true colors

High above the tops of thunderstorms, flashes of light dance like sprites in Earth's atmosphere. For the first time, researchers have captured the true colors of these scintillations – red bursts resembling jellyfish and blue ones shaped like flaring trumpets.

Though the bursts reveal an electrical link between the ionosphere and the atmosphere's lower depths, their origin remains a mystery. Adding to the puzzle, other scientists have noted a possible correlation between thunderstorms and flashes of gamma rays and radio waves.



Geophysical Institute, U. of Alaska at Fairbanks

True-color image of a flash of light, dubbed a sprite, high above a Midwestern thunderstorm. White-blue area beneath the red sprite is normal lightning.

Scientists confirmed the visible-light flashes last year. Flying over the Midwest, they videotaped in black and white several bursts above storm clouds.

This June 28 through July 12, Davis Sentman and Eugene Wescott of the University of Alaska at Fairbanks took to the skies again, this time with a color video camera. Coordinating with ground-based teams, the pair made observations from two aircraft, allowing them to accurately measure the flashes' speed, position, and altitude.

Sentman and Wescott found that the flashes fall into two categories. "Sprites" appear higher in the atmosphere, some 75 to 85 km above ground, and last for a few thousandths of a second. Though they don't connect to the storm clouds, some of these blood-red flashes have dangling blue tendrils.

In contrast, says Wescott, "jets" last longer, originate at the tops of storm clouds, and shoot up to an altitude less than half that of sprites. Jets are narrower than sprites, fanning out like a trumpet in a blue or purple hue.

Wescott speculates that the electric field generated by lightning travels up through the atmosphere and then discharges, creating the sprites. But the jets, although also associated with thunderstorms, appear to be "a completely new phenomenon," he adds.

Daniel N. Holden of the Los Alamos (N.M.) National Laboratory and his colleagues have now linked some 300 unusual radio bursts detected by the ALEXIS satellite to possible thunderstorm activity (SN: 2/12/94, p.100).

Holden says it's tempting to associate these bursts with those seen in visible light, as well as the atmospheric gamma-ray flashes recorded by the Compton Gamma Ray Observatory. But he cautions that the much shorter duration of the radio bursts suggests that they may come from a far smaller source in the atmosphere. *– R. Cowen*