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

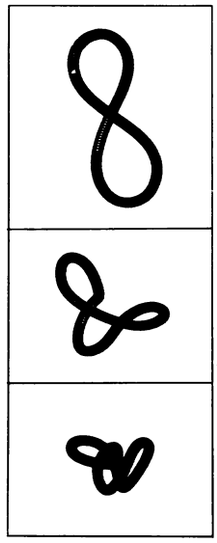
Using a detailed computer model, says Schlick, her group has demonstrated clear differences in DNA shape and behavior depending on the amount of salt in a simulated molecule's environment. The team also posits a mechanism for these changes.

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