EPA bans six new chemicals

Last week, for the first time, the Environmental Protection Agency banned the manufacturing of several new chemicals slated for market introduction on the grounds that they may pose serious risks to human health and the environment. The six phthalate esters in question are plasticizers, used to make polyvinyl-chloride plastics more flexible.

What makes this order particularly interesting and important is that "evidence" used to call suspicion to the plasticizers was not based on toxicological tests of those chemicals. In fact, the manufacturer (which EPA is prohibited by law from identifying by name) offered no test data on health or environmental effects associated with the chemicals when it notified the agency that it was planning to introduce them. That such data have never been required of manufacturers for the introduction of new chemicals has proved a stumbling block for regulators when later they try to regulate those chemicals based on health risks eventually associated with them.

But citing a provision of the Toxic Substances Control Act, EPA can now shift the burden of proof—from EPA having to show which chemicals are toxic to the manufacturer's having to prove that its chemical is not toxic — "when there is reason to believe a chemical may prove toxic," according to Ernest Rosenberg of EPA's premanufacture-notification office. As the manufacturer offered no data, EPA culled its files to make some kind of determination.

What it found was that the proposed chemicals were structurally similar to the chemical DEPH [di-(2-ethylhexyl) phthalate], some 400 million to 600 million pounds of which is produced annually. DEPH has shown a potential to cause chronic health effects in aquatic animals. But more seriously, a yet-unpublished report has linked DEPH exposure with cancer in rats and mice. "And our best judgment," says Rosenberg, "was that because of a variety of factors, [these six new chemicals] are likely to be at least as bad as DEPH in terms of a biological effect and possibly worse."

According to EPA's Carl Mazza, this order represents one of the first times the agency has used "structure activity arguments," wherein the similarity in structure between two chemicals is used to indict, or to raise suspicions, about a new chemical based on health-effects concerns associated with an existing chemical. Even DEPH has not been proved dangerous, though that is suspected. What the new EPA order does say is that unless and until data refute suspicions raised by the phthalate esters' similarity to DEPH, EPA will not permit their introduction.

To remove existing chemicals, like DEPH, from the market — should toxicity worries prove valid — takes much stronger evidence. That's an inconsistency under the law, Rosenberg says, but one Congress chose for us to live with. □

Jog those blood clots away

Exercise reduces the risk of heart attack, according to a variety of epidemiological studies. But the mechanisms linking jogging, swimming and calisthenics with good health are still under investigation, and the relationship between exercise and health is often called "circumstantial." One concrete, biochemical finding has been that exercise reduces some types of cholesterol in the blood, while increasing levels of another, the protective high-density lipoprotein (SN:12/9/78, p. 408). Now Duke University investigators report a separate healthful development among persons on a vigorous activity program. Exercise increases the ability of the blood to respond to vein blockage via a boost in the factor that dissolves clots. Such a defense against blood clots may protect against strokes and pulmonary embolism, as well as against heart attacks.

In the May 1 NEW ENGLAND JOURNAL OF MEDICINE cardiologist R. Sanders Williams and colleagues report on 69 healthy adults between 25 and 69 years of age who participated in a 10-week physical conditioning program. Williams and co-workers used a new assay to measure the blood clot dissolving, or fibrinolytic, activity of the subjects' blood.

To stimulate the biochemical response to a medical crisis — blockage of a vein by a blood clot — the investigators inflated a blood pressure cuff around each subject's arm. After 5 minutes, blood drawn from the arm had an increased level of fibrinolytic activity. The researchers found that after the exercise program the background level of fibrinolytic activity had decreased and the increment in activity between the background and stimulated state was significantly increased.

The greatest boost in fibrinolytic activity in the simulated blood clot situation was among those who were least fit before the exercise program. In general women had a greater increase during the program than did men, and those with the lowest stimulated activity levels initially increased more than those who had higher levels at the start.

Smoking habits and levels of other blood proteins do not explain the results, the researchers say. They observed the expected increase in high-density lipoprotein, but the boost in fibrinolytic activity appears to be a separate effect. □

Weight and mortality: Skinny isn't best

In spite of modern society's emulation of Twiggy models and the adjuration that "thin is in," skinny persons have a higher mortality rate than do more robust persons, findings reported in the May 9 JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION suggest. These results are all the more surprising because they do not jibe with the results of an earlier, long-accepted investigation.

In 1959 researchers analyzed the heights, weights and mortality data for thousands of Americans who had been insured by various life insurance companies between 1935 and 1953. They found that in general the heavier the subjects were for their stature the greater their mortality rate. These findings, part of the Build and Blood Pressure Study, provided the basis for tables of desirable weights that hang in doctors' offices and to which the public is encouraged to adhere.

Now Paul Sorlie and Tavia Gordon of the National Heart, Lung and Blood Institute and William B. Kannel of Boston University Medical Center have analyzed the heights, weights and deaths of 5,209 men and women who participated in the Framingham, Mass., heart study from 1948 to 1972. The researchers used the same definitions of body build and follow-up periods for mortality as did the BBPS. As they report, their findings only partially confirm those of the earlier study. The new results show minimum mortality for persons of average weight, increased mortality for those weighing less or more than the average.

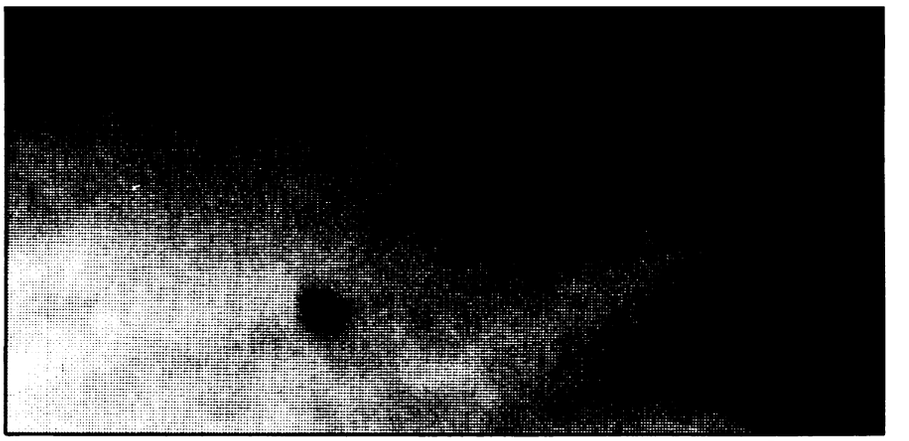
The crucial question, of course, is why this study found a higher mortality rate for lean persons than did the BBPS. Cigarette smoking was ruled out as a confounding factor in the Framingham study—the mortality curve was similar for current smokers and nonsmokers and, in fact, among nonsmoking men, the leanest group was shown to have the highest mortality rate. Overt disease (not a significant factor in the BBPS due to the medical screening involved in insurance underwriting) was also eliminated as a contributing factor. The researchers tentatively suggest that

	Build Group by Weight, lb				
	1	2	3	4	5
Man's height					
58″-62″	<115	115-134	135-154	155-174	175-254
63″-66″	<115	115-154	155-174	175-194	195-254
67″-70″	<135	135-174	175-194	195-214	215-254
71″-74″	<155	155-194	195-214	215-234	235-254
75″-79″	<175	175-214	215-234	235-254	—
Woman's height					
52″-58″	< 95	95-114	115-134	135-154	155-254
59″-62″	<105	105-134	135-154	155-174	175-254
63″-66″	<115	115-154	155-174	175-194	195-254
67″-70″	<135	135-174	175-194	195-214	215-254
71″-75″	<155	155-194	195-214	215-234	235-254

Body builds: Group 3 lives longest.

Science Service, Inc. is collaborating with JSTOR to digitize, preserve, and extend access to *Science News*. www.jstor.org

there is "some indirect evidence" that the higher mortality rate can be accounted for by the presence of subclinical disease, yet are at a loss to explain it. They point out that since the two studies involved different time periods unknown factors may have been involved. Nonetheless, they believe that their results "raise some questions as to the health benefits from weight reduction in persons of average or near-average weight." □



JPL

Newfound satellite and its shadow, seen crossing Jupiter by Voyager 1. Dark streak extending from shadow is cloud feature coincidentally aligned with shadow's position.

Another moon: Jupiter's 15th

Last year a 14th satellite of Jupiter was discovered in one of the Voyager 2 spacecraft's photos by scientists who had originally thought the point of light to be a star (SN: 10/20/79, p. 263). Now a researcher who was looking for the same object in photos from Voyager 1 has discovered yet another satellite, Jupiter's 15th.

Attempting to measure the 14th moon's orbit more precisely, Stephen P. Synnott of Jet Propulsion Laboratory (the Voyager control center) had been looking at Voyager 2 photos taken when the object should have been one or two orbits behind or ahead of its position in the photo that led to its discovery. Failing to find it at the time, he turned to pictures from Voyager 1, and in one of them, he says, "I saw what looked like a shadow against Jupiter's disk." The next frame showed the same shadow in a different position, and Synnott assumed it to be number 14 (designated 1979 J1) until an orbit calculated from the two images showed it to be on a clearly different path. After a search of "weeks and weeks," Synnott finally located a single photo that showed both the shadow and its source — a previously unknown satellite, now designated 1979 J2.

It orbits the planet at a mean distance of slightly more than 151,000 kilometers from the cloud tops, Synnott calculates from the several frames in which the object has by now been identified. This puts it between the orbits of Amalthea (about 107,000 km out) and Io (nearly 350,000), circling Jupiter once every 16 hours 16 minutes. It also becomes easy to see how as few as two photos could enable Synnott to tell that the object's orbit was not that of the 14th moon, whose mean distance from the clouds had been calculated as slightly over 57,000 km, just about along the edge of Jupiter's ring system (also discovered from Voyager pictures). Synnott has now revised his calculations, in fact, and determined that the true distance is only about 56,400 km — actually *in* the ring.

The newly found satellite appears tiny in the photos, and Synnott estimates it to be only about 80 km across — "at least in the direction we can observe." It has only been spotted in photos showing it against the disk of Jupiter, and since it is almost certainly gravitationally "locked" with its longest axis pointing at the planet, it is possible that the Voyagers were only able to see it, in effect, "end on." Amalthea, for example, measures 155 km in one dimension, but 270 km in the direction radial to Jupiter. To measure the new satellite's long axis, Synnott hopes to be able to pick out the object against the dark sky in photos taken off the edge of the planet, when the spacecraft would have been facing it broadside.

Its composition, too, is uncertain from the limited data, although Synnott says that it is "about as dark as Amalthea," whose surface has been found to approximate laboratory spectra of carbonaceous chondrite material augmented with sulfur possibly transported inward from Io. The yet-unnamed number 15, in fact, could turn out to be more sulfur-rich still, since its orbit is about 44,000 km closer to Io's than is that of Amalthea.

Jupiter may actually have not merely 15 satellites, but 16. In 1975, Hale Observatories astronomer Charles Kowal spotted a possible candidate in an earth-based photograph (SN: 10/11/75, p. 229), but subsequent observations failed to confirm its presence around the planet. It was seen far from Jupiter, so at least it cannot be confused with the two "Voyager moons," but if it is someday confirmed, it will probably be assigned a place as the 14th satellite, bumping the newcomers to 15th and 16th. And no one is ruling out the possibility that still more may be discovered. □

Waking up to a biological alarm

The human body, keeping time to a variety of biological clocks, somehow knows there is a time to every purpose. But where and what are the body's timepieces? According to recent work by Harvard Medical School researchers, one behavioral timer may have been found — the clock that dictates the time to be asleep and the time to be awake. If validated, the find will be the first identification of a biological timekeeper in humans.

The clock, described by Martin Moore-Ede, Ralph Lydic and co-workers at a recent meeting on sleep in Mexico City, is a small cluster of neurons in the hypothalamus of the human brain. A similar area was identified eight years ago in the brains of rodents and called the suprachiasmatic nuclei (SCN). When the region was destroyed, the rodents lost their circadian (approximately 24-hour) rhythm in feeding and other behaviors.

With these findings in mind and interested in the physiological basis for "jet lag" and sleep disorders, Moore-Ede and co-workers began to search for a structure that might be responsible for such behavior. They found that the brains of squirrel monkeys contain structures similar to the rodent SCN — actually two clusters of neurons in the hypothalamus located on either side of the tip of the brain's third ventricle (a fluid-filled cavity). When the neuronal clusters were destroyed, the rest-activity cycle of the monkeys was disrupted, while other rhythms — such as body temperature — were unaffected. "Obviously this is not the only biological clock, but it is certainly a major one," says Moore-Ede.

Moving systematically up the primate tree the researchers then studied the brains of eight New World (Western Hemisphere) and Old World (Eastern Hemisphere) primates and sections of 16 human brains, ranging in age from 28 weeks gestation to 50 years. In each, says Moore-Ede, the same cluster of small, rounded neurons — identified microscopically — was present. As the scientists moved up the evolutionary tree, however, the position of the 300-micron-wide clusters shifted, so that in humans the cells are more diffusely distributed and placed on the sides rather than at the tip of the ventricle. This more diffused distribution may be related to the observation that the sleep-wake cycle in humans is not as rigorous as in other species, says Moore-Ede.

While the exact mechanism is not understood, Moore-Ede says the cells are apparently an "endogenous, self-contained pacemaker" sending out precise signals to the rest of the brain. Because the appropriate human experiments cannot be done, proof that the cell clusters

Continued on p. 301