other tones, even in the traditional musical scale, it is most unlikely that we remember musical sequences by storing the absolute pitches of the component tones. Rather, it appears that we must rapidly discard absolute pitch information and store musical sequences in a recoded form. How this might be achieved is discussed in detail elsewhere (6).

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Cyclamate Acceptance

Recent letters have been written on the relative wisdom of the government ban on the use of cyclamates in foods (1). Many references have also been made to published (2) and unpublished research which seems to show that cyclamate, cyclohexylamine, and even saccharin may have carcinogenic effects.

In the majority of these studies either the chemical was surgically implanted in body tissue or else it was placed in the only available source of food or water. In both situations, the animal didn't have much of a choice about whether or not it ingested the substance under investigation.

Humans have voluntarily chosen to accept cyclamate-sweetened foods (projected consumption for 1970 was 21 million pounds prior to the ban), although one national magazine did suggest that "most cyclamates end up in the stomachs of Americans because of advertising campaigns," not because of preference per se (3). It would, therefore, seem logical to give experimental animals such a choice since it has been pointed out that rats and mice behave toward sweets somewhat as humans do (4).

Two groups of investigators found that rats avoid cyclamate solutions for water and that C 57 black mice preferred a 1 percent cyclamate solution to water (5). Four species of deer mice preferred glucose to either calcium or sodium cyclamate, regardless of sweetness, but chose the sweeter solution when the choice was glucose or saccharin (6). More recent work in my group (7) has shown that various strains of laboratory rats as well as wild rats avoid cyclamates in favor of water, glucose, or saccharin. These same rats

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also tend to avoid a 10:1 cyclamatesaccharin mixture. The evidence seems to show that rodents do not choose cyclamate if a more palatable choice is available (even if that happens to be only plain tap water).

In light of the physiological evidence mentioned above, which suggests that cyclamates produce toxic effects, it might be proposed that aversions for sweet cyclamates are somehow related to this toxicity. Indeed, taste preferences and aversions have elsewhere been reported to be intimately related to experienced ill-effects. Exposure to x-radiation has been used to condition saccharin aversions (8) and rats avoid toxic lithium chloride (9).

How, then, can one reconcile the reported similarities in human and rodent sweet preferences on the one side, with the rodent aversions and the massive human acceptance of cyclamate-flavored foods on the other side? Do humans really prefer cyclamates? Most anecdotal evidence indicates that people find cyclamate-flavored foods have an undesirable off-taste, characterized as "thin" or "metallic." Therefore, it seems more plausible to account for human acceptance by two alternatives such as: (i) the advertising campaigns that have stressed the dietary and healthful aspects of cyclamates, thereby motivating human consumption in spite of the undesirable taste, and/or (ii) cyclamates have found their greatest use in foods and beverages that are already highly flavored, such as chocolates, coffee, colas, and citrus drinks, which partially obscure the off-taste.

If one accepts the foregoing account of the basis for human consumption of potentially toxic substances (like

cyclamates), but if one opposes an arbitrary and perhaps hasty ban on a sweetener which has proved helpful to diabetics and others in our society, what are the possible alternatives? How can we capitalize on the sensory dislike people may have for cyclamates to induce a decreased consumption without the correlated arbitrary government action?

If people are being motivated to buy cyclamate-flavored foods primarily by advertising campaigns, it seems more logical to impose existing legal apparatus to prevent misleading or outright fraudulent claims or appeals to the dieting and weight-watching public by marketing agencies. The promises of weight control and sex appeal should be somehow discouraged. At the same time, the public could become the target of an educational campaign stressing proper weight control as well as the potential dangers of excessive cyclamate ingestion.

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Antarctic Ice and Interglacial

High Sea Levels

Emiliani (1) suggests that high interglacial sea levels were the result of significant melting of the Greenland Ice Sheet, caused by the coincidence of perihelion with northern summer solstice. Presumably, therefore, Emiliani must refer to occasions when the mass

of the oceans was greater than it is today; however, his list of high sea levels confuses the issue, because it includes those (for example, at 81,000 and 100,000 years ago) that are thought by many to have been lower than today's (2). The last occasion when, by general agreement, absolute sea level was higher than it is today was about 120,000 years ago, and Emiliani's hypothesis should be examined in the light of events at that time.

What reason is there to believe that the Greenland Ice Sheet was seriously depleted during an interglaciation? The ice sheet appears to be well protected against a moderate temperature rise; it is mostly hemmed in by coastal mountains, so that only a narrow peripheral zone now lies beneath the snow line. Most of the ablation is by calving, not melting. Emiliani's hypothesis to account for melting of the Greenland Ice Sheet cannot be used as evidence that melting did, in fact, take place. Thus his sole evidence is high interglacial sea level, and here the possible role of Antarctic ice cannot be ignored, even if current astronomical hypotheses are unable to explain its melting at that time.

Emiliani summarily dismisses the Antarctic Ice Sheet as a factor in controlling interglacial high sea levels on the grounds that its situation in high latitudes and centered on the pole precludes significant interglacial ablation. This is probably valid for about ninetenths of the ice sheet-that on the continent of East Antarctica-but not for the peculiar ice cover that is grounded far below sea level in West Antarctica. Emiliani ignores an earlier hypothesis (3) that sought to explain interglacial high sea levels by the deglaciation of West Antarctica. This hypothesis pointed out that fringing ice shelves, which are essential for the continued existence of an ice sheet grounded far below sea level, must consist of "cold" ice below the pressure melting point, and will rapidly disintegrate by calving if the average temperature of the warmest month rises above freezing point at sea level. An ice sheet on land, on the other hand, can consist of temperate ice at the pressure melting point and is able to survive under much warmer conditions. Thus the ice of West Antarctica is not only much less tolerant of summer warmth than is the ice cover of either East Antarctica or Greenland, but it would also disintegrate faster under adverse temperature conditions.

Significantly, there is evidence apart from high interglacial sea level that temperatures in Antarctica were too high for the survival of the West Antarctic ice during at least one Northern Hemisphere interglaciation (3, 4). [Unfortunately, because neither isotopic dates nor conclusive stratigraphic information is available, these warm episodes cannot be assigned to a particular interglaciation]. Disappearance of this ice would have raised sea level by 4 to 5 m. Much evidence indicates an absolute sea level of about + 6 m120,000 years ago (2, 3), suggesting that if the high sea level was glacially controlled, it was mainly the result of the deglaciation of West Antarctica, not of Greenland.

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I misinterpret nothing. Absolute dating shows perihelion coincidence for Greenland, anticoincidence for Antarctica. So it must be Greenland. However, West Antarctica could contribute by responding to albedo changes produced by events in Greenland.

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Herbicide Usage

Galston (1) has called for additional restrictions on use of phenoxy herbicides. His recommendation is based on preliminary evidence that these chemicals may be teratogenic (2). Galston says that a pregnant women might be continuously exposed to 1 mg of herbicide per kilogram of body weight per day as a result of water contamination from defoliation operations in Vietnam. His estimate is based on an application rate of 27 pounds of phenoxy herbicide per acre (1 lb/acre = 1.04 kg/hectare).

We have monitored a number of normal operational aerial spray projects in

cool, temperate forests of the Pacific Northwestern United States where application rates of phenoxy herbicides do not exceed 4 lb/acre. Here, spray residues do not move into water in a constant process of contamination. During chemical brush-control operations, small amounts of stream contamination occur during aerial application, but once this water leaves the treatment area, movement of additional herbicides to the stream is negligible (3). A combination of rapid degradation (4) and resistance to leaching (5) prevents stream contamination by herbicide residues after heavy rains. The likelihood of chronic exposure of man or animals to phenoxy herbicide residues from forest spraying seems remote (6).

The treatments that showed the teratogenic capacity of the phenoxy herbicides consisted of repeated high doses in mice and rats. Data pointing to human health hazards are limited in scope and qauntity; more information is needed on the effects of phenoxy herbicides on humans. Toxicity is related to magnitude and duration of dose, and the assumption of chronic persistence and continuous flow of phenoxy herbicides into water is not supported by the available records. A long history of field use of phenoxy herbicides has demonstrated that they interfere little with the quality of the environment (7).

Regulatory agencies are under increasing pressure to restrict the use of some pesticides. An adequate assessment of hazard requires consideration of both the likelihood of exposure to a significant dose and the toxicity of the chemical. Any decision to further restrict the use of phenoxy herbicides in the United States must be based on careful consideration of risks, both of use and nonuse, of these valuable chemical tools.

The scientific community certainly should support restrictions on the hazardous use of any chemical. Equally, this community should support retention of chemical land management tools that research and long experience have demonstrated to be safe. Our studies of normal use of herbicides in Pacific Northwest forests indicate that further restrictions on these uses of the phenoxy herbicides are not justified.

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