norepinephrine-depleted rats in the previous experiment (Table 1).

It could be argued that after a nearly complete depletion of norepinephrine an additional small and undetectable decrease in norepinephrine might surpass some critical threshold and lead to the release of eating. However, if this were true we would also expect this threshold would have been surpassed in at least some of the more than 150 rats in our laboratory which have undergone VNAB destruction via 6-OH-DA injection or electrolytic lesions in previous experiments. In actuality, seven of the eight animals in the combined lesion group ate more food per day than any of the previous VNAB animals studied in this laboratory.

Our results would explain why Gold's (*5*) most effective hypothalamic lesions coincided with the distribution of the ventral bundle. Lesions in the medial hypothalamus that destroy portions of the diffuse projections of the VNAB should lead to exceptional hyperphagia such as that observed with dual lesions in the present study.

Thus, medial hypothalamic hyperphagia and ventral bundle hyperphagia are separable phenomena; the evidence is: (i) norepinephrine loss caused hyperphagia only at night and less hyperphagia overall, (ii) hypothalamic lesions caused overeating without norepinephrine depletion, and (iii) the two forms of destruction combined produced a level of hyperphagia equal to or greater than the sum of their separate effects. In addition, other studies from this laboratory (*1*) indicate that hyperphagia following norepinephrine depletion is dependent on pituitary function, is not associated with finickiness, and decreases rather than increases amphetamine anorexia. In these respects, also, the syndrome is different from classical hypothalamic hyperphagia (*11*). This suggests that classical lesions that produce hypothalamic hyperphagia disrupt other systems involved in satiety besides the VNAB.

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Nomenclature of Eukaryotic DNA Polymerases

An international conference on eukaryotic DNA polymerases, organized by David Korn of Stanford University, was held at the Asilomar Conference Center, Pacific Grove, California, 11 to 15 May 1975. This meeting was attended by 48 scientists who discussed recent data concerning various DNA polymerases found in a number of eukaryotic species. Because of the many proposals for systems of naming the DNA polymerases, those attending the conference decided to establish a uniform system of nomenclature. It was decided to include only those enzyme classes for which there is common agreement that they represent distinct entities. Four such polymerase classes were identified. A fifth class of enzyme, the reverse transcriptases or RNA tumor virus–associated DNA polymerases, is distinctive enough not to need further identification (*1*). Other virus-induced DNA polymerases, such as herpes-induced DNA polymerase (*2*) or vaccinia-induced DNA polymerase (*3*), can be referred to by the name of the causative virus.

Consideration was given to devising a nomenclature connoting the size, cellular localization, or template preference of the various DNA polymerases. Such approaches were abandoned because of ambiguities and uncertainties inherent in them. Instead, it was decided that a system of Greek letters, assigned to the DNA polymerases according to the order of discovery, would be the most useful approach because it would differ from any of the systems previously used by ourselves and others.

The nomenclature proposed is as follows (*4*):

1) *DNA polymerase-α* is proposed for the high-molecular-weight (>100,000) DNA polymerase, which was the first to be detected in mammalian cells (*5*). Even though this enzyme is often the major activity in cytoplasmic extracts of growing cells, its actual location in vivo may be in the nucleus. It is particularly active in copying "activated" double-stranded DNA (*6*), and has almost no ability to copy the oligo-homopolymer $A_n \cdot dT_{15}$. The α-polymerase is strongly inhibited by reagents that block sulfhydryl groups, and isoelectric focusing experiments show it to be an acidic protein. It is free of nuclease activity.

2) *DNA polymerase-β* is suggested for the low-molecular-weight (< 50,000) enzyme recovered almost entirely in nuclear extracts (*7*). This enzyme is a basic protein and is resistant to reagents that block sulfhydryl groups. It copies activated DNA well and, to a somewhat lesser extent, the synthetic template $A_n \cdot dT_{15}$. It contains no detectable nuclease activity.

Table 1. Systems of naming DNA polymerases.

New system	Formerly used systems				
	Baltimore (*12*)	Bollum (*13*)	Gallo (*14*)	Korn (*15*)	Weissbach (*10*)
Polymerase-α	C	6*S* to 8*S* (maxi)	I	Cytoplasmic N_2	II
Polymerase-β	N	3.45 (mini)	II	N_1	I
Polymerase-γ	A		III		R

3) *DNA polymerase-γ* is suggested for the most recently described DNA polymerase that copies $A_n \cdot dT_{15}$ with high efficiency, but does not copy DNA well (*8*). This enzyme has been called R-DNA polymerase to suggest its propensity for copying synthetic polyribonucleotides, but no evidence exists at present as to its ability to copy natural RNA. Polymerase-γ is acidic, has a molecular weight >100,000, and requires sulfhydryl-containing compounds for maximal activity.

4) *Mitochondrial DNA polymerase* (DNA polymerase-mt) is an enzyme separable from the others (*9, 10*); it is named for its subcellular localization, which is so characteristic of the enzyme.

Table 1 shows how α, β, and γ have been named in some previous publications by some workers.

The nomenclature of DNA polymerases -α, -β, -γ, and -mt is being and will be utilized by us in the future (*11*). As new or different eukaryotic DNA polymerases are identified, the system could be expanded by the use of additional Greek letters or other symbols. It is our hope that others will utilize this system in order to avoid ambiguity.

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Histamine and Radiation-Induced Taste Aversion Conditioning

Levy, Carroll, Smith, and Hofer (*1*) reported that rats treated with an antihistamine do not subsequently develop radiation-induced taste aversions, and suggested that radiation-induced histamine production is the critical event responsible for the formation of radiation-induced taste aversions. In an attempt to evaluate the generality of this conclusion, I have found that if the Levy *et al.* experiment is repeated with a slight alteration of design, the administration of antihistamine potentiates, rather than blocks, radiation-induced taste aversions.

The design employed by Levy *et al.* was exceptional in that the initial exposure to the taste stimulus, a 0.1 percent saccharin solution, occurred after drug treatment and irradiation. The typical taste aversion design would have called for the initial saccharin ingestion before any drug or radiation treatment. This would have precluded the possibility that the administration of the drug prior to drinking might have interfered with conditioning by making the animals ill before they tasted the saccharin. I have conducted an experiment similar to that of Levy *et al.*, but following a design in which the antihistamine was administered after the ingestion of the saccharin solution and before exposure to radiation.

The procedures described by Levy *et al.* were followed as closely as possible, with the exception that the four groups of seven water-deprived rats were allowed 20-minute access to the saccharin solution *before* they were injected (intraperitoneally) with saline or the antihistamine chlorpheniramine maleate (20 mg/kg). Irradiation with 105 roentgens of x-rays or sham irradiation followed the injections. Twenty-four hours later, the rats were again allowed to drink saccharin solution. The saline-injected, sham-irradiated group drank 16.7 ml (mean) during this test. The saline-injected, irradiated group drank 5.2 ml, demonstrating their conditioned aversion for the solution. The drug-treated, sham-irradiated group also showed a significant aversion, drinking only 7.5 ml. The drug-treated, irradiated group drank 1.8 ml, significantly less than either group that received drug or radiation alone (*2*).

There are two points of disagreement between these results and those of Levy *et al.* First, their chlorpheniramine-treated, sham-irradiated animals did not develop aversions, while the corresponding animals in my study did. Also, their drug-treated, irradiated animals did not develop aversions, while the corresponding group in my study showed the most severe aversions of all, contrary to what the Levy *et al.* histamine hypothesis would have predicted.

The timing of the chlorpheniramine injections appears to be the crucial difference between the two studies. It is clear from the present results that chlorpheniramine maleate itself produces taste aversions when injected after saccharin consumption. In addition, many of the rats injected with the drug showed abdominal muscular spasms and assumed abnormal postures within 10 minutes after their injections, indicating toxic effects of the drug. It may be suggested then, that both chlorpheniramine-treated groups in the Levy *et al.* experiment did not learn aversions because they were already ill when they first tasted the saccharin solution. Thus, the effect of chlorpheniramine illness and not attenuation of radiation-induced histamine release provides the best explanation of the results of both studies.

Levy *et al.* clearly recognized this issue, for in another experiment they effectively showed that prior treatment with chlorpheniramine did not interfere with taste aversion conditioning resulting from lithium chloride poisoning. However, this does not rule out the possibility that the prior treatment with chlorpheniramine in the original experiment could have resulted in toxic interference with conditioning when x-rays were used.

Taken together, the present work and the Levy *et al.* experiment provide examples of an inherent methodological problem involved in attempting to demonstrate radioprotective properties of drugs, with the use of taste aversion as the dependent