

390. Alpha tocopheryl-*p*-quinone was prepared by the oxidation of natural or synthetic alpha tocopherol with ferric chloride (2) and purified by the method of Tishler and Wendler (3) to an $E_{1\text{cm}}^{1\%}$ (268 mμ) in iso-octane of 450.

References

1. EMMERIE, A. and ENGEL, C. *Rec. trav. chim.*, 1938, **57**, 1351.
2. EVANS, H. M., EMERSON, O. H., and EMERSON, G. A. *J. biol. Chem.*, 1939, **131**, 409.
3. GOLUMBIC, C. *Oil and Soap*, 1943, **20**, 105.
4. HOVE, E. L. and HARRIS, P. L. *J. Nutrition*, 1947, **33**, 95.
5. JOHN, W., DIETZEL, E., and EMTE, W. *Z. physiol. Chem.*, 1939, **257**, 173.
6. MACKENZIE, C. G. and MCCOLLUM, E. V. *Science*, 1939, **89**, 370.
7. MILHORAT, A. T. *et al.* *Ann. N. Y. Acad. Sci.*, 1949, **52**, Article 3.
8. TISHLER, M. and WENDLER, N. L. *J. Amer. chem. Soc.*, 1941, **63**, 1532.

Taste Blindness to Phenyl-Thio-Carbamide as a Function of Saliva

Jozef Cohen and Donald P. Ogdon

Department of Psychology, University of Illinois

In 1931, A. L. Fox (6, 7) of the E. I. du Pont de Nemours Company discovered that approximately 60% of the Causasian population are able to taste phenyl-thio-carbamide (P. T. C.) as bitter, while the remaining 40% find it to be as tasteless as chalk. The literature of the physiological, genetic, environmental, and ethnological characteristics of this phenomenon has been reviewed by Cohen and Ogdon (3).

Blakeslee (1), Fox (4), Mee (5), and Blakeslee and Salmon (2) have all suggested that it may be the saliva which is the determiner of the ability to taste or not, and not the "taste apparatus" itself. Fox specifically suggested the possibility that nontasters have in their saliva a product, possibly a protein or a colloid, which precipitates the P. T. C. as a very insoluble substance which does not give rise to a taste sensation.

The question as to the effect of the saliva may be settled by allowing tasters and nontasters to taste P. T. C. using another taster's or nontaster's saliva. A population of 35 American college students was first tested with P. T. C. crystals and each individual classified as a taster or a nontaster. Then each person (whether taster or nontaster) was tested with each of the following tests: 1) D test, using a saturated solution of P. T. C. in tap water on a dry (dried by air from an atomizer) tongue; 2) G test, using a saturated solution of nontaster's saliva on a dry tongue; 3) P test, using a saturated solution of P. T. C. in taster's saliva on a dry tongue. The taster's and nontaster's saliva was obtained from other individuals selected purely at random. No saliva from any person was used for more than one test on more than one subject. The results are given in Table 1.

As a control, 19 individuals were tested with the R test, using saturated solutions of P. T. C. in their own

saliva, which had been allowed to remain exposed to air about 5-10 min, on a dry tongue. The results are given in Table 2.

TABLE 1

SENSATIONS OF 35 OBSERVERS TO P. T. C. DISSOLVED IN WATER, AND TASTER'S AND NONTASTER'S SALIVA ON A DRY TONGUE

	D Test	G Test	P Test
26 Tasters*	T=0 NT=26	T=0 NT=26†	T=0 NT=26
7 Nontasters	T=0 NT=7	T=0 NT=7	T=0 NT=7‡

T = tastes bitter.

NT = no taste.

* Four subjects reported only weak taste of bitter.

† One subject reported slight sensation, but was unable to describe it.

‡ One subject reported a "warm" sensation.

These data seem to indicate that an individual will taste P. T. C. as bitter when the following two necessary conditions are met: 1) He must have the correct "taste apparatus," and 2) he must have his own saliva (or,

TABLE 2

SENSATIONS OF 19 OBSERVERS TO P. T. C. DISSOLVED IN THEIR OWN SALIVA ON A DRY TONGUE

	R Test
17 Tasters	T=16 NT=1*
2 Nontasters	T=0 NT=2

* This subject had previously reported a weak bitter taste to the crystals.

presumably, its chemical equivalent). A nontaster cannot taste in any event, even when he uses the saliva of another taster. A taster cannot taste under any circumstances, except when he uses his own saliva; he cannot taste if he uses the saliva of another taster or nontaster. He can taste if he uses his own saliva, even though the saliva is placed on his tongue in exactly the same manner as the saliva from another individual. No subject can taste P. T. C. when the crystals are dissolved in water and no saliva is used at all.

Salivas are probably as different as fingerprints. Blakeslee and Salmon (2) have also found that saliva is important to taste P. T. C. Our finding that it must be the individual's own saliva may be brought about by the fact that the "taste apparatus" becomes, over the years, extremely sensitive and specialized to the particular saliva which the individual possesses; or these differences in saliva may be congenital or genetic. This being the case, when other saliva is introduced, it is equivalent to water, and no taste sensation results.

As a check on whether saliva influences other tastes, 27 observers were tested with saturated solutions (in water) of saccharin and salt on dry tongues, and on tongues wet with saliva. All of the individuals were able

to taste both the salt and the saccharin using wet tongues, but 16 failed to detect the saccharin and nine failed to detect the salt with a dry tongue. Apparently, therefore, saliva aids in many taste sensations, but its effect is most pronounced with P. T. C.

References

1. BLAKESLEE, A. F. *Science*, 1931, **74**, 607.
2. BLAKESLEE, A. F. and SALMON, T. N. *Proc. nat. Acad. Sci.*, Wash., 1935, **21**, 78.
3. COHEN, J. and OGDON, D. P. *Psychol. Bull.*, in press.
4. FOX, A. L. *Proc. nat. Acad. Sci.*, Wash., 1932, **18**, 115.
5. MEE, A. J. *Sci. Progr. Twent. Cent.*, 1934, **29**, 228.
6. ANON. *Science*, 1931, **73**, no. 1894, suppl., 14.
7. ANON. *Sci. News Lett.*, 1931, **19**, 249.

Sex Influence on Embryonic Death Rate in Chicks

F. A. Hays

Massachusetts Agricultural Experiment Station,
Massachusetts State College, Amherst

Thornton (7) reported evidence from this laboratory that the death rate in chick embryos up to 5 days of age was greater in females than in males. Landauer and Landauer (5) in summary data showed that the sex ratio of chicks at hatching was 48.77. Byerly and Jull (1) reported the sex of embryos that died after 9 days of incubation to be 48.59% males. These data would suggest that the mortality rate during this period was higher in females. Hays (2) gave the sex ratio in chicks at 8 weeks of age as 50.85% males. Hays (3) showed that the primary sex ratio in chickens is about 50-50, with considerable variation between families. In general, observations of different workers suggest that there may be considerable variation between breeds and strains with respect to sex ratio (6).

Data collected in the spring of 1949 on the Massachusetts Experiment Station flock of Rhode Island Reds strongly indicate that among embryonic deaths up to 5 days of incubation there is a higher incidence in females than in males. In the fowl the female is the heterogametic sex, and the reduced ratio in females corresponds to the reduced ratio of males reported for most animals where the male is the heterogametic sex.

The data presented in the table include 5450 eggs set in six weekly hatches, including eggs laid from February

5 to March 25, there being a 1-week interval in which eggs were not saved between the third and fourth hatches.

TABLE 1

Hatch	Egg production	Total embryonic mortality %*	Early embryonic mortality %†	Sex ratio at 8 weeks
1	1337 (F5-11)	20.5	19.8	50.0
2	1238 (F12-18)	27.5	28.7	50.0
3	1097 (F19-25)	32.4	55.0	56.6
4	991 (M5-11)	20.8	32.5	52.8
5	948 (M12-18)	24.1	31.1	53.4
6	921 (M19-25)	27.6	38.4	56.5

* Based on fertile eggs.

† Percentage of embryos that died early.

A very mild epidemic of bronchitis appeared in the breeding pens soon after the collection of hatching eggs began. This disease outbreak caused a linear decline in production during the period, in contrast to the normal rapid increase expected at this season (4). Effects of the disease were observed both on fertility and embryonic death rate. Sex of the chicks was not determined until they were 8 weeks of age, but the postincubation death rate was low in the 3200 chicks retained.

The table shows that total embryonic mortality did not increase greatly through the hatching season, but the early embryonic death rate almost doubled as the season progressed. This observation suggests that the disease virus had a lethal effect which operated early in the development of the chicks. The last column gives the percentage of survivors at 8 weeks of age that were of the male sex. The abnormally high percentage of males from the last four hatches strongly indicates that the majority of embryos that died early must have been females. These data, together with those of Byerly and Jull (1), show that the embryonic death rate in females is higher than in males, at all stages of embryonic development.

References

1. BYERLY, T. C. and JULL, M. A. *Poultry Sci.*, 1935, **14**, 217.
2. HAYS, F. A. *Amer. Nat.*, 1941, **75**, 187.
3. ———. *Amer. Nat.*, 1945, **79**, 184.
4. ———. *Poultry Sci.*, 1949, in press.
5. LANDAUER, W. and LANDAUER, ANNA B. *Amer. Nat.*, 1931, **65**, 492.
6. ———. *Conn. agric. exp. Sta. Bull.*, 1948, 262.
7. THORNTON, E. J. *Possible genetic factors and embryonic mortality in relation to the sex ratio of chicks at hatching*. Thesis, Mass. State College.