DISPUTANDUM

Conformational Control of Bond Migration in the D-Homo Rearrangement of 17-Hydroxy-20-Keto Steroids

In a recent paper[1] with the above title, it is claimed that 'a convincing explanation for the differences in bond migration invoked by reagent as well as by steroid configuration at C-17 [in 17-hydroxy-20-keto steroids] has not yet appeared' and 'it is proposed that the factor controlling the bond migration in the rearrangement of these systems is a conformational one'.

As far as can be ascertained this is the first time that this 'unified rationale for the observed phenomena[1], has been published in English. The same suggestion was, however, made some years ago in French[2] and experimental support for it has since been obtained in the case of the Lewis acid catalysed rearrangement[3].

IRÈNE ELPHIMOFF-FELKIN

Centre National de la Recherche Scientifique, Paris, July 7, 1959.

[1] N. L. WENDLER, D. TAUB, and R. FIRESTONE, Exper. *15*, 237 (1959).

[2] I. ELPHIMOFF-FELKIN, Bull. Soc. chim. *1956*, 1845. See p. 1849: *Influence de la conformation de l'état de transition sur le résultat de la réaction.*

[3] I. ELPHIMOFF-FELKIN and A. SKROBEK, C. R. Acad. Sci., Paris *246*, 2497 (1958); Chem. Abstr. *52*, 18524 (1958). Details in Bull. Soc. chim. *1959*, 742.

In our recent article of the above title in Experientia we regret that through inadvertence on our part, we failed to cite an earlier exposition on this subject by I. ELPHIMOFF-FELKIN (Bull. Soc. chim. *1956*, 1845) dealing with the application of this concept to the Lewis-acid catalyzed rearrangement.

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Merck & Co., Rahway, N. J., July 27, 1959.

Informations - Informationen - Informazioni - Notes

STUDIORUM PROGRESSUS

On Factors Involved in the Mechanism of 'Taste-Blindness'

By R. FISCHER and F. GRIFFIN*

Taste sensitivity to phenylthiourea, also called phenylthiocarbamide (PTC), and to other structurally related anti-thyroid compounds has been shown to follow a bimodal distribution[1,2]. Recent data[3] confirm the 7:3 ratio of tasters to non-tasters in a Caucasian population of adults and are in agreement with the genetic hypothesis that non-tasting is a single recessive character.

It has also been reported[4] that it is the individual's own saliva that is necessary for tasting PTC and related compounds. The nature of this 'saliva factor', however, was not elucidated. FAWCETT and KIRKWOOD[5] have presented evidence as to the presence of a soluble enzyme system, tyrosine iodinase[6], in the parotid and submaxillary glands, and asked emphatically: 'What on earth is this enzyme system doing in the salivary glands?[8]. However, a connection between tyrosine iodinase and the 'saliva factor' was not suspected.

Recently, while confirming the existence of the disputed[9,10] 'saliva factor' (to be published) we were fortunate in obtaining (through the courteous assistance of Dr. S. GARN, Yellow Springs, Ohio) a paper of N. TURNER *et al.*[11] reporting protein bound iodine (PBI) and total iodine values in the saliva of thirty children 8–14-years old from among the school population brought to the Forsyth Dental Infirmary for Children for dental examination and treatment. When analyzing the distribution of free iodine (which we obtained by deducting the PBI from the total iodine for each child) in the saliva of TURNER's subjects, we found that it resembled that of taste blindness to PTC and related compounds measured in young individuals[12]. It is probable that the resemblance would be more striking if TURNER's subjects were a random sample rather than a 'dental' population and if the age group of both populations were exactly matched; the reason for this being that taste threshold for PTC is apparently distributed on a continuum at birth and becomes only gradually bimodal during the growth process.

Moreover, an examination of the data of WOLFE and TURNER[13] shows that the amount of salivary peroxidase

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[1] H. HARRIS, *An Introduction to Human Biochemical Genetics*, Chap. VIII (Cambridge University Press, London 1955), p. 69.

[2] J. MOHR, Ann. Eugen., London *16*, 282 (1951).

[3] B. B. MERTON, Acta Genet. statist. med. *8*, 114 (1958).

[4] J. COHEN and D. P. OGDON, Science *110*, 522 (1949).

[5] D. M. FAWCETT and S. KIRKWOOD, J. biol. Chem. *209*, 249 (1954).

[6] Which needs cupric ion and tyrosine to synthetize free monoiodotyrosine; the system also requires hydrogen peroxide for the peroxidation of iodide[7].

[7] N. M. ALEXANDER, J. biol. Chem. *234*, 1530 (1959).

[8] S. KIRKWOOD, Chem. Canada *7*, 23 (1955).

[9] W. C. BOYD, Psychol. Bull. *48*, 71 (1951).

[10] J. COHEN and D. P. OGDON, Psychol. Bull. *48*, 1 (1951).

[11] N. C. TURNER, P. H. DEMOGE, J. T. ANDERS, and G. E. CROWELL, N. Y. State dent. J. *22*, 134 (1956).

[12] H. HARRIS and H. KALMUS, Ann. Eugen., London *15*, 24 (1949).

[13] A. D. WOLFE and N. C. TURNER J. dent. Res. *36*, 843 (1957).