3 Chogan, G.A., Papsidero, L.D., Valenzuela, L.A., Nemoto, T., Penetrante, R., and Chu, T.M., Tissue distribution of an epithelial and tumor-associated antigen recognized by monoclonal antibody F36/22. Cancer Res. *43* (Oct. 1983) 4980–4988.
4 Epenetos, A.A., Britton, K.E., Mather, S., Shepard, J., Granowska, M., Taylor-Papamitriou, J., Nimmon, C.C., Durbin, H., Hawkins, L.R., Malpas, J.S., and Bodmer, W.F., Targeting of Iodine-123-labelled tumor associated monoclonal antibodies to ovarian, breast, and gastrointestinal tumors. Lancet *2* (Nov. 1982) 999–1005.
5 Köhler, G., and Milstein, C., Continuous cultures of fused cells secreting antibody of predefined specificity. Nature *256* (1975) 495–497.
6 Mach, J.P., Chatal, J.F., Lumbroso, J.D., Buchegger, F., Forni, M., Ritschard, J., Berche, C., Douillard, J.Y., Carrel, S., Herlyn, M., Steplewski, E., and Koprowski, H., Tumor localization in patients by radiolabeled monoclonal antibodies against colon carcinoma. Cancer Res. *43* (Nov. 1983) 5593–5600.
7 Marjani-Constantini, R., Colnaghi, M.I., Leoni, F., Menard, S., Cerasoli, S., and Rilke, F., Immunohistochemical reactivity of a monoclonal antibody prepared against human breast carcinoma. Virchows Arch. (A) *402* (1984) 389–404.
8 McGee, J., Woods, J.C., Ashall, F., Bramwell, M.E., and Harris, H., A new marker for human cancer cells. 2. Immunohistochemical detection of the Ca antigen in human tissues with the Ca1 antibody. Lancet *2* (1982) 7–10.
9 Pallesen, G., Jepsen, F.L., Hastrup, J., Ipsen, A., and Hvidberg, N., Experience with the oxford tumor marker (Ca1) in serous fluids. Lancet (June 1983) 1326.
10 Saiki, O., and Ralph, P., Clonal differences in response to T cell replacing factor (TRF) for IgM secretion and TRF receptors in a human B lymphoblast cell line. Eur. J. Immun. *13* (1983) 31–34.
11 Soule, H.R., Linder, E., and Edginton, T.S., Membrane 126-kilodalton phosphoglycoprotein associated with human carcinomas identified by a hybridoma antibody to mammary carcinoma cells. Proc. natn. Acad. Sci. USA *80* (1983) 1332–1336.
12 Stähli, C., Staehelin, T., Miggiano, V., Schmidt, J., and Häring, P., High frequencies of antigen-specific hybridomas: dependence on immunization parameters and prediction by spleen cell analysis. J. immun. Meth. *32* (1980) 297–304.
13 Stähli, C., Staehelin, T., and Miggiano, V., Spleen Cell Analysis and optimal immunization for high-frequency production of specific hybridomas. Meth. Enzym. *92* (1983) 26–36.
14 Stähli, C., Miggiano, V., Stocker, J., Staehelin, T., Häring, P., and Takacs, B.J., Distinction of epitopes by monoclonal antibodies. Meth. Enzym. *92* (1983) 242–253.
15 Stocker, J., Pers. comm.
16 Stocker, J., and Heusser, C., Methods for binding cells to plastic: application to a solid-phase radioimmunoassay for cellsurface antigens. J. immun. Meth. *26* (1979) 87–95.
17 Takacs, B.J., and Staehelin, T., Biochemical characterization of cell surface antigens using monoclonal antibodies, in: Immunological Methods,. vol. 2, pp. 27–56. Eds I. Lefkovits and B. Pernis. Academic Press, London 1981.
18 Towbin, H., Staehelin, T., and Gordon, J., Electrophoretic transfer of proteins from polyacrylamide gels to nitrocellulose sheets: Procedure and some applications. Proc. natn. Acad. Sci. USA *76* (1979) 4350–4354.

0014-4754/85/111377-05$1.50 + 0.20/0

Deletion mapping of a new gustatory mutant in *Drosophila melanogaster*

M. Morea

Institute of Genetics, University of Bari, via Amendola 165/A, I-70126 Bari (Italy), 3 December 1984

Summary. A gustatory mutant of *Drosophila melanogaster* insensitive to the taste of salt has been isolated. Genetic crosses and a deletion mapping analysis show that this mutation, designated *gust-M_1* is located in the $93C_{3-6}$–$93D_{6-7}$ region of the third chromosome. *gust-M_1* is also insensitive to the taste of quinine sulfate. The behavior of this mutant may be explained by assuming that *gust-M_1* could be a mutation perturbing functions in the central nervous system affecting the responses to both compounds.
Key words. Behavior genetics; deletion mapping; taste; chemotaxis.

Introduction

In Diptera, the contact chemoreceptors that mediate feeding behavior are primarily located on both the tarsal segments of the leg and the labellum of the proboscis[6]. When these chemoreceptors are in contact with solutions of various sugars, a hungry fly generally extends its proboscis, whereas sugars mixed with salts elicit no proboscis extension.

During the last decade several researchers have isolated gustatory mutants of *Drosophila melanogaster*, which are deficient in their responses to sugars, salt or quinine[5, 10, 19, 20, 23, 24, 26]. Falk and Atidia[10] reported three mutations affecting the tolerance to the taste of salt. They were designated '*Lot*' and were located on the X-chromosome; the mutant flies showed reduced salt sensitivity. In particular, it was found that the mutation designated *'Lot-94'* is 'a mutation of a perception center, affecting the recognition of salt, with no detectable effect on that of sugar'[5].

Mutants insensitive to either NaCl or to quinine or to both compounds have also been described by Tompkins[24, 26]. Some of them are temperature-sensitive[25].

Siddiqi and Rodrigues have isolated mutations in an X-linked gene, *gust-A*, that block the responses of *Drosophila melanogaster* to a group of pyranose sugars[20]. It has been shown that the behavioral effects of this mutation are correlated with a loss of electrical responses in taste receptors. The mutation affects the chemoacceptors for pyranose sugars leaving the furanose acceptors intact. Mutants insensitive to NaCl and to quinine were also isolated by Siddiqi and Rodrigues[22]. They tested the electrophysiological responses of the mutants and found that the receptor responses of the mutants were indistinguishable from those of the wild-type. Therefore Siddiqi and Rodrigues think that 'the blocks produced by these genes are presumably central'[22].

Recently I have started a study of the taste responses of *ebony* flies, which show a number of behavioral abnormalities[3, 4, 8, 11–13]. Different alleles of *ebony* have been

tested; the stocks examined, raised at 25 °C ± 1 °C, have been wild in their responses to sucrose and to NaCl (data not presented here). During this study the *In(3R)AFA,e* stock appeared mutant to the taste of NaCl and quinine sulfate; the *In(3R)AFA,e* males respond abnormally to NaCl with mistactic behavior (i.e. they are attracted by the stimulus in contrast to *Canton-S* males that avoid NaCl); in addition, the *In(3R)AFA,e* males show also a mistactic response to quinine sulfate (i.e. they are attracted to quinine sulfate in contrast to *Canton-S* males, which reject the quinine salt)[26]. I have earlier reported preliminary results on this mutant[17]. In the present work I describe behavioral experiments carried out to elucidate the nature of this mutant designated *gust-M_1*. Further, using genetic methods I have tried to locate the gene for *gust-M_1*.

Methods

Genetic strains

Drosophila stocks were maintained at 25 °C ± 1 °C on a standard maize, sugar, water, agar and yeast medium, under constant illumination. The *wild-type* strain used here was *Canton-S*. Chromosome localization and mapping experiments utilized the *SM5/Bl;TM1/H,* the *In(3LR)TM1, Mè ri cu sbd²/1(3R) cu*^{D9} and the *'rucuca'* stock homozygous for eight recessive third-chromosome markers (i.e. *ru h th st cu sr e*^{s} *ca*)[15]. The deficiencies used in these studies are: *Df(3R)e*^{GC9} described by Mohler and Pardue[16], *Df(3R)e*^{D7} described by Scalenghe and Ritossa[21] and *Df(3R)e*^{N12} with the left break point in $93B_{1-2}$ and the right break point that does not remove the puff locus in $93D_{6-7}$ region; this last deficiency was obtained by Ritossa (personal communication). Further information on genetic symbols and terminology is given by Lindsley and Grell[15].

Behavioral assay of adults

To test the responses of individual flies to sucrose, salt and quinine, *assays of proboscis extension* were performed according to Deak[2], with some minor modifications concerning the molarities of test solutions. After being starved, attached to a slide, and water satiated, a fly was tested as follows: five trials on the tarsus of left leg with 0.01 M sucrose, 10 min wait, during which the tarsus was washed, and the fly water satiated, five trials on right leg with 1 M NaCl+0.01 M sucrose (Mix) or 0.01 M quinine sulfate + 0.01 M sucrose (QS). Behavioral assays were also performed on the right leg with 0.01 M sucrose and, after 10 min, on the left leg with Mix or QS. No significant difference between the two groups was observed; in fact in the latter case the 92% and the 89% of *In(3R)AFA,e* males responded positively to Mix and to QS respectively.

Statistics

Each experiment was replicated m times, and each time n_i flies were observed. Statistical analysis was performed assuming that the distribution of positive responses to a test solution is of the Poisson's type. Then, for the statistical analysis, I have used the transformation $Z = (X+0.50)^{1/2}$, where X = number of positive responses to a test solution in each replication. The mean was calculated weighting each Z value with the number of flies (n_i) of each replication, thus:

$$\bar{z} = \frac{\Sigma z_i n_i}{\Sigma n_i}$$

and, in the same way, the SD:

$$s_z = \Sigma (z_i - \bar{z})^2 n_i / \Sigma n_i \cdot m/(m-1)^{1/2}.$$

Converting again Z average value into the number of positive responses it turns out that

$$\bar{z} = \bar{z}^2 - 0.50$$

from which percent positive responses to a test solution is $\bar{p} = \frac{\bar{x}}{\bar{n}}$ where $\bar{n}$ is the mean of n_i.

p_{min} is the minimum proportion of flies showing positive responses to a test solution;

p_{max} is the maximum proportion of flies showing positive responses to a test solution.

Results

Responses of gust-M_1

As shown in table 1 the *gust-M_1* mutation was found in the *In(3R)AFA,e* stock selected as an *ebony (e)* mutation after X-irradiation of *Canton-S* males[1]. The mutant is unable to detect NaCl and quinine, but its response to sucrose is normal. About 89% of the males of the *In(3R)AFA,e* stock responded positively to 1 M NaCl in 0.01 M sucrose; when the leg tarsi of *Canton-S* were similarly stimulated only 18% of the flies responded positively (table 1).

Genetic mapping of gust-M_1

Virgin females from the *In(3R)AFA,e* stock were mated to *SM5/Bl;TM1/H* males. F_1 males of the genotype +/*SM5; In(3R)AFA,e/H* and F_1 males of the genotype +/*Bl;In(3R)AFA,e/TM1* were mated to virgin F_1 females of

Table 1. Response of wild-type (*Canton-S*) and mutant *In(3R)AFA,e* males to test solutions

	m	p_{min}	p_{max}	$\bar{z}$	s_z	$\bar{x}$	$\bar{n}$	$\bar{p}$
a) *Canton-S*								
Suc	6	0.75	0.87	3.65	0.79	12.82	13.33	0.96
Mix	4	0.14	0.22	1.92	0.36	3.19	17.50	0.18
QS	4	0.10	0.17	1.35	0.20	1.32	8.50	0.15
b) *In(3R)AFA,e*								
Suc	8	0.90	1.00	3.60	0.43	12.46	12.75	0.98
Mix	7	0.77	1.00	3.30	0.37	10.39	11.71	0.89
QS	4	0.78	0.87	3.08	0.62	8.99	10.00	0.90

Response of wild-type *(Canton-S)* and mutant *In(3R)AFA,e* males to 0.01 M sucrose (Suc), to 1 M NaCl+0.01 M sucrose (Mix) and to 0.01 M quinine sulfate + 0.01 M sucrose (QS). Proportion of flies showing a proboscis-extension response when tarsi were stimulated by a test solution (see Behavioral assay of adults in 'methods').

the genotype *+/SM5;In(3R)AFA,e/TM1*. From these crosses I selected the following two stocks: *+/+;TM1/H* and *Bl/SM5;In(3R)AFA,e/In(3R)AFA,e*. The proboscis-extension response elicited when *+/+;TM1/H* males were presented with Mix (i.e. 1 M NaCl in 0.01 M sucrose) is shown in table 2: the response was like that of *Canton-S* and *SM5/Bl;TM1/H* flies. On the contrary, *SM5/Bl;In(3R)AFA,e/In(3R)AFA,e* males responded as *In(3R)AFA,e* stock (tables 1 and 2). This suggests that the gene for *gust-M_1* is located on the third chromosome. To map the mutation, virgin *In(3R)AFA,e* females were mated to *rucuca* males. F_1 females from this cross were back crossed to *rucuca* males, and the single crossover progeny was made homozygous by using the *TM1,Mè ri cu sbd²/1(3R)cu*^{D9} balancer stock. *ru h th st* recombinants resulted mutant to salt (table 2). This led me to think that the mutation might be located on the right arm of the third chromosome. The third chromosome of *In(3R)AFA,e* stock shows an inversion with break points in $86C_{4-5}$–$93D_{1-2}$ region. Deletion mapping was therefore tried, using three different deficiencies covering the 93D region: *Df(3R)e*^{GC9}, *Df(3R)e*^{N12} and *Df(3R)e*^{D7}. Table 3 shows that *gust-M_1* expresses itself against *Df(3R)e*^{GC9}, *Df(3R)e*^{N12} and *Df(3R)e*^{D7}; these results lead me to think that *gust-M_1* is located in the $93C_{3-6}$–$93D_{6-7}$ region of the third chromosome (fig.). As one can see on the fourth line of table 3, 35% of *Df(3R)e*^{N12}*/TM3* males positively respond to NaCl; this value is higher than that of *TM3/Df(3R)e*^{GC9} and *TM3/Df(3R)e*^{D7} males. The increase in the response of *TM3/Df(3R)e*^{N12} males to Mix could be due to the genetical background of this stock.

Table 2. Genetic localization of *gust-M_1*

Males	m	p_{min}	p_{max}	$\bar{z}$	s_z	$\bar{x}$	$\bar{n}$	$\bar{p}$
+/+;TM1/H	3	0.11	0.27	1.51	0.39	1.78	8.33	0.21
ru h th st	6	0.71	1.00	3.35	0.52	10.72	12.17	0.88
Sm5/Bl;TM1/H	4	0.15	0.20	1.63	0.28	2.16	11.25	0.19
rucuca	3	0.17	0.27	1.63	0.27	2.16	9.33	0.23
SM5/Bl; In(3R)AFA,e/ In(3R)AFA,e	6	0.82	0.90	2.99	0.28	8.44	9.67	0.87

Response of +/+,TM1/H, *ru h th st*, *SM5/Bl;TM1/H*, *rucuca* and *SM5/Bl;In(3R)AFA,e/In(3R)AFA,e* stocks to 1 M NaCl + 0.01 M sucrose (Mix).

Table 3. Deletion mapping analysis of *gust-M_1*

Males	m	p_{min}	p_{max}	$\bar{z}$	s_z	$\bar{x}$	$\bar{n}$	$\bar{p}$
Df(3R)e^{GC9}*/ In(3R)AFA,e*	5	0.61	0.83	3.18	0.69	9.61	11.00	0.87
Df(3R)e^{N12}*/ In(3R)AFA,e*	4	0.67	0.85	3.57	0.58	12.24	15.25	0.80
Df(3R)e^{GC9}*/ TM3*	4	0.09	0.17	1.22	0.00	0.99	8.50	0.12
Df(3R)e^{N12}*/ TM3*	3	0.33	0.36	2.28	0.12	4.70	13.33	0.35
Df(3R)e^{D7}*/ TM3*	4	0.10	0.14	1.52	0.36	1.81	11.50	0.15
Df(3R)e^{D7}*/ In(3R)AFA,e*	10	0.73	0.85	3.21	0.57	9.80	10.90	0.89

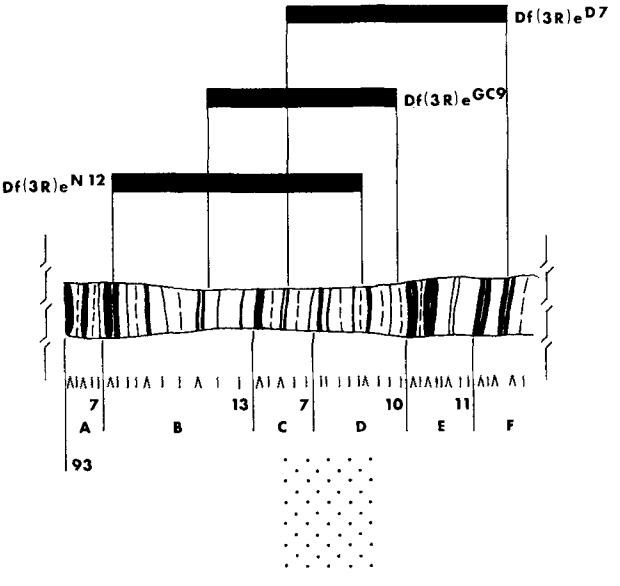
Response to Mix of males bearing the *In(3R)AFA,e* chromosome against three different deficiencies of the 93 region and response to Mix of males with the same deficiencies against balancer third chromosomes.

Discussion

During the study of taste responses of different alleles of *ebony* mutant a gustatory mutant was isolated; this mutant, designated *gust-M_1*, appeared in the *In(3R)AFA,e* stock. Populations of *In(3R)AFA,e* adults are attracted to 1 M NaCl in 0.01 M sucrose. The results of genetic mapping suggested that the gene for *gust-M_1* was located on the right arm of the third chromosome that in the *In(3R)AFA,e* stock shows a large inversion with a break at the *ebony* locus. The *ebony* mutant of *Drosophila melanogaster* shows a number of behavioral abnormalities which lead to lowered mating success of males. The movement of these *ebony* flies is retarded and apparently uncoordinated as compared with normal animals[11]. Heterozygotes show superior mating success in competition with either homozygotes[3,4,12,13]. The deletion mapping was therefore performed by using three different deficiencies covering the 93D region in which *ebony* has been located[14,16]. *gust-M_1* expresses itself against those deficiencies (table 3). The gene for *gust-M_1* is located in the $93C_{3-6}$–$93D_{6-7}$ region of the third chromosome. The fact that even though *rucuca* and *TM3/Df(3R)e*^{N12} stocks have *ebony* phenotype they do not behave as chemosensorial mutants (tables 2 and 3) leads me to exclude that the gene for *gust-M_1* is located at the *ebony* locus.

Apparently a general increased 'hunger' or a reduced satiation response is a dominant component in the feeding behavior of *gust-M_1* flies. *gust-M_1* flies drink longer (and more) of the salt solutions applied directly to their proboscis than *Canton-S* flies (data not presented here). A similar behavior has been reported by Falk and Atidia[5] for *Lot-94* flies; these mutant flies, however, did not show a reduced sensitivity of the proboscis extension test, when salt solutions were applied to their tarsal bristles.

The third line of the part b of table 1 shows the mistactic



A drawing of a segment of the 93 region of the salivary gland polytene chromosomes of *Drosophila* showing the extent of the deficiencies discussed here. The numbering of the bands or chromomeres is that used in Bridges' revised map[15]. The shaded region indicates the location of *gust-M_1* as results by the deletion mapping analysis (table 3).

response of the *In(3R)AFA,e* stock to quinine salt; *In(3R)AFA,e* males are attracted to quinine sulfate in contrast to *Canton-S* males, which avoid quinine salt (see the third line of the part a of table 1). Siddiqi and Rodrigues[22] have analyzed the electrophysiological responses of taste sensillae to quinine sulfate in *Drosophila melanogaster;* unlike salt and sugar, quinine does not appear to excite individual neurons; 'the only effect quinine seems to have is to inhibit the firing of the other chemosensory neurons. Since the S cell is inhibited more strongly than the two L cells, the presence of quinine is likely to favor rejection by changing the ratio of S spikes to L spikes'[22]. *gust-M_1* responds mistactically to NaCl and quinine sulfate in contrast to *wild-type* males that avoid both compounds; this phenomenon can be accounted for assuming that *gust-M_1* could be a mutation perturbing functions in the central nervous system affecting the responses to both compounds. Alternatively, *gust-M_1* could be defective in a common part of the peripheral gustatory machinery that might bind both salt and quinine sulfate. The existence of *gus* mutations which alter the proboscis extension response is of practical significance, because mutations that are detectable in individual flies can be analyzed with genetic mosaics to locate anatomical foci of mutant behavior[9]. These mutants are useful because they provide us with a way of studying the organization of the chemoreceptor system which in *Drosophila* is sufficiently simple, for a correlated study of electrophysiological and behavioral responses of mutants to be likely to throw interesting light on the sensory code, whose exact nature so far remains unknown[7,18].

Acknowledgments. I thank Prof. F. Ritossa, Prof. G. Palumbo and Prof. G. Casadoro for criticism and helpful discussion and Prof. M. Chieppa for advice about statistical analysis.

1 D'Alessandro, A., Ritossa, F., and Scalenghe, F., Cytological localization of the '*ebony*' locus in *Drosophila melanogaster*. Drosoph. Inf. Serv. *52* (1977) 46.
2 Deak, I.I., Demonstration of sensory neurones in the ectopic cuticle of *spineless-aristapedia*, a homeotic mutant of *Drosophila*. Nature *260* (1976) 252–254.
3 Elens, A.A., Le rôle de l'hétérosis dans la compétition entre *ebony* et son allèle normal. Experientia *14* (1958) 274–275.
4 Elens, A.A., Studies of selective mating using the melanistic mutants of *Drosophila melanogaster*. Experientia *21* (1965) 145–146.
5 Falk, R., and Atidia, J., Mutation affecting taste perception in *Drosophila melanogaster*. Nature *254* (1975) 325–326.
6 Falk, R., Bleiser-Aviv, N., and Atidia, J., Labellar taste organs of *Drosophila melanogaster*. J. Morph. *150* (1976) 327–342.
7 Fujishiro, N., Kijima, H., and Morita, H., Impulse frequency and action potential amplitude in labellar chemosensory neurones of *Drosophila melanogaster*. J. Insect Physiol. *30* (1984) 317–325.
8 Hotta, Y., and Benzer, S., Abnormal electroretinograms in visual mutants of *Drosophila*. Nature *222* (1969) 354–356.
9 Hotta, Y., and Benzer, S., Genetic dissection of the *Drosophila* nervous system by means of mosaics. Proc. natn. Acad. Sci. USA *67* (1970) 1156–1163.
10 Isono, K., and Kikuchi, T., Autosomal recessive mutation in sugar response of *Drosophila*. Nature *248* (1974) 243–244.
11 Jacobs, M.E., Influence of light on mating of *Drosophila melanogaster*. Ecology *41* (1960) 182–188.
12 Jacobs, M.E., The influence of light on gene frequency changes in laboratory populations of *ebony* and not-*ebony Drosophila melanogaster*. Genetics *46* (1961) 1089–1095.
13 Jacobs, M.E., *Beta-alanine* and adaptation in *Drosophila*. J. Insect Physiol. *20* (1974) 859–866.
14 Korge, G., Report in Drosoph. Inf. Serv. *48* (1972) 20.
15 Lindsley, D.L., and Grell, E.H., Genetic variations of *Drosophila melanogaster*. Publ. Carnegie Inst. Wash. *627* (1968).
16 Mohler, J., and Pardue, M.L., Deletion-mapping of the 93D heat shock locus of *Drosophila melanogaster*. Chromosoma (Berl.) *86* (1982) 457–467.
17 Morea, M., Analysis of a chemosensory behavior mutant in *Drosophila melanogaster*. Atti Ass. genet. ital. *29* (1983) 183–184.
18 Perkel, D.H., and Bullock, T.H., Neural coding. Neurosci. Res. Progm. Bull. *6* (1968) 221–348.
19 Rodrigues, V., and Siddiqi, O., Genetic analysis of chemosensory pathway. Proc. Indian Acad. Sci. (B) *87* (1978) 147–160.
20 Rodrigues, V., and Siddiqi, O., A gustatory mutant of *Drosophila* defective in pyranose receptors. Molec gen. Genet. *181* (1981) 406–408.
21 Scalenghe, F., and Ritossa, F., Controllo dell'attività genica in *Drosophila:* Il puff al locus *ebony* e la *Glutamina sintetasi 1*. Atti Accad. naz. Lincei *13* (1976) 439–528.
22 Siddiqi, O., and Rodrigues, V., Genetic analysis of a complex chemoreceptor in Development and Neurobiology of *Drosophila*. Eds O. Siddiqi, P. Babu, L. Hall and J. Hall. Plenum, New York 1980.
23 Tanimura, T., Isono, K., and Kikuchi, T., Partial 'sweet taste blindness' and configurational requirements of stimulants in a *Drosophila* mutant. Jap. J. Genet. *53* (1978) 71–73.
24 Tompkins, L., and Sanders, T.G., Genetic analysis of chemosensory mutants of *Drosophila melanogaster*. Genetics *86* (1977) s 64.
25 Tompkins, L., Developmental analysis of two mutations affecting chemotactic behavior in *Drosophila melanogaster*. Devl Biol. *73* (1979) 174–177.
26 Tompkins, L., Cardosa, J., White, F., and Sanders, T.G., Isolation and analysis of chemosensory behavior mutants of *Drosophila melanogaster*. Proc. natn. Sci. USA *76* (1979) 884–887.

0014-4754/85/111381-04$1.50 + 0.20/0

Chemical evidence for interactions between vitamins E and C

P. Lambelet, F. Saucy and J. Löliger[1]

Nestlé, Research Department, Nestec Ltd., Avenue Nestlé 55, CH-1800 Vevey (Switzerland), 11 February 1985

Summary. Experimental proof is provided for interactions between radicals of vitamin E/vitamin C as generated by air-oxidized lipids (liquid fraction of subcutaneous chicken fat). Using ESR spectroscopy, hydrogen atom exchange is shown to take place between vitamin C and the radical of vitamin E. Sequential consumption of these two vitamins in oxidized lipid, first vitamin C then vitamin E, is demonstrated by means of differential pulse polarography. These results elucidate the in vitro radical scavenging functions attributed to vitamin E and vitamin C as well as their synergism in lipid antioxidation.
Key words. ESR; vitamin E; vitamin C; antioxidation; radical; exchange; mechanism.