positively correlated (fig. 2). However, no such correlation was found between the magnitudes of the inversions going from suppression to emission. This behavior of neuron A was not unique, because across all 11 RN neurons studied, and a total of 44 paired BP/SWS episodes which encompassed the distribution of 72,400 and 197,021 hexagram patterns respectively, the correlation between the emission magnitudes and subsequent suppressions below chance level was significant ($r_w = +0.65$; $p \cong 0.02$; weighted correlation coefficient obtained by Fisher's 'z' transformation method of individual r values[23]). However, the magnitudes of reversals going from suppressions to emissions showed no correlation ($r_w = -0.21$; $p > 0.8$).

Discussion. Receptor desensitization is known to be affected by agonist concentration, exposure time, and metabolic alterations, and is therefore a graded process[16,17]. Hence, it offers a plausible explanation for the correlation between the emission magnitudes or patterns and their subsequent deficits. In addition to the RN neurons, comparable inversions of pattern emissions have also been observed in the feline nucleus centrum medianum[24,25]. Such a behavior of single neurons has implications for the function of SWS and its nature, which still remain elusive despite the evidence that SWS is indispensable for normal behavior[1,2] and survival[26]. Hence, the question arises of whether or not there is a good reason to suspect that unabated but inversely distributed patterns of neuronal firing may be linked to a recovery process of SWS. In vitro studies showed that, upon simple removal of a receptor agonist, the resensitization of brain β-adrenergic receptors is a slow process. However, a brief potassium-induced depolarization promptly resensitizes the receptors[17]. Similar results have been reported for cholinergic receptors. This indicates that barrages of depolarizing impulses at soma-dendritic sites, presumably adjacent to those that had been desensitized, may indeed have a recuperative effect during SWS.

1 Feinberg, I., and Evarts, E.V., Biol. Psychiat. *1* (1969) 331.
2 Moruzzi, G., Ergebn. Physiol. *64* (1972) 1.
3 Marczynski, T.J., Burns, L.L., and Marczynski, G.T., Brain Res. *185* (1980) 139.
4 Steriade, M., and Hobson, J.A., Progr. Neurobiol. *6* (1976) 155.
5 Schlag, J., and Waszak, M., Brain Res. *21* (1970) 286.
6 Skinner, J.E., in: Multidisciplinary Perspectives in Event-Related Brain Potential Research, p.616. Ed. D. Otto. U.S. Government Printing Office, Washington, D.C., 1976.
7 Jones, E.G., J. comp. Neurol. *162* (1975) 295.
8 Scheibel, M.E., and Scheibel, A.B., Exp. Neurol. *34* (1972) 316.
9 Filion, M., Lamarre, Y., and Cordeau, J.P., Exp. Brain Res. *12* (1971) 499.
10 Frigyesi, T.L., Brain Res. *48* (1972) 157.
11 Purpura, D.P., in: The Neurosciences, 2nd Study Program, p.372. Ed. F.O. Schmitt. Rockefeller University Press, New York 1970.
12 Hoffert, M.J., Miletic, V., Ruda, M.A., and Dubner, R., Soc. Neurosci. Abstr. *8* (1982) 805.
13 Miletic, V., Hoffert, M.J., Ruda, M.A., Shigenaga, Y., and Dubner, R., Soc. Neurosci. Abstr. *8* (1982) 805.
14 Gilbert, P.F.C., and Thach, W.T., Brain Res. *128* (1977) 309.
15 Calvin, W., and Graubard, K., in: The Neurosciences, 4th Study Program, p.513. Eds. F.O. Schmitt and F.G. Worden. The MIT Press, Cambridge, MA, 1979.
16 Dunwiddie, T., Neurosci. Res. Prog. Bull. *18* (1980) 411.
17 Wagner, H.R., and Davis, J.N., Proc. natl Acad. Sci. USA, *76* (1979) 2057.
18 Kafka, M.S., Wirz-Justice, A., Naber, D., and Wehr, T.A., Neuropharmacology *20* (1981) 421.
19 Brudno, S., and Marczynski, T.J., Brain Res. *125* (1977) 65.
20 Marczynski, T.J., Wei, J.Y., Burns, L.L., Choi, S.Y., Chen, E., and Marczynski, G.T., Brain Res. Bull. *8* (1982) 565.
21 Jasper, H., and Ajmone-Marsan, C., A Stereotaxic Atlas of the Diencephalon of the Cat. National Research Council of Canada, Ottawa 1954.
22 Armitage, P., Statistical Methods in Medical Research, p.373. J. Wiley and Sons, New York 1971.
23 Zar, J.H., Biostatistical Analysis, p.620. Prentice-Hall, Inc., Englewood Cliffs, N.J. 1974.
24 Burns, L.L., and Marczynski, T.J., Soc. Neurosci. Abstr. *5* (1979) 606.
25 Marczynski, T.J., and Burns, L.L., Soc. Neurosci. Abstr. *5* (1979) 496.
26 Rechtschaffen, A., Gilliland, M.A., and Bergmann, B.M., 22nd Annual Meeting, Ass. Psychophysiol. Study Sleep, San Antonio, Texas, June 16–20, 1982.
27 Scheibel, M.E., and Scheibel, A.B., in: The Neurosciences, p.577. Eds G.C. Quarton, T. Melnechuk and F.O. Schmitt. The Rockefeller University Press, New York 1967.

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Accumulation of taurine in the nasal mucosa and the olfactory bulb

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Summary. Using whole-body autoradiography of ^{14}C-taurine in mice we have observed a high concentration in the nasal mucosa followed by accumulation in the olfactory bulb at longer survival times. When ^{14}C-taurine was administered in the nasal cavity unilaterally, a high accumulation was observed in the ipsilateral olfactory bulb.

The sulfonic amino acid taurine is known to be involved in the conjugation of bile acids[2,3], and has also been suggested to act as an inhibitory neurotransmitter or a neuromodulator in the eye and in the brain[4–6]. High uptake of taurine has been observed in the retina and in the CNS especially in the olfactory bulb[7].

In our laboratories, the distribution of ^{14}C-taurine in mice was studied using whole-body autoradiography. ^{14}C-taurine, uniformly labeled, with a spec.act. of 113 mCi/mmole was obtained from the Radiochemical Centre, Amersham, England. The radiochemical purity was 99%. Mice of the C57BL strain, of both sexes, were used. Nonpregnant mice weighed about 20 g and the pregnant mice 28–35 g. The day of conception (day 0) was determined by the presence

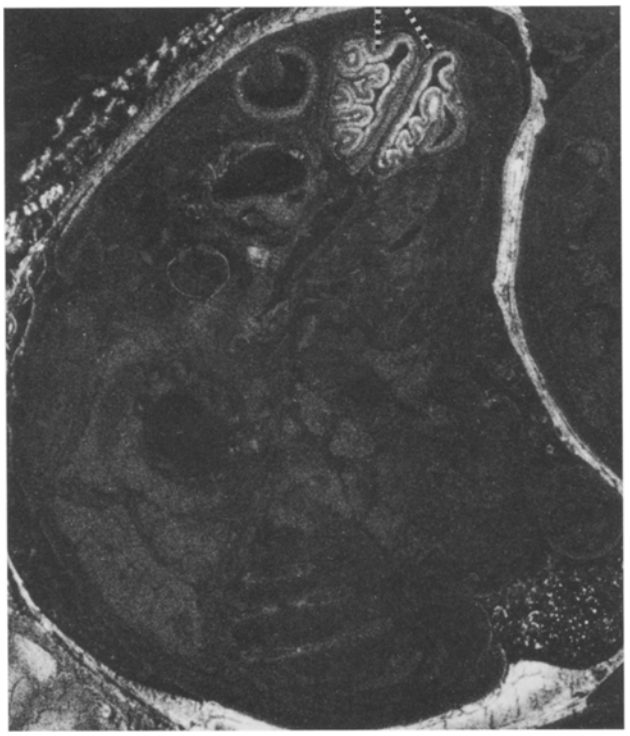

Figure 1. Detail of autoradiogram of a pregnant mouse (18th day of gestation) 1 h after an i.v. injection of ^{14}C-taurine. There is high activity in the foetal nasal mucosa.

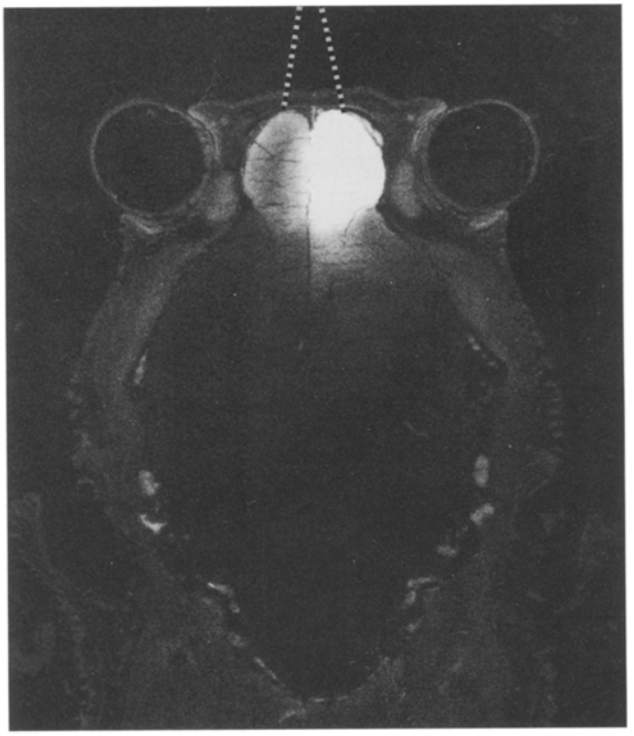

Figure 2. Detail of an autoradiogram of a mouse 24 h after administration of ^{14}C-taurine in the nasal cavity unilaterally. High concentration is observed in the ipsilateral olfactory bulb.

of a vaginal plug. Male and nonpregnant female mice were injected i.v. with 5 µCi (corresponding to 6 µg) of ^{14}C-taurine dissolved in physiological saline. The animals were killed after 1 h, 4 h, 24 h, 4 days and 15 days. Pregnant mice were each given an i.v. dose of 7.5 µCi C-taurine and were sacrificed after 20 min, 1 h, 4 h and 24 h. All pregnant animals were killed at the 18th day of gestation. Whole-body autoradiography was performed according to Ullberg[8,9]. The sections were exposed against X-ray films (Industrex C, Kodak) for 1 to 4 weeks.

A high concentration was observed in the nasal mucosa from 20 min to 4 days after the injection in the adult mice and at all studied survival times (20 min to 24 h) in the mouse foetuses (fig. 1). The level in the adult brain was rather low, except for in the olfactory bulb, where a high accumulation was observed from 24 h to 4 days after the administration. Initially, the level in the foetal brain was low, but exceeded that of the maternal brain at 24 h and 4 days after the injection. The concentration in the foetal olfactory bulb was higher than in other brain areas at 24 h after the administration.

In another series of mice 4 µCi of ^{14}C-labeled taurine, dissolved in 20 µl physiological saline was administered in the nasal cavity unilaterally. In order to facilitate the administration procedure, the animals were anesthetized with ether. The mice were killed after 24 h, 4 and 8 days. Horizontal sections were taken on tape and were exposed against X-ray films 1–2 weeks. High accumulation was observed in the ipsilateral olfactory bulb 24 h after the administration (fig. 2). After 4 days there was a high concentration in both olfactory bulbs. Retention was observed in the brain 8 days after the administration (the level in the olfactory bulbs no longer exceeding that of other brain areas).

Taurine has earlier been observed to be transported in the optic nerves of goldfishes, rabbits and rats[10–14]. Other amino acids such as γ-aminobutyric acid and cysteine have been reported not to migrate in the goldfish visual system[15]. Margolis and Grillo[16] have demonstrated that β-alanine is incorporated specifically into the dipeptide carnosine (β-alanyl-L-histidine), which has been suggested to be a neurotransmitter or neuromodulator in the olfactory system. In autoradiographic studies after intranasal administration of β(^{3}H) alanine in hamsters, Burd et al.[17] observed that carnosine is transported in the axons of the olfactory neurons from the nasal mucosa to the olfactory bulb.

The results of the present investigation indicate that taurine is taken up by the nasal mucosa and transported to the olfactory bulb. Further studies are needed to establish the mechanisms involved in the accumulation in the nasal mucosa and the transport in the olfactory pathway. One possibility may be that taurine is incorporated into a peptide, which may, as carnosine, be a neurotransmitter or neuromodulator in the olfactory system.

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2 Strecker, A., Annls Chim. Phys. *70* (1849) 149.
3 Danielsson, H., Adv. Lipid Res. *1* (1963) 335.
4 Mandel, P., Pasantes-Morales, H., and Urban, P.F., in: Transmitters in the visual process, p. 89. Ed. S.L. Bonting. Pergamon Press, New York 1976.
5 Oja, S.S., and Kontro, P., in: Taurine and neurological disorders, p. 181. Eds A. Barbeau and R.J. Huxtable. Raven Press, New York 1978.
6 Mandel, P., and Pasantes-Morales, H., Rev. Neurosci. *3* (1978) 157.

7 Sturman, J.A., Rassin, D.K., Hayes, K.C., and Gaull, G.E., J. Nutr. *108* (1978) 1462.
8 Ullberg, S., Acta radiol., suppl. *118* (1954) 1.
9 Ullberg, S., Sci. Tools (The LKB Instrument J.) pp.2-29 (1977).
10 Ingoglia, N.A., Sturman, J.A., Lindquist, T.D., and Gaull, G.E., Brain Res. *115* (1976) 535.
11 Ingoglia, N.A., Sturman, J.A., Rassin, D.K., and Lindquist, T.D., J. Neurochem. *31* (1978) 161.
12 Sturman, J.A., J. Neurobiol. *10* (1979) 221.
13 Politis, M.J., and Ingoglia, N.A., Brain Res. *166* (1979) 221.
14 Sturman, J.A., in: The effects of taurine on excitable tissues, p.187. Eds S.W. Schaffer, S.I. Baskin and J.J. Kocsis. MTP Press Ltd, Lancaster 1981.
15 Ingoglia, N.A., Sturman, J.A., Lindquist, T.D., and Gaull, G.E., Brain Res. *115* (1976) 535.
16 Margolis, F.L., and Grillo, M., Neurochem. Res. *2* (1977) 507.
17 Burd, G.D., Davis, B.J., Macrides, F., Grillo, M., and Margolis, F.L., J. Neurosci. *2* (1982) 244.

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Cyanidin-3-β-glucoside, a newly recognized basis for resistance in cotton to the tobacco budworm *Heliothis virescens* (Fab.) (lepidoptera: Noctuidae)[1]

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Summary. Cyanidin-3-β-glucoside was shown to be an important factor of resistance in cotton *Gossypium hirsutum* L. leaves to the tobacco budworm *Heliothis virescens* (Fab.). This provides a potential basis for achieving insect resistance in non-glanded cotton and other crops infested by *Heliothis.*

We report the identification of cyanidin-3-β-glucoside (chrysanthemin) as a major factor of resistance in cotton, *Gossypium hirsutum* L., to the tobacco budworm *Heliothis virescens* (Fab.) and we reaffirm the reported effectiveness of gossypol[2-4]. We also present data to show that the correlation between condensed tannins (proanthocyanidins) in terminal leaves and growth of larvae feeding on terminal leaves in the field was small and positive. Paradoxically, these 3 compounds when incorporated in diets are equally toxic for larvae. Two important implications of these findings are that a basis (anthocyanin content) is provided for achieving insect resistance in non-glanded low gossypol cotton, and potentially for selecting for resistance in crops, world-wide, to various *Heliothis.* The larvae of 3 additional *Heliothis* spp. are also important pests of cotton, tobacco, corn, and other food crops[5].

Anthocyanins were first found in cotton in the envelope of pigment glands by Stanford and Viehoever in 1918[6]. We identified the red pigment in the cotton flower as chrysanthemin in 1967[7]. Recently, Chan and Waiss[8] confirmed the presence of an anthocyanin in the pigment glands and identified it as the same pigment in cotton flowers, chrysanthemin. Gossypol occurs in association with a number of gossypol-related triterpenoids, sesquiterpenoid quinones, hemigossypols, and heliocides, all possessing comparable insect toxicity[9,10]. Condensed cotton tannin[11-13] and flavo-

Table 1. Inhibition of tobacco budworm larval growth by cotton constituents, ED_{50} as percent of diet

Constituent	ED_{50}, %[a]		
	Chan et al.	Stipanovic	Miss. State
Gossypol	0.12	0.05	0.113
Hemigossypolone	0.03	0.29	-
Heliocide H_1	0.12	0.10	-
Heliocide H_2	0.13	0.46	-
Methyl sterculate	0.41	-	N.T.
(+)-Catechin	0.13	-	0.052
Condensed tannin	0.15	-	0.063
Quercetin	0.05	-	0.042
Isoquercitrin	0.10	-	0.060
Cyanidin	-	-	0.166
Delphinidin	-	-	0.138
Chrysanthemin	-	-	0.070

[a]Percent of compound required to reduce weight gain by 50%.

Table 2. Relative effects of cotton flower petal allelochemics on tobacco budworm growth and survival

Cultivar	Petal color	Tannins (%)[a]	Gossypol (%)[a]	Chrysanthemin (%)[a]	Larval wt (mg)[d]	Insects surviving (%)
ST-7AGN (NG)[b]	W[c]	5.79	0.10	0.07	4.72[a]	39.5
	R	8.68	0.11	0.67	0.46	7.0
DH 66 (NG)	W	5.40	0.17	0.07	4.18[a]	31.0
	R	8.55	0.13	0.73	0.56	16.5
ST-213 (G)	W	3.49	0.52	0.13	1.52[b]	28.0
	R	8.75	0.79	0.59	0.41	8.5
DH 126 (G)	W	3.16	1.72	0.18	[e]	-
	R	6.25	2.46	0.65	[e]	-

[a]% of dry weight. [b]NG, nonglanded; G, glanded. [c]W, white: 1st day flower color; R, red; 2nd day flower color. Means of larvae fed on white petals not significantly different at 0.05 level if followed by same letter. [d]Average tobacco budworm weight after feeding 5 days on petals. [e]Not fed.