affects the appearance and stability of the photoreceptors therefore leading to a row pattern. It was precisely this observation (described in my paper[2]), which led to the discovery of the change of the square mosaic in the light to a row mosaic in the dark. My publication, therefore, carried the implicit advice to investigators who isolate retinae to obtain absorbance spectra by microspectrophotometry, not to use these types of preparations for the establishment of cone mosaics[6].

Because his results on a cichlid did not match mine on the guppy, Fernald[1] doubts that there are species-specific differences between fish. We have since shown that in the weever fish, *Trachinus vipera,* the square mosaic does not change in the dark (tested on retinal mounts, histological and electron microscopical sections of whole eye-cups)[7]. Although it is difficult to judge an unlabeled 3 µm section of another author (Fernald's fig. 2, b), it would seem that the structural reason for the persistence of the square mosaic in the dark in *Haplochromis burtoni* was the same as described by me for *Trachinus vipera.*

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0014-4754/83/091049-02$1.50 + 0.20/0

The effect of l-DOPA and benserazide on the amount of dopamine in the corpus striatum of X-irradiated rats

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Summary. The effect of a mixture of l-DOPA and benserazide on the amount of dopamine in the corpus striatum of rats irradiated with 850 R of X-rays was investigated. The amount of dopamine in the corpus striatum was measured fluorimetrically at various times after irradiation. It was found that i.p. administration of l-DOPA and benserazide 1,2 and 5 days after irradiation produces significant replenishment of dopamine in the striatum, thus indicating that precursor uptake and metabolism, and probably amine storage, remained unaltered after irradiation.

Evidence was presented earlier that catecholamine stores in the heart atria, hypothalamus and corpus striatum of the rat were significantly depleted after whole-body X-irradiation[2-5]. The content of adrenaline and noradrenaline in the adrenal medulla and salivary gland is also reduced after irradiation[6-8]. On the other hand, immobilization stress has been known to produce a significant increase of plasma concentration of catecholamines and marked elevation of urinary catecholamines[9-11]. It has been known that application of tyrosine or l-DOPA produces a marked increase of catecholamines in the brain of rats pretreated with the peripheral inhibitors of DOPA-decarboxylase[12-15]. We have previously found that the administration of a mixture of l-DOPA and benserazide significantly increased the content of catecholamines in the heart atria and brain of irradiated rats 24 h after irradiation[5]. It seemed interesting to us to examine whether l-DOPA undergoes metabolism in rat brain for longer periods of time following exposure to lethal doses of X-rays. In the present experiment, an attempt has been made to investigate the effect of l-DOPA on the amount of dopamine in the corpus striatum of irradiated animals after inhibition of peripheral DOPA-decarboxylase by benserazide, as measured on day 1,2 and 5 following irradiation.

Materials and methods. Male albino rats of the Wistar strain bred under standard conditions and weighing 200-210 g, were used. The animals were whole-body X-irradiated with 850 R. Irradiation parameters were: 200 kV; 16 mA; 0.5 mm Cu; D-42 cm. The dose rate was 112 R/min. Nonirradiated animals of similar body weights served as control. The animals were sacrificed 1,2 or 5 days after irradiation. 3 h and 1 h before sacrificing, the animals were treated as follows: a number of irradiated and control rats were each time injected i.p. with 125 mg/kg b.wt of a 4:1 mixture of l-DOPA and benserazide hydrochloride (Madopar®, Hoffman-La Roche), suspended in 5 ml of 1% methylcellulose, whereas at the same time a separate group of control rats received i.p. 100 mg/kg b.wt of l-DOPA only, dissolved in 4 ml of destilled water. Immediately after sacrifice, the corpora striata were dissected and homogenized in the cold. The striata from 2 rats were pooled. Catecholamines were extracted and their content estimated according to the method of Manuhin et al.[16], based on the methods of Carlsson and Waldeck[17], and Laverty and Taylor[18] for extraction and quantitative estimation of dopamine. Recovery of dopamine was 85-90% throughout the experiment. Fluorimetric estimation was done using Aminco-Bowman spectrophotofluorimeter. Results were expressed

The effect of a mixture of l-DOPA (100 mg/kg) and benserazide (25 mg/kg) on the amount of dopamine in the corpus striatum of rats irradiated with 850 R (mean ± SE µg/g of fresh tissue), 1, 2 and 5 days after irradiation. The number of experiments is indicated in parenthesis

Treatment	1 day	2 days	5 days
1. Controls	9.08 ± 0.251 (12)	9.11 ± 0.228 (13)	9.01 ± 0.258 (12)
2. Irradiated animals	6.52 ± 0.229 (16)	6.21 ± 0.389 (13)	6.71 ± 0.242 (9)
3. Irradiated animals treated with l-DOPA plus benserazide	17.85 ± 1.47 (16)	17.77 ± 0.342 (16)	19.95 ± 0.717 (9)
4. Control animals treated with l-DOPA plus benserazide	30.07 ± 1.24 (14)	29.80 ± 1.73 (12)	30.10 ± 1.15 (12)
5. Controls treated with l-DOPA	11.13 ± 0.485 (8)	11.50 ± 0.533 (10)	10.87 ± 0.650 (8)

as mean ± SE of the mean and statistical significance was calculated by Student's t-test.

Results and discussion. The results of the present experiments are summarized in the table. It can be seen that the amount of dopamine in the corpus striatum of rats irradiated with 850 R was significantly decreased on day 1,2 and 5 following irradiation, as compared with the control values ($p < 0.001$). On the other hand, peripheral application of l-DOPA and benserazide produced a significant increase in the amount of dopamine in the corpus striatum of irradiated rats regardless of the post-irradiation period investigated ($p < 0.001$). Similarly, pretreatment of nonirradiated control animals with a mixture of l-DOPA and benserazide resulted 1,2 and 5 days later in a remarkable rise of dopamine in the corpus striatum ($p < 0.001$). When nonirradiated controls were treated with 100 mg/kg of l-DOPA only, the dopamine content in the corpus striatum was also higher in all the 3 investigated points than in the nontreated controls ($p < 0.05$). As expected, a significant difference was found between l-DOPA-treated and l-DOPA plus benserazide treated nonirradiated controls irrespective of the post-irradiation period studied ($p < 0.001$). The dopamine content was twice as large in the corpus striatum of the latter, due to the protective action of benserazide on l-DOPA by the inhibition of DOPA-decarboxylase.

Aromatic l-amino acid decarboxylase can be selectively inhibited in the cerebral and extracerebral tissues by a variety of drugs. For instance, pretreatment with N-(DL-seryl)-N′-(2,3,4-trihydrohybenzil) hydrazine (Ro-4-4602), DL-αmethyl-αhydrazino-3,4-dihydrophenylpropionic acid (HMD) or NSD 1015 (m-hydroxybenzyl-hydrazine) markedly enhances the DOPA-induced increase of catecholamines in the brain, and to a lasser extent in the liver, heart and kidney[12–15]. The administration of l-DOPA after inhibition of peripheral DOPA-decarboxylase is thought to induce an increase in brain catecholamines as a consequence of increased penetration of this amino acid into the brain where decarboxylation is taking place.

The above finding that the i.p. injection of l-DOPA and benserazide significantly increase the content of dopamine in the corpus striatum of rats irradiated with 850 R, as measured 1, 2 and 5 days after irradiation, suggests that these animals are still able to metabolize the catecholamine precursor after peripheral inhibition of l-DOPA-decarboxylase. We have previously shown that, in rats irradiated with 650 or 850 R, the application of l-DOPA and benserazide produced a significant increase in the amount of noradrenaline or dopamine in the heart atria and hypothalamus, as measured 24 h after irradiation, compared to irradiated controls[5].

The results obtained suggest that precursor uptake and metabolism, and probably amine storage, remained unaltered after irradiation.

1 Acknowledgment. We wish to acknowledge the expert technical assistance of Gordana Milenković.

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0014-4754/83/091050-02$1.50 + 0.20/0

Glyceryl-1,2-dioleate-3-palmitate, a brood pheromone of the honey bee (*Apis mellifera* L.)

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Summary. Glyceryl-1,2-dioleate-3-palmitate, a brood pheromone, was isolated and identified from drone pupae of *Apis mellifera* L. Broodcare is an essential element in the life of the honey bee, but it is still unknown how bees recognize their brood, some aspects of this question are examined in this paper. Glyceryl-1,2-dioleate-3-palmitate, a brood pheromone, was isolated and identified from drone pupae of *Apis mellifera* L.

Material and methods. When 2 samples of brood are offfered to a group of hive bees, they choose, settle on and warm only one. Experiments have shown that mechanical and chemical stimuli (demonstrated with ether extracts of worker, queen or drone pupae) are essential for the induction of incubation of brood[1].

A bioassay was used to isolate and identify the biologically active compounds: A group of 400–800 hive bees was confined to a cage (21 × 12 × 6 cm). The cover of the cage had 3 openings. The center hole contained a honey feeder and the side holes – 6 cm distant from the central one – were closed by semi-artificial queen cells. They were com-