sperm-egg-mixture, containing $0.6–1.2 \times 10^6$ sperm/ml, was incubated for 4.5 h and then fixed with 2 ml Türk's solution at 5 °C over night. The ova were then stained with 0.5% lacmoid in 40% acetic acid, mounted on slides and examined for presence of enlarged sperm head(s) or pronucleus(ei), indicating fertilization. The significance of the difference in fertilization frequency between the control and the treated eggs was estimated with the t-test and the Mann-Whitney U-test.

Results and discussion. It appears from the table that at 5×10^{-9} M 17β-estradiol fertilization is stimulated, an effect not shown by 17α-estradiol at the same level. However, no effect on fertilization frequency was exerted by 17β-estradiol at concentrations of 5×10^{-6}, 5×10^{-7}, 5×10^{-8} and 5×10^{-10} M.

Despite efforts to eliminate sources of systematical error the number of ova was always greater in the test pool than in the control pool. This could possibly be explained by an unintentional inclination to choose the larger of the 2 pools of eggs to be used as the test pool.

Earlier it has been reported[5,6] that 10^{-5} M 17β-estradiol inhibits fertilization. The same authors found, too, that as the concentration was lowered to 10^{-6} M the inhibition was abolished, a finding which is in conformity with the present results.

Since stimulation of fertilization appears at 5×10^{-9} M 17β-estradiol it may be assumed that at the higher concentrations tested, the stimulating and the inhibiting effects balance each other to the degree that no significant change in fertilization frequency can be observed against the background of variation in the present data.

As the concentration of added steroid is lowered the relative contribution from the naturally-occurring estrogens increases. An estimate of the latter is of necessity uncertain, since several factors, e.g. rate of synthesis may influence the result. It can, however, be said that if endogenous estrogens are necessary for fertilization they should have a concentration not very different from that at which exogenous 17β-estradiol stimulates fertilization.

The stimulation is probably mediated by a structure-recognizing mechanism, since 5×10^{-9} M 17α-estradiol fails to influence the frequency of fertilization. Furthermore, binding sites for which 17β- but not 17α-estradiol compete have been demonstrated by Briggs[7] and these binding sites appear to be located near to the plasma membrane[8]. On the basis of results reported by Szego[9], Lindahl[10] suggested that HHA induced by 17β-estradiol involves activation of adenylate cyclase. Indeed, Cheng and Boettcher[11] recently reported that 17β-estradiol stimulates spermatozoal adenylate cyclase, an enzyme which is present immediately inside the sperm head plasma membrane[12]. The ability of cAMP and catecholamines to induce HHA also suggest the involvement of adenylate cyclase; besides, the effect of the latter is inhibited by adrenergic receptor blockers[13]. The catecholamines are, moreover, capable of inducing the acrosome reaction[14] and, as pointed out by Lindahl and Sjöblom[2], it is likely that HHA and the acrosome reaction have reaction links in common. Thus, it is possible that the increase in fertilization rate caused by 17β-estradiol depends on the stimulation of certain steps in the acrosome reaction.

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Diurnal changes of cone mosaic in a teleost retina

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Summary. In *Poecilia reticulata*, a surface-living fish, the square mosaic changes into a row mosaic in the dark.

Fernald[1] reports that he did not find any mosaic pattern changes in the retina of *Haplochromis burtoni* during dark adaptation. He, therefore, questions my results on changes from square to row mosaic observed in the guppy *Poecilia reticulata*[2]. Fernald suggests that my findings were due to our method of preparing the tissue, i.e. the separation of retina from pigment epithelium. I would like to draw his attention to the 'Material and methods' section in our paper, where we refer to a previous publication[3] which describes in great detail the preparation of the specimens from which our electronmicrographs (figs 2, A and B) were obtained. A reading of the above-mentioned publication would have made it clear that our electronmicrographs of light and dark adapted photoreceptors were obtained from intact hemisected eye-cups, fixed immediately after sacrifice of the fish, and that neither Ca-free Ringer nor removal of pigment epithelium was involved. Moreover, I would like to stress that the figures shown in my publication[2] were taken at the level of the inner segment (ellipsoid), and not outer segment, as Fernald states. We have since shown that the rotation of the inner segments of twin cones during retinomotor activity develops already in the guppy embryo and is established in the neonate. Therefore, at the onset of vision the change from square to row pattern in the dark is already present as an intrinsic property of the retina[4,5].

Fernald is correct when he states that the preparation of isolating the retina in Ca-free Ringer solution greatly

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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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)