recover on the following days. The experiment was terminated on the 27th day.

Discussion. The mechanism of post-irradiation saccharin avoidance behavior is virtually unknown to investigators in this field. The question whether the effect is a result of a patho-physiological change or a chemical change in the taste perception is still unanswered.

While the problem of post-irradiation avoidance in mice are still unresolved pathologically, physiologically or otherwise, our results have shown that the irradiation of the solutions (saccharin and sucrose) did induce the same phenomenon of avoidance as if the animals themselves were irradiated. Many recent works have confirmed earlier results performed at the beginning of this century that hydrogen peroxide is indeed a product of irradiated water and other aqueous solutions. FREY and POLLARD[9] recently reported $3.5 \cdot 10^{-6}$ gm/ml of hydrogen peroxide produced in glucose solution irradiated with $2.7 \cdot 10^4$ R and that this irradiated medium cause a cessation of cell growth. The same effect can be obtained with reagent hydrogen peroxide. MOLIN and EHRENBERG[10] suggested that hydrogen peroxide or other peroxides produced in irradiated glucose may be responsible for the bactericidal effect. Earlier, HANNAN and SHEFFHERD[11], indicated that 'hydrogen peroxide produced from water by irradiation of a medium, and remaining thereafter the end of the irradiation, may be responsible for some of the effects seen in the systems studied'.

Although we have not determined quantitatively the amount of hydrogen peroxide produced in either irradiated saccharin, sucrose or water, we are positively certain that this reagent is produced when the liquids are exposed to low radiation dosages. We were able to verify that hydrogen peroxide was indeed a product of irradiated saccharin solution, sucrose solution and water by using the techniques of BRANDT and KESTON[12] and diacetyl 2′,7′-diachlorofluorescin from Eastman Kodak Company. The animals avoiding both the sweet solutions and plain tap water when these liquids were irradiated raises the question whether the presence of saccharin and sucrose is really necessary or only amplifying this observed avoidance behavior[13].

Zusammenfassung. Es wird gezeigt, dass röntgenbestrahlte Mäuse Saccharin- und Sucrose-Lösungen gleicherweise vermeiden wie Lösungen, denen minimale Mengen von H_2O_2 zugesetzt wurden. Es wird angenommen, dass der Effekt auf der radiolytischen Produktion von H_2O_2 in der Lösung zurückzuführen ist.

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A Quantitative Histochemical Study of Succinic Dehydrogenase in the Kidney and Liver of Rats Subjected to Nephrectomy and Injected with Egg-White

The formation of large droplets in the cells of the proximal convolutions has been interpreted as a mechanism whereby either the excessive absorbed protein is metabolized by the mitochondrial enzymes or the protein is segregated by the lysosomes or phagosomes in the cell[1,2]. In this investigation, succinic dehydrogenase was studied to ascertain whether or not it plays an active part in this process. Rat kidneys in different functional states were used, and the liver also was examined.

Methods. Young adult male rats were treated as follows: (1) a single i.p. injection of 25 ml of strained solution of hens' egg-white in saline 24 h prior to sacrifice (2) right nephrectomy 7 days before sacrifice, and (3) a combination of the foregoing treatments. A single experiment involved 2 experimental and 1 each of untreated and sham-treated controls (saline injection or sham operation). At the end of the experiments the rats were weighed and decapitated, and the left kidney and adrenal gland were weighed individually. From the cortex of the kidney and from the liver, tissue 'punch-outs' (5 mm in diameter) were made[3] and promptly frozen in a mixture of dry ice and acetone. In a cryostat, each frozen 'punch-out' was used to obtain duplicate pairs of adjacent sections cut at 5 μ. One member of each pair was used for the spectrophotometric determination of succinic dehydrogenase activity[4], in which the hydrogen acceptor was 2-(*p*-iodophenyl)-3-(*p*-nitrophenyl)-5-phenyl tetrazolium chloride (INT). The other member of the pair was used for the spectrophotometric determination of protein nitrogen content[5], in which the reading was converted into the amount of nitrogen from a standard curve obtained from micro-Kjeldahl analyses. The final expression of enzyme activity was made by these data. In the outer part of the cortex of the kidney, the only portion used, the predominant epithelial structure positive to the enzyme reaction was the proximal convoluted tubules[6]. In the Table, any differences between the data of the untreated and the sham-treated controls were not statistically significant. Accordingly, in applying the *t*-test to the experimental results, the data for these 2 types of controls were averaged and used as a single control.

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Organ weights and the succinic dehydrogenase activities in kidneys and livers of rats (mean ± standard deviation)

Treatment	No. of rats	Kidney (g) / Body wt. (g) · 10^4	Adrenal (g) / Body wt. (g) · 10^6	Enzyme activity[b] Kidney	Liver
Untreated control	17	37 ± 3.9	44 ± 8.0	147 ± 16	132 ± 11
Sham control	17	38 ± 3.5	51 ± 6.3	145 ± 14	134 ± 12
Egg-white injection	11	38 ± 4.1	53 ± 12.0	159 ± 28	223 ± 21[a]
Right nephrectomy	12	47 ± 4.7[a]	53 ± 3.5	126 ± 11[a]	143 ± 15
Combined treatment	12	49 ± 5.3[a]	66 ± 18.0	132 ± 13[a]	173 ± 37

[a] P of 0.05 or less, [b] mμg formazan/μg protein nitrogen/5 min.

Results and discussion. The left kidneys of rats subjected to right nephrectomy, with or without egg-white injections, were significantly heavier than those of the controls, whereas injection alone did not alter the weight. No significant change in the adrenal weight was observed in any group. The enzyme activity in the kidney of injected rats showed a slight increase but with no significance (P of 0.8). In contrast, the activity in the remaining kidneys of unilaterally nephrectomized rats was significantly less than in the controls, and a similar reduction was noted also in the rats of the combined treatment. The difference between these 2 diminutions, however, was not significant (P of 0.4). The enzyme activity in the livers of injected rats was significantly greater than in the controls.

The reduced enzyme activity in the remaining kidneys of the operated rats is probably related to the phenomenon of compensatory hypertrophy itself. Although both mitoses and anabolism in the renal cells of compensatory hypertrophy are above normal[7,8], the renal clearance is diminished[9]. The enzymatic functions of both regenerating and functionally impaired kidney cells are subnormal[10,11]. The lack of enzymatic change from the original level in the kidneys of both groups of injected rats does not support the view that the droplet formation involves mitochondrial enzymes[1,12]. The work of other enzymes likewise tends to cast doubt on the direct relationship[13,14]. The hepatic parenchymal cells do not develop droplets in response to protein injection[15], and the present data do not point to any meaningful relation between the enzymes of kidney and liver.

Résumé. L'activité de la déshydrogénase succinique du rein ou du foie ne peut expliquer la formation de gouttelettes dans les cellules rénales après injection i.p. de blanc d'œuf associée ou non à une néphrectomie.

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Experiments on Mitochondrial Swelling in vivo

Much work has been devoted to the study of active swelling and contraction of isolated mitochondria in vitro and a number of chemical swelling agents have been described; however, the intracellular significance of these mitochondrial changes remains to a large extent a matter of conjecture.

Mitochondria have been known to be capable of undergoing changes of movement, shape and volume, also in the living cells since the early investigations on tissue cultures[1], and more recent observations, with the aid of the phase-contrast microscope and cinemicrography, have shown that mitochondria can be induced to swell in vivo by addition to the tissue culture medium of a variety of chemical agents such as 2,4-dinitrophenol (DNP), adenosine triphosphate (ATP), ethylenediaminetetraacetate (EDTA), adrenochrome, as well as by oxygen deprivation and by illumination with intense light of 556 nm wave-length[2]. Also fluoroacetate[3] and diphtheria toxin[4,5] have been shown to induce swelling of mitochondria in vivo. These findings showed that mitochondria may undergo reversible swelling in the living cells; however, the available data indicate that marked differences must exist between the in vitro and the in vivo processes. In fact the mitochondrial swelling agents in vivo include compounds which prevent, like DNP and EDTA, or even reverse, like ATP, the active swelling of isolated mitochondria in vitro. In order to gather information on the

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