Effect of different times of exposure. Some teratogens produce abnormalities after exposure has ceased 10, 11, and in an attempt to analyse whether there is a critical period when embryos were vulnerable to tolbutamide, an experiment was conducted in which embryos were exposed for different lengths of time to a teratogenic concentration of tolbutamide. Early cleavage embryos (stage 5) were exposed to 3×10-4 M tolbutamide for 1 h, 3 h or continuously. Following exposure, embryos were washed in 2 changes of 10% Steinberg's saline and cultured in fresh saline. Exposure for 1 h or 3 h had no effect ($\chi^2 = 2.02$ and $\chi^2 = 2.02$), but as in the previous experiment, continuous exposure produced abnormal development ($\chi^2 = 37$, p < 0.001). The types of abnormalities were the same as those found previously. Thus it would appear that either continuous exposure is required for tolbutamide to be teratogenic, or that the vulnerable period is after the mid- to late blastula stage (3 h after stage 5). Effect of age of embryos. In the last experiment, early cleavage embryos (stage 5), midblastulae (stages 7-8) and late blastulae (stage 9) were exposed continuously to 3×10^{-4} M tolbutamide. When exposure was commenced at stage 5, 77% of the embryos were abnormal ($\chi^2 = 43$,

p < 0.001); when exposure was commenced at the midblastula stage, 73% of the embryos were abnormal $(\chi^2 = 38.5, p < 0.001)$ and when exposure was commenced at the late blastula stage, 53% of the embryos were abnormal ($\chi^2=15.8,~p<0.001$). The types of abnormal malities found were similar to those found previously, except that there was a tendency for embryos exposed at the blastula stage to form more neurulae with vesicles. These results show that under certain conditions exposure of early embryos of Xenopus laevis to tolbutamide leads to abnormal development. The type of abnormality produced is apparently not analogous to that produced in echinoderm embryos, but it is reasonably consistent. It is not known whether the action of tolbutamide in mammals is due to the drug or to the metabolic state it induces. The present results confirm that tolbutamide itself may be teratogenic4, and further experiments to analyse its mechanism of action in amphibian embryos are being undertaken.

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Dietary restriction and fetal development¹

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Summary. The effect of deficient nutrition of pregnant Wistar rats on the fetal weight has been studied. It has been established that the fetal weight of the group of rats fed with a restricted amount of stock diet lags behind the fetal weight of the group receiving unrestricted amounts of the same stock diet. The differences in weight between the 2 groups were, on each day of the observation period, significant at the level of p < 0.05 and p < 0.01, respectively.

Nutrition of the pregnant female certainly plays an important part in fetal development2. The effect of restriction of some food components, especially proteins, has been experimentally studied³. So far the studies have been mainly directed towards the effect of malnutrition on reproduction 4 and weight of the offspring 5 but little attention has been paid to the consequences of restricted

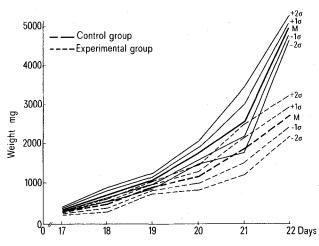


Fig. Mean weight values curves and curves indicating SE in the experimental and the control group.

total daily quantity of stock diet. The intention of this study was to gain an insight into the effect of malnutrition on the fetal development of rats. Restricted daily quantity of stock diet was applied only during the gestation. Material and methods. Experimental ad libitum feeding has shown that a female Wistar rat consumes 12 g of pelleted food, a produce of the pharmaceutical firm 'Pliva', composed of: crude proteins (min. 18%), crude fats (min. 4.5%), crude fibres (max. 3%), Ca (max. 1.10%), P (min. 0.65%), ash (max. 4%), moisture (max. 13%), vitamin A (22,000 IU), vitamin D₃ (2000 IU), Fe (400 mg) and I (0.37 mg) per kg of pelleted food.

Virgin female rats of Wistar strain were mated. The day of insemination was determined by the finding of the sperm in the vaginal smear and was taken as the first day of embryonic development. On that day the females were placed into single cages and were put on the restricted diet consisting of 6 g of the commercially prepared pellets besides unrestricted water consumption. The pregnant femals of the control group continued to receive both

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food and water ad libitum. Each day, beginning from the 17th to the 22nd day of gestation, females were sacrificed by ether. The fetuses, recovered on autopsy were all alive. They were weighed and their weight was recorded in mg. The mean weight of fetuses in one and the other group was calculated for each day of the observation period. The total number of observed fetuses was 145 in the experimental group, and 151 in the control group. The table shows the number of fetuses observed on individual days of fetal development in each of the 2 groups. Statistical analysis has shown that there were no significant differences between the groups with respect to the daily number of fetuses observed ($\chi^2 = 3.66$; [11, 10]). Results and discussion. The figure displays graphically the mean values of fetal weight by day of fetal development in both, experimental and control group. The difference between the fetal weight of the experimental and control group for each day of the observation period is statistically significant at the level of p < 0.05 and p < 0.01, respectively. The difference in weight was the smallest on the 17th day of fetal development and the greatest on the 22nd day. During the period between the 17th to 22nd day the fetal weight of the experimental group increased only for 2.430 mg, and that of the control group for 4.560 mg.

These results show a negative influence of the restriction of the daily quantity of food on fetal development. The negative effect manifested itself as a statistically significant retardation in fetal b.wt between the 17th and 22nd day of the fetal development. The mechanism of the effect of restricted daily quantity of food on the weight of the fetus is very complex 6. In parallel with the quantitative reduction of the total amount of food and its individual components, the supply of calories is reduced too. These are the factors that directly influence the fetal development.

Number of observed fetuses in the experimental and control group by day of gestation.

Days of gestation	17	18	19	20	21	22
Restrected	15	20	31	24	35	20
Unrestrected	18	16	35	19	32	31

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The relationship between basal bodies and the motility of Polytoma papillatum flagella

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Summary. The maternal flagellar apparatus of dividing Polytoma papillatum cells lose their basal bodies but retain their motility. It is concluded from this fact that basal bodies are neither essential for the structural maintenance of flagella nor for their motility.

During the course of vegetative cell division, the most familiar species of Polytoma and of Chlamydomonas differ in their behaviour of motion; Chlamydomonas cells lose their motility, whereas Polytoma cells retain theirs 2-7. During division, Chlamydomonas cells lack flagella but contain basal bodies 8-11. However, in Polytoma both flagella of the mother cell survive long enough for all the daughter cells to form their own flagellar apparatus 3, 4, 6. As conjectured by Schneider², exactly observed by Prowazek⁵ and confirmed by present electron microscopic investigation, the bases of the mother flagella of Polytoma are connected with the posterior end of one of the daughter cells until the daughters swarm out. The daughter cell, which provides locomotion for the whole maternal complex, finally leaves the mother wall by breaking away from the bases of the mother flagella with a distinctly perceptible jerk⁵.

Culture conditions and all the other preparation procedures were as described previously 18.

The structural details in the flagellar apparatus of Polytoma coincide nearly exactly with those of Chlamydomonas 11, 12; for this reason we can dispense with a detailed description. Comparison of our figures using Cavalier-Smith¹¹ nomenclature with descriptions and illustrations of Chlamydomonas flagellar apparatus 11, 12

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