e cell had been burst artificially during the preparation ig. 3). This shows that at least some of the molecules containing iron-59 were distributed in the enchylemma of the cytoplasm and were not bound to the morphological structures of the cytoplasm.

An accumulation of grains around and on the nucleus ould be seen in some autoradiographs. However, the umber of the grains per mm² on the cytoplasm was learly greater than those on the nucleus. The chromosomes were either unlabelled or showed very weak autoradiography. In squash preparations where the cytoplasm and the nucleus were burst artificially one could observe rains on the chromosomes, too (Fig. 4). But the number grains seems to be less than those on the intact nucleus nd varies from chromosome to chromosome. Therefore, can be suggested that some iron-59 molecules are not ound to the chromosome, but are localized in the nucleoasm as observed by Feldherr. No relation could be ound between labelling of cytoplasm and chromosomes. or example, in some cells the cytoplasm showed a light adioactivity, whereas the chromosomes were not labelled, nd vice versa. In some cases the nucleus showed stronger utoradiography than the cytoplasm.

Similar results were obtained in another experiment, when *Drosophila melanogaster* was used instead of *Chironomus plumosus*.

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BIOLOGY

Glucose Utilization in Pregnant Sheep by a Continuous Infusion Isotope Dilution Method

The importance of carbohydrate as a metabolic substrate in the ruminant has given rise to much discussion[1]. The question of the source(s) of glucose is of special interest because there is no evidence that hexose is absorbed from the ruminant alimentary tract and because acetate is the major end product of ruminal fermentation. The fœtal lamb, however, has considerable hexose requirements[2], and, as a consequence, it is thought that the pregnant female sheep is normally in a precarious state of carbohydrate balance[3]. It is known that even a slight fall in food intake in the pregnant sheep may cause the accumulation of ketone bodies in the tissues and blood stream followed by clinical signs of pregnancy toxæmia.

The continuous infusion isotope dilution technique[4] has been used to study glucose utilization rates in normal pregnant sheep and those with induced pregnancy toxæmia. Four determinations on two normal Clun Forest ewes, pregnant for 130 and 142 days respectively, gave a mean utilization rate of 1·35 ± 0·18 mg/min/kg, and two ketotic ewes, pregnant for 135 and 140 days, gave a mean rate of 1·22 ± 0·13 mg/min/kg. This difference is not significant ($P > 0·5$). The opportunity was also taken to use 1-^{14}C-glucose and 6-^{14}C-glucose in the same animal on successive days and the specific activities of the expired carbon dioxide were compared. It is generally accepted that the ratio of the specific activity of expired carbon dioxide, produced when 1-^{14}C-glucose is injected, to the specific activity of expired carbon dioxide, produced when 6-^{14}C-glucose is injected, that is, the C1/C6 ratio, should be unity if the main catabolic route for glucose is the Embden–Meyerhof pathway to pyruvate, followed by conversion to carbon dioxide in the tricarboxylic acid cycle. Deviation from unity is considered to indicate the existence of another pathway which incorporates C1 of the glucose into carbon dioxide at a greater rate than C6, for example, the pentose cycle.

The C1/C6 ratios for the two normal pregnant sheep were 1·24 and 1·38, respectively, and for the two ketotic animals 1·74 and 2·05. The mean value for the ketotic sheep did not differ significantly from the mean for the normal sheep ($0·1 > P > 0·05$). The situation in ketotic pregnant sheep therefore differs from that in ketotic lactating cattle in which lower C1/C6 ratios than in normal lactating cattle were found[5].

The glucose utilization rates in the pregnant sheep are of the same order as those in non-pregnant sheep after 24 h starvation, but only about half the rates in fed non-pregnant sheep[6]. It would seem therefore that tissue glucose supply is indeed marginal in pregnant sheep, but is not significantly reduced when the animals are showing clinical signs of ketosis. The C1/C6 ratio is of special interest. The decreased ratio in ketotic cattle has been interpreted[5] as indicating a reduction of glucose metabolism by the hexose monophosphate pathway. In a detailed discussion on the use of glucose labelled with carbon-14 for evaluating glucose metabolism[7] it has been pointed out that the C1/C6 ratio may be influenced by at least four variables, that is, the activity of the pentose and of the glycolytic pathways, the use of glucose in synthetic pathways and the proportion of triose phosphate oxidized to carbon dioxide. It is therefore not possible to interpret the C1/C6 ratio in pregnant sheep in terms of percentage pentose cycle activity, but it can be concluded that 1C of glucose is incorporated into CO_2 more readily than 6C in both normal and ketotic pregnant sheep.

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Effects of Lysergic Acid Diethylamide on the Nesting Behaviour of Male Pigeons

Present-day advances in psychotherapy have added impetus to investigations into the actions of drugs which affect the central nervous system and thus cause changes in behaviour patterns. One such psychomimetic agent, lysergic acid diethylamide (LSD-25), exerts both peripheral and central actions[1]. Some of these effects in various animals and humans are nausea, vertigo, hyperhydrosis, hypersalivation, mydriasis, vasodilation or vasospasm, hypotension or hypertension, bradycardia or tachycardia, and changes in the white blood cell count[2].

The vasomotor changes are partially related to disturbances in the hypothalamic-hypophyseal-adrenal axis[2]. Rinkel *et al.*[3], studying the effects of LSD-25 on human beings, suggested that this drug stimulated the pituitary-adrenal axis yet rendered the adrenal glands unresponsive to adrenocorticotrophic hormone. It is reasonable to assume that this pituitary response may also cause a change in the blood or pituitary levels of prolactin, which has been shown to be responsible for broodiness or nesting behaviour as well as the development of the crop in pigeons[4–6].

Thus, we considered that an investigation into the effect of LSD-25 on the nesting cycle of pigeons would shed further light on the above concepts.

Eight mated male homing pigeons were removed from their respective nests in a loft between 11 a.m. and noon 3 days after their mates had laid eggs. Observations have shown that at this time the nesting behaviour becomes stabilized into a distinct cyclic pattern. The males sit on the eggs from about 10 a.m. until about 3 p.m. The females will then sit from 3 p.m. until 10 a.m. on the following day. Four of the male birds received LSD-25 in a single dose of 0·2 mg/kg (intraperitoneal). The other 4 males served as controls and received an equivalent volume of distilled water as a placebo. 0·5 h after treatment, the females were removed from the loft and all 8 males were returned. The time required for each bird to return to its own nest, after being placed back in the loft, was recorded and the data were statistically analysed. This programme was repeated until each of the 8 pigeons had received 5 doses of the drug and 5 doses of water with at least a 48-h interval between repeated administrations.

Table 1. Inhibition of Nesting Behaviour of Pigeons with LSD-25

Bird No.	Drug-treated*	Control*
1	62·7	0·6
2	13·0	7·4
3	73·8	0·0
4	48·4	5·5
5	87·2	0·8
6	55·0	8·6
7	18·4	13·8
8	76·2	11·4
Grand mean ± *S.D.*	57·4 ± 35·8	6·0 ± 12·2

* Average time (min) for return to nests.

The results obtained are summarized in Table 1. They represent the average time (min) required by the pigeons to return to their nests. Each value shown is based on 5 injections of the drug or distilled water. The grand mean was derived from the individual responses of each of the 8 birds. The difference between the experimental and control grand means is highly significant as determined by the standard error of the difference between the means ($P < 0{\cdot}01$).

It is interesting to note that 2 pigeons (2 and 7) showed very little change in behaviour. These birds responded to the drug only during the first 3 exposures, after which they did not appear to show any reaction.

This temporary inhibition of the nesting response is not due to a peripheral motor inhibition. In view of the rapid metabolic rate of the pigeon and the fact that the peripheral symptoms wear off within 30–40 min (ref. 7) leads us to believe that the effect is due to changes within the central nervous system. It is possible that the drug, by acting on the hypothalamic centre in the brain, causes an inhibition of prolactin release from the pituitary. This lowering of the prolactin-level would decrease or inhibit the 'desire' to nest. It is also possible that the drug causes a change in the bodies' response to prolactin as it does with adrenocorticotrophic hormone. In order to substantiate these mechanisms, biochemical or bioassay studies are required.

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Effect of Reserpine on Rate of Mortality and Growth of Duck Embryos

Adams *et al.*[1], who injected varying doses of reserpine into the yolks of eggs of White Plymouth Rock hens during the first week of incubation, reported that in the embryos treated the mortality rate increased with increasing dosage, and that the average body-weight of the surviving embryos was lower than that of the controls. On the other hand, van Limborgh *et al.*[2] were unable to demonstrate a similar influence on the mortality rate in one-day Khaki Campbell duck embryos. Uchida[3] observed a rise of the mortality-rate with increasing dosage but no significant changes in the body-weight in eighteen-day-old White Leghorn egg embryos.

The discrepancies in these results seemed to justify a re-examination of the influence of reserpine. For this work eggs of Khaki Campbell ducks were used. Three experimental series were drafted: in series I reserpine was injected after four days of incubation, in series II after eleven days, and in series III after eighteen days. Each series consisted of 96 eggs with living embryos, and these were subdivided at random into four series of 24 eggs (sub-series *a*, *b*, *c* and *d*). Each time doses of 10, 50 and 250γ of reserpine (0·20 ml.) in the form of 'Serpasil' were injected into the yolks of the eggs of the sub-series *b*, *c* and *d*; only the solvent was injected into the eggs of sub-series *a*. Four days after injection the eggs were opened and the number of deaths determined. The surviving embryos were all fixed and weighed under standardized conditions. Statistical analysis of the results included determination of the significance of differences between percentages, Student's *t* tests on the body-weights and Terpstra's tests against progression in samples.

The results of the experiments are summarized in Table 1.

Table 1. Summary of the Results of the Experiments

Series	No. of embryos treated	No. of embryos died	Mortality-rate (%)	Surviving embryos No.	Surviving embryos Mean body-weight (g)
I*a*	24	2	8·3	22	0·271
I*b*	24	3	12·5	21	0·232
I*c*	24	4	16·7	20	0·222
I*d*	24	10	41·7	14	0·214
II*a*	24	4	16·7	20	3·61
II*b*	24	6	25·0	18	3·90
II*c*	24	9	37·5	15	3·56
II*d*	24	10	41·7	14	3·62
III*a*	24	4	16·7	20	22·0
III*b*	24	8	33·3	16	21·9
III*c*	24	14	58·3	10	18·7
III*d*	24	23	95·8	1	(17·1)

With regard to the mortality-rate, Table 1 shows that, within each series, this rate rises with increasing dosage. The differences between the individual mortality percentages of the various sub-series are significant in only part of the cases, but the rise noted is highly significant in each series (series I: $P < 0{\cdot}002$; series II: $P < 0{\cdot}02$; series III: $P < 0{\cdot}0001$). Comparison of the mortality-rates found in sub-series I*a*, II*a* and III*a*, shows that in the controls mortality does not rise with increasing age ($P > 0{\cdot}20$). However, following the administration of any dose of reserpine the mortality rate rises significantly with the age of the embryos (10γ, $P < 0{\cdot}05$; 50γ, $P < 0{\cdot}02$; 250γ, $P < 0{\cdot}002$).

With regard to the body-weights of the surviving embryos, Table 1 shows that the mean body-weight of the controls of series I is much higher than that of the reserpine-treated embryos of this series. The differences are significant ($P < 0{\cdot}05$, $P < 0{\cdot}001$ and $P < 0{\cdot}001$ respectively). The smaller differences between the sub-series I*b*, I*c* and I*d* are not significant. Nevertheless, there is a significant correlation between increase of the dose and decrease of the body-weight ($P < 0{\cdot}0003$). In series II there are neither significant differences nor a systematic rise or fall of body-weight with the dose; however, in this series the individual body-weights varied extremely. In