

Twenty-five seedlings were used for each beaker, and in each experiment 50 seedlings were used for the 0.46 per cent. D₂O and the same number for the H₂O. The cultures were kept in darkness at 19°–20° C. for 96 hours, and at the end of this period the roots were measured. The results, shown in Table 3, indicate no significant difference in the effects of 0.46 per cent. D₂O and ordinary H₂O on the root elongation of the wheat seedlings.

The rate of the respiration of wheat seedlings at 28.5° C., as indicated by O₂ consumption, was measured by means of Warburg manometers. The seeds were soaked in the appropriate water for 3 hours and then allowed to germinate for about 24 hours on moist filter paper at 28° C. in a dark incubator. At the end of this period there was no difference in the appearance of the seedlings. Seedlings of the same root length were then placed on moist filter paper in the Warburg vessels. In each experiment three vessels, each containing 8 seedlings, were used for the dilute D₂O and three for the ordinary H₂O.

No difference was found in the rate of O₂ consumption by the seedlings in dilute D₂O and H₂O. The plot of O₂ consumption against time for the seedlings in each vessel gave practically a straight line over the period of an experiment (approximately $\frac{3}{4}$ hour). In each experiment the six lines (three for dilute D₂O and three for ordinary H₂O) lay close to one another, in a random order. The numerical data are given in Table 4.

TABLE 4
OXYGEN CONSUMPTION BY WHEAT SEEDLINGS^a

Exp.	Root length of seedlings used, mm	O ₂ consumption by 8 seedlings, cmm/min.	
		H ₂ O	D ₂ O
1	1–3	2.92	2.93
2	3–5	3.04	3.02
3	1–3	2.84	2.96
4	1.5–3.5	3.03	3.05
5	3.5–6	3.28	3.26
6	1–3	2.87	2.82

^a The rate given is the average from 3 vessels, the average deviation being less than ± 4 per cent. In experiments 1 to 4, 0.46 per cent. D₂O was used; in 5 and 6, 0.05 D₂O. In experiment 6 quartz sand was used instead of filter paper.

These results may be summarized by saying that in the investigation of four biological processes—namely, growth of *Aspergillus*, germination of conidia of *Erysiphe graminis tritici*, root growth of wheat and O₂ consumption by wheat seedlings—no significant difference was observed between the influence of dilute heavy water and that of ordinary water.

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PITYROSPORUM OVALIS AS A CAUSATIVE AGENT OF SEBORRHEIC DERMATITIS

It has been popularly considered by many dermatologists that *Pityrosporum ovalis*, the so-called "bottle bacillus" of Unna, is the etiological agent of seborrhea capitis simplex (dandruff) and seborrhea corporis (scaling of the body). Unfortunately, the inability to grow the organism for any length of time kept the clinicians from proving this belief in compliance with Koch's postulates. The organism is a yeast-like, budding, non-filamentous fungus, ovoid to spherical in form. When seen in scales, the cells are usually bottle- or gourd-shaped, ovoid and thin-walled, 2–4 × 2–3 μ in size, or spherical and thick-walled, 3–9 μ in diameter.

Because of its supposed relationship to bacteria, early investigators used bacteriological media for cultivating the microbe, with no success. Later workers, finding it to be a fungus, had somewhat better results using mycological substrates. No one had been able to cultivate *Pityrosporum ovalis* beyond one or two subcultures. One of the writers (Moore) inoculated fresh slants of wort agar (product of the Digestive Ferments Company), pH 4.8, with scales from scalps having seborrhea and was able to grow the fungus in approximately 12 per cent. of the attempts (8 out of 90). The microbe was successfully subcultured and used in experiments to test its pathogenicity, or at least its rôle in seborrhea.

Laboratory animals, including rabbits, guinea pigs, rats, white mice and hairless mice, were inoculated as follows: Intradermally with a saline suspension; percutaneously (scratching the skin) with and without a lipid salve; cutaneously; and controls. Rabbits and guinea pigs gave favorable reactions with percutaneous inoculations. Mice, rats and hairless mice were negative.

A number of humans were inoculated as were the animals and, in addition, auto-inoculations with scales from the patient's scalp were made on the chest and in the axillae. The percutaneous inoculation with the application of whole culture of *Pityrosporum ovalis* produced seborrheic dermatitis in 50 per cent. of the patients, while the same inoculation with the rubbing in of a lipid salve gave approximately 85 per cent. reactions. Intradermal injections produced a distinct erythema in approximately 75 per cent. of the cases. Cutaneous tests were not very convincing, while auto-

inoculations were successful in 50 per cent. of the attempts. The controls were negative in over 90 per cent. of the patients. These results seem to indicate that *Pityrosporum ovalis* may produce seborrheic dermatitis under favorable conditions.

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A DWARF MUTATION IN THE RAT

In rodents genetic dwarfs have been reported in the guinea pig,^{1,2} in the mouse by Snell³ and recently in the rabbit by Green, Hu and Brown.⁴ The dwarfism in the mouse was shown by Smith and MacDowell⁵ to be caused by a hereditary deficiency of the anterior pituitary. Preliminary studies in the rabbit suggest that the defect may be due to an endocrine abnormality, while in the guinea pig the nature of the dwarfism was not determined.

The dwarf mutation in the rat described herein was first observed in our colony in the summer of 1933. It appeared as one individual in a litter of nine young in a strain of rats that had been closely inbred in our laboratory for several years. Shortly afterward another dwarf was observed in a litter from a closely related pair, and subsequently in the litters of other closely related rats. At present 22 dwarfs have been produced in 12 litters, the ratio of normal to dwarf in these litters being 80 to 22. These results suggest that the dwarf is the result of a simple autosomal recessive mutation, the appearance of both dwarf males and females showing that the gene is not sex-linked.

At birth normal and dwarf rats can not be distinguished, and it is impossible to separate the dwarf rats with certainty until about the twelfth day after birth. At this time a distinct difference in rate of growth becomes manifest, and the hair of the dwarfs appears much thinner and of finer texture than the hair on their normal litter mates. This difference in the hair is a characteristic feature of the dwarfs throughout life. The mature weight of the dwarfs is approximately 50 per cent. that of the normal males and 70 per cent. of the normal females of this strain. A remarkable feature is the failure of the males and females to become differentiated in size, whereas the male normally becomes distinctly larger than the female.

Thus far all dwarfs have proven to be sterile. No sexual activity has been observed in dwarf males and in only one instance has copulation been noted among the dwarf females. The size of the testes remains infantile, becoming less than one-half normal size. Spermatogenesis occurs, but at a greatly reduced rate, and the few sperm observed were abnormal in appearance. As yet studies have not been made of the ovary.

In all cases so far observed a distinct opacity of the lens of the eyes has been associated with the dwarfism, although some variation in the degree of this opacity exists. Skeletons of the dwarfs have not been prepared, but the general proportion of the body parts appears the same as in normal rats. The effect seems to be a general reduction in size of all parts, and the retardation in rate of growth becomes manifest at a very early date. Using the method of analysis of variance on the weights of litters in which dwarfs appeared, it has been determined that the retardation in growth had begun on the fifth day. As pointed out above, however, individual dwarfs can not be separated with certainty until about the twelfth day after birth. The dwarf rats are weaker than normals, are more susceptible to infections, and are shorter lived.

Individuals heterozygous for the dwarf gene are normal in appearance, in vitality and they attain the same size as homozygous normal rats. Recently, however, some individuals have been observed among the progeny of dwarf-producing pairs that were slightly slow in developing their hair and whose early growth seemed somewhat retarded. Tests are now under way to determine whether such individuals are heterozygous for the dwarf gene.

The decreased size of the dwarf rats, their sterility and their general appearance suggests that the defect may be of endocrine origin. It is, however, apparently not due to a pituitary deficiency, since in preliminary tests with the implantation of pituitary bodies from normal rats, following the technique of Smith and MacDowell, growth was not produced in treated dwarfs. Further experiments to determine the physiologic cause of this dwarfism are at present under way and the colony is being expanded in order to insure a constant supply of the dwarfs.

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