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Relation of Humidity to Resistance to Infection in Rachitic Rats.

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McDowell,[1] showed that the mortality following an intraperitoneal injection of pneumococci in adult rats kept for 2 weeks in a room at a temperature of 84°F., was much higher when the relative humidity was 84% than when it was 44%. When the temperature varied between 65° and 72°F., a larger percentage of all the rats died, but variations in the humidity had no effect. Kligler and Olitzki[2] reported very similar results with adult mice, but, in their work, the exposure to the special environment took place only 3 days before and for one month after an oral infection with *Salmonella enteritidis*. At 68°F. differences in humidity did not influence the mortality rate.

The following experiments differ from those above mentioned in that (1) young rats were used; (2) a rickets-producing diet was fed; (3) the exposure to the various temperatures and humidities was for the 4 weeks preceding infection; (4) the effect of air drawn from inside and outside the building was compared. Litters of rats which had just been weaned were divided into 4 equal groups, and put in divided cages in 4 closed wooden boxes through which the conditioned air was passed. The air for 2 of the boxes, one high and one low humidity, was drawn from outside the building, and that for the other 2, also one high and one low humidity, was drawn from the large animal room which housed the boxes. The air for each pair of boxes was drawn from its source by a pump and passed through a tank of anhydrous crude calcium chloride, through an electric heater, and into the box. The heaters were under the control of thermo-regulators in the boxes, and the temperature of the conditioned air surrounding the rats was kept constant to within a few degrees. This warm dry air was flowing at the rate of about 30 liters per minute to each box, enough to ensure a complete change of air in each box every 10 minutes. A small jet of steam was led into the intake pipe of one box of each pair, and it was found that this almost saturated the atmosphere in those boxes with

* Under the direction of Alan Brown.

[1] McDowell, C., *Am. J. Hyg.*, 1923, **3**, 521.

[2] Kligler, I. J., and Olitzki, L., *Am. J. Hyg.*, 1931, **13**, 349.

water vapor without appreciably raising the temperature. Except for about one hour of the 24 daily, when the boxes were open for feeding, cleaning or inspection, the air conditions were kept fairly constant. The low humidity boxes were set at 83°-84°F. and the air entering them was 21% saturated with water vapor in the case of the one receiving outside air, and 24% saturated in the one receiving inside air. The high humidity boxes were somewhat less constant but maintained temperatures of about 81°F. and humidities of 80% to 100%, usually the latter. The rats were fed a modification of Steenbock's[3] rickets-producing diet, which contained considerable amounts of vitamins A, B and E, as follows: dried ordinary bread, 12½%; yellow corn, 59½%; wheat gluten, 17%; wheat germ, 6%; calcium carbonate, 3%; sodium chloride, 1%; machine dried alfalfa, 1%. The animals showed a marked degree of rickets after 28 days on this diet. The rate of growth was affected very slightly, if at all, by the high temperature, and the rats in all 4 boxes grew equally well.

After the rats had been kept under these conditions for 28 days, they were starved for 7 hours, put in individual cages, and fed a small amount, 0.0029 cc. (0.1 cc. of a 1 in 35 broth dilution) of an 18 hours' culture of *Salmonella muriotitis,*[4] a strain of *Salmonella enteritidis,* on a cube of dried bread. After infection the rats were kept at room temperature in individual cages for 28 days, when the experiments were terminated, and the degree of resistance was estimated by the percentage of each group which survived. All of the dead rats except one, which was in a state of decomposition, yielded *Salmonella muriotitis* from the heart's blood, usually in pure culture. The organism was identified by its non-utilization of lactose, and by a positive slide agglutination in a dilution of 1 in 100. No gross pathology other than that usually seen in this infection[5] was found. The livers and spleens of the survivors were cultured, but only one liver culture yielded *Salmonella muriotitis.*

TABLE I.

	Relative Humidity					
	21%–24%			80%–100%		
	No. Rats	No. Survived	% Survived	No. Rats	No. Survived	% Survived
"Inside" Air	9	5	55	10	0	0
"Outside" Air (April 18-May 17)	9	6	66	8	2	25

[3] Steenbock, H., and Black, A., *J. Biol. Chem.*, 1925, **64**, 263.

[4] McCordock, H. A., *Bull. Johns Hop. Hosp.*, 1925, **37**, 412.

[5] Robertson, E. C., and Ross, J. R., PROC. SOC. EXP. BIOL. AND MED., 1930, **27**, 999.

From the table it is seen that a much larger percentage of the rats kept at the lower humidity survived. Also, the rats which were supplied with air from outside the building withstood the infection slightly better, especially when the humidity was high, but whether this difference is of real significance will be further investigated.

In a similar preliminary experiment, the details of which are not given, too small a dose of the bacterial culture was fed, with the result that the organism was recovered from the cardiac blood of only one-third of the dead rats, and the mortality throughout was lower, but the deleterious effect of the high humidity was again evident, as 74% of 18 rats kept in the dry air, and 52% of 21 rats kept in the moist air survived.

Conclusions. When kept at a high external temperature (83°F.), young rachitic rats exposed to a relative humidity of 22% for 4 weeks survive a subsequent oral enteritidis infection in much larger numbers than litter mates exposed to a relative humidity of 90%.

These findings are similar to those reported by McDowell and Kligler.

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I. Penetration Through Tissue of Iodine in Different Solvents.*

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It has been shown by Karns[1] and by Karns, Cretcher and Beal,[2] that certain aqueous solvents for antiseptic iodine preparations are superior to those commonly in use, with respect to the iodine adsorption behavior at surfaces. This controls surface dosage, and hence becomes a very important factor in germicidal effectiveness[3] and in penetration through tissues. It has been further demonstrated by Karns[4] that the quantity of iodine absorbed on a treated surface is not a necessary function of the concentration of the iodine in the solvent used, and that surface tension plays no important rôle

* Contribution from the Mellon Institute Fellowship for Research in the Pharmacology of Iodine.

[1] Karns, G. M., *J. Am. Pharm. Assn.*, 1932, **21**, 779.

[2] Karns, G. M., Cretcher, L. H., and Beal, G. D., *J. Am. Pharm. Assn.*, 1932, **21**, 783.

[3] Gershenfeld and Miller, *J. Am. Pharm. Assn.*, 1932, **21**, 894.

[4] Karns, G. M., *loc. cit.*