several large feeding places and a network of runs to these from the burrows. Apparently they seldom left the beaten paths, and bait placed only a few inches off the route was never taken. Even the addition of codliver oil to the bait had little attracting power. It seemed necessary for the animals to come directly upon the food, almost touching it, before they were aware of its presence.

The animals exhibited some rather peculiar responses to the poisons used during the campaign. The usually recommended poisons[7,8] (prepared rodent poison and red squills) were found to be of the least value. This might be explained by the fact that the procedure had to be varied according to the habits of the animals. Barium carbonate, with meat and codliver oil, proved to be an efficient poison and was invariably effective on control rats in 8 to 12 hours. The recommended mixture of red squills had no effect whatever on controls. When mixed in higher percentages the rats would not take the bait satisfactorily. Strychnine-treated oats, prepared as rodent poison by the U. S. Biological Survey Station in Boise, Idaho, and distributed by the local county agent, were without effect on control rats. The most satisfactory poison was that prepared in the Missoula Health Department laboratory, consisting of strychnine-treated oats sweetened with Karo syrup and saccharin. This was safe to use and invariably effective within a few minutes.

Throughout the course of the extermination campaign rats were examined for signs of disease. Inspections for tularemia and plague lesions were negative. Fleas were rare. Liver cysts, in which scoleces were demonstrable, were found in several rats, and a few animals were seen with crusted eyes and the symptoms of the common mouse-pneumonia. A relatively large number showed hair loss, which might be the result of a diet deficiency, or perhaps due to parasitic infection. A large number, particularly among the younger rats, appeared to be rachitic.

The observations on this colony seem to indicate that albino rats, although supposedly weakened by years of domestication, are able to maintain themselves outdoors and to resist extermination. The absence of predatory animals, particularly the gray Norway rat, is probably an important factor in explaining the survival of the colony. It seems apparent, then, that food and water, underground shelter and absence of predatory animals comprise an environment suitable to the continued existence of albino rats under non-domestic conditions.

[7] W. C. Rucker, in "The Rat and its Relation to the Public Health," Gov't. Printing Office, Washington, pp. 153–162, 1910.

[8] J. C. Munch, J. Silver, E. E. Horn, *Jour. Am. Pharmaceut. Asn.*, 19 (8): 837–840, 1930.

SUMMARY

(1) A colony of albino rats existing under non-domestic conditions has been observed in Montana. The colony is known to have survived two winters.

(2) Some peculiarities in their habits and responses to common poisons are pointed out.

(3) Albino rats appear to be able to survive extreme weather conditions and to resist extermination from causes such as disease and unfavorable diet.

(4) The absence of predatory animals, particularly the gray Norway rat, is probably an important factor in the survival of the colony.

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CENTRIPETAL DRIFT: A FALLACY IN THE EVALUATION OF THERAPEUTIC RESULTS

THE fallacy to be described here has been observed twice in recent numbers of carefully edited medical journals,[1] and for that reason alone deserves the attention of investigators. It is likely that a search of the literature of therapeutics would yield numerous instances.

Using an instrument whose readings are affected by large chance errors, an investigator examines, say, 100 subjects. He selects the 10 people whose performance on the test happens to be the poorest, and gives them some kind of treatment. Next day he reexamines the 100 subjects. The average performance of the 100 is exactly what it was the day before, but the 10 who did most poorly then are now found to have improved strikingly.

To illustrate the principle involved, one may take 10 playing cards numbered consecutively from 1 to 10. One shuffles and distributes the cards among 10 people, here identified by the letters A to J:

A	B	C	D	E	F	G	H	I	J
3	7	4	6	2	9	8	10	1	5

One may express sympathy for A, E and I, who did so poorly on the test, tell them it must be the lack of vitamins in their diets and administer any desired treatment, such as the laying on of hands. Then the cards are again shuffled and distributed:

A	B	C	D	E	F	G	H	I	J
7	2	5	6	8	3	9	1	4	10

This time B, F, and H happen to hold the low cards,

[1] H. Jeghers, *Jour. Am. Med. Asn.*, 109: 756, 759, September 4, 1937. M. B. Corlette, J. B. Youmans, H. Frank and M. G. Corlette, *Am. Jour. of the Med. Sciences*, 195: 58, 59, 62, 63, January, 1938.

while A, E and I are found to have improved strikingly. It is even possible to show that the improvement is statistically significant, for their average before treatment was 2.00 ± 0.38, while after treatment it was 6.33 ± 0.80.

The shuffling of the cards and the subsequent dealing are, of course, analogous to the taking of measurements by means of instruments so poor, or under conditions influenced by so many uncontrolled variables, that the readings are completely determined by chance. But it is evident that the phenomenon must be at work whenever one works with tests whose results have a perceptible degree of chance error. This is the case with many of the tests used in biochemistry, physiology and psychology. This phenomenon must be suspected of being at work whenever one finds that the "supernormals" selected by the same test (subjects F, G and H in the above illustration) are adversely affected by the therapy. This fact, that *both* extremes on reexamination are found to gravitate in the direction of the mean for the whole group, suggests the name "centripetal drift" for the phenomenon.

A remarkable thing about this fallacy is that it can not be avoided by taking more readings on the subjects of the experiment. Thus one may deal the cards *twice*, and average the results:

	A	B	C	D	E	F	G	H	I	J
1st	3	10	2	8	7	4	9	6	5	1
2nd	6	2	10	7	9	1	3	8	4	5
Ave.	4.5	6	6	7.5	8	2.5	6	7	4.5	3

The four lowest are A, F, I and J. After treatment one again deals the cards twice:

	A	B	C	D	E	F	G	H	I	J
1st	8	2	5	6	7	9	3	4	10	1
2nd	4	1	2	6	9	8	5	7	3	10
Ave.	6	1.5	3.5	6	8	8.5	4	5.5	6.5	5.5

The experiment has now been done more carefully than before, and the improvement shown by A, F, I and J is very convincing; their average before treatment was 3.62 ± 0.44, and after treatment it was 6.62 ± 0.82.

One way to avoid the fallacy of the centripetal drift is to compute the index of reliability of the instrument by the method of self-correlations.[2] When this method is applied to the last set of readings above (8 and 4, 2 and 1, 5 and 2, etc.) one obtains an index very near zero; a very reliable instrument gives readings whose index is very near one. Some instruments used clinically give disturbingly low indexes.

Another way to avoid the fallacy is the time-honored device of dividing the subnormal group itself into a treated and an untreated (control) group.

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2 H. Sorenson, "Statistics for Students of Psychology and Education," New York, McGraw-Hill, 1936, p. 339*ff*.

THE CONCEPT OF ATAVISM

DURING the days when biologists were sedulously engaged in supplying the finishing touches to the house that Darwin built, it was the usual practise to regard every possible arrest in development or unusual character of an organism as the persistence or recurrence of an erstwhile normal feature of the particular organism's ancestor or ancestors. Such a reversion to the presumed ancestral condition was termed an *atavism*, from the Latin *atavus*, an ancestor. This concept, and the term expressing it, abounds in the writings of nineteenth century biologists. Haeckel's Biogenetic Law really represents a generalized synoptic version of this concept applied to a particular case, and calculated to resume a certain supposed routine of phenomena under a particular law. To-day few biologists believe that in its ontogenetic development any animal actually repeats the developmental stages of its phylogenetic history. In development the organism apparently passes only through those stages of development which are akin to similar stages passed through in the *ontogenetic* development of its ancestors; and this is essentially what von Baer said in his Hypothesis of Recapitulation, which is not to be confused with the so-called Biogenetic Law. In development the organism does not repeat the *adult* stages of its ancestors, but only those stages of development through which its ancestors as a whole have, more or less, in common passed. This, as von Baer originally pointed out, is why the early stages of related animals resemble one another more closely than do the differentiated adults. Modern recognition of these facts has brought the Biogenetic Law into disrepute.

The conception of atavism, however, persists. Reference to many modern texts on embryology, general biology and the writings of a fair number of morphologists, will supply examples of the uncritical usage of this term. One recent work by a notable worker has a section entitled "Reappearance of Lost Ancestral Structures in Man," and as *the* example of such structures we are given "the gill-pouches . . . [of] the early embryo . . . [which] may . . . persist and form an open fistula on the side of the neck." Surely, it is clear that such a fistula is due to the mal-development or arrest in development of an embryonic character of the individual and not to the reappearance of a character which the species and class has lost but which may have been present in some remote phyletic ancestor. In this connection it is worth drawing attention to the fact that the conventionally accepted homology between the gill-pouches or arches of fishes and the branchial arches of mammals is open to serious question.[1]

1 E. Gaupp, *Ergeb. Anat. u. EntwickGesch.*, Bd. 14, p. 808, 1905; A. C. Bruni, *Arch. Ital. Biol.*, vol. 51, p. 11, 1909; G. R. De Beer, "The Development of the Vertebrate Skull," Oxford, p. 406, 1937.