

$$R_s^T = e^{-2\pi i S_{12}} R_s = (-1)^{2S} R_s;$$

the space reflection matrix is symmetrical for integral S , antisymmetrical for half-integral S . Hence, in any theory where I appears as a symmetrical matrix that commutes with R_s , the symmetry properties of B and R_s must coincide—which is the connection between spin and statistics. This situation is assured for a theory so constructed that the Lagrange functions of the individual fields are invariant under space reflection (I commutes with R_s) and make no explicit reference to internal, charge degrees of freedom (the invariant matrix I is not antisymmetrical). It should also be realized that the restriction to matrices I that commute with R_s is not necessarily a physical assumption but can be a choice of representation. Thus, for half-integral spin fields, the transformation

$$\chi \rightarrow e^{\pi i/4 R_{st}} \chi$$

converts $R_s I$ into $R_s i R_{st} I$, where $R_{st} I$ also commutes with all $S_{\mu\nu}$ but reverses the commutation properties of I relative to R_s .

Finally, it should be acknowledged that all these remarks concerned with the connection between spin and statistics have counterparts in the earlier work of W. Pauli.

¹ J. Schwinger, these PROCEEDINGS, 44, 223, 1958.

² Some examples of the systematic use of this generator to derive field commutation properties are presented in "Differential Equations of Quantum Field Theory" *Lectures at Stanford University* (1956).

THE RELATION BETWEEN LONGEVITY AND OBESITY IN HUMAN BEINGS

BY LINUS PAULING

GATES AND CRELLIN LABORATORIES OF CHEMISTRY,* CALIFORNIA INSTITUTE OF TECHNOLOGY, PASADENA, CALIFORNIA

Read before the Academy, April 28, 1958

During recent years there has developed an increased interest in the question of the dependence of life span on various factors, such as weight, sex, country versus city dwelling, lipoprotein concentration in blood plasma, cigarette smoking, and exposure to high-energy radiation, and much statistical information about it has been gathered.¹ Some effort has also been made, in the case of factors, such as radiation exposure, that can assume a series of values, to express by an equation the relation between the average life span of populations differing with respect to this factor and the parameter representing the factor. Usually a linear equation has been used, often a single term, corresponding to proportionality between the decrease in expected life span and the parameter.

Sometimes a linear term plus a negative constant has been used, corresponding to a threshold value of the parameter, below which the linear expression is not valid. It is, of course, not to be expected that the function would have a discontinuous slope, and a better function can easily be formulated.

The average life span $L(x)$ of populations differing with respect to the parameter x and random with respect to other factors may be expressed as a power series in x :

$$L(x) = \sum_n l_n x^n. \quad (1)$$

In some cases the constant term (normal life span) and the linear term provide a satisfactory representation of the available information. An example is the decrease in life span due to increased incidence of leukemia following exposure to ionizing radiation, as discussed by Lewis.² As more information becomes available, it may be found that the linear function is not a satisfactory approximation and that further terms are needed.

Moreover, the first two terms of equation (1) provide an unsatisfactory representation of the function for large values of x , in that the life span becomes negative. In order that the simple function be valid for large as well as for small values of

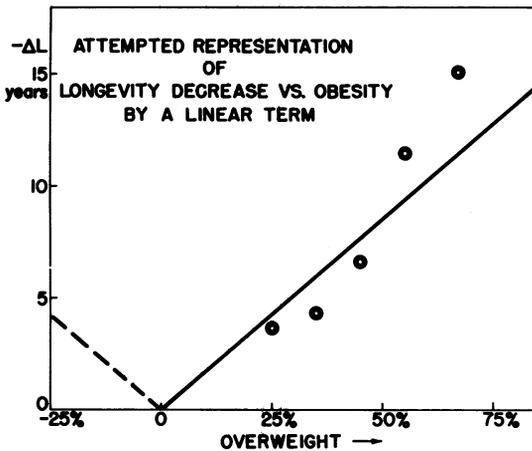


FIG. 1.—The best straight-line approximation to the five experimental points and passing through the assumed normal weight is shown. The dashed line indicates decreased longevity due to emaciation.

than the life span. $\Lambda(x)$ can be represented by a power series:

$$\Lambda(x) = \sum_n \lambda_n x^n. \quad (3)$$

There are some factors affecting longevity for which the power-series expression must include the quadratic term. An example is the dependence of longevity on body weight. Both obesity and emaciation decrease the life span. There is, accordingly, a body weight (related to height and build) that gives the maximum life span. If the parameter x is the fractional deviation from this optimal body weight the linear term $l_1 x$ or $\lambda_1 x$ vanishes, and the simplest approximate expression relating decrease in longevity to body weight is the quadratic term $l_2 x^2$ or $\lambda_2 x^2$. Because of the presumed difference in nature of action of obesity and emaciation, a cubic term would be expected to be required for a good approximation.

Statistical information about the decrease in life span in relation to obesity has been published by Dublin and Marks.³ The reported average decrease in life-span

of the parameter, the reciprocal of the life span or some other suitable function of it may be used. A convenient quantity, equivalent to the reciprocal of the life span, is the relative increase in longevity accompanying change of the parameter to zero, defined as

$$\Lambda(x) = \frac{L_0 - L(x)}{L(x)}, \quad (2)$$

with $L_0 = L(0)$. Λ becomes infinite as $L(x)$ becomes zero. In the case of a factor that is applied at some time in life, rather than beginning at birth, L may be taken as the life expectancy at that time rather

is 3.6 years for 25 per cent overweight; 4.3 for 35 per cent; 6.6 for 45 per cent; 11.4 for 55 per cent; and 15.1 for 67 per cent. These values have been described¹ as 0.17 year average effect of 1 per cent overweight, and the linear function has been assumed by other authors to be valid even for 1 ounce (0.04 per cent) of overweight.⁴

The five points and assumed linear function are shown in Figure 1. The approximation is rather poor, the mean deviation being 1.8 years. Moreover, as mentioned above, the function is an unsatisfactory one, in that there is a discontinuity in the slope at normal weight. A better approximation is two straight lines—one, with value zero, up to 18 per cent overweight, and the other, with slope about 0.31 year per per cent overweight, above 18 per cent. This function, too, is unsatisfactory because of the discontinuity in its derivative.

The best representation by a quadratic function with minimum at the assumed normal weight is shown in Figure 2. A somewhat better representation is given by a general quadratic function. The two functions are $36 w^2$ and $27.7 (w + 0.07)^2$, respectively, where w is the fraction over normal weight. The mean deviations for these two functions are 0.7 and 0.6 years, respectively.

The indication that a body weight 0.93 times that taken as normal maximizes longevity is unreliable. If equation (3) is used, rather than equation (1), a still better approximation is obtained (mean deviation 0.4 year) with no significant difference between optimal weight and normal weight. The best function is $\Lambda = 42.0 (w + 0.003)^2$.

The idea of 1 ounce of overweight (0.04 per cent) is, of course, a ludicrous one, inasmuch as the optimal weight is uncertain by some pounds, and it would not be justified to mention it had it not been introduced into a serious discussion. The effect predicted for 1 ounce overweight by the erroneous linear function is about 1,000 times greater than that predicted by the quadratic functions.

However, there is some significance to the question of the predicted decrease in longevity for 10 pounds of overweight. The decrease predicted by our treatment is about 50 days, as compared with a little over 1 year given by the linear function. The new interpretation of the evidence about longevity and obesity may diminish somewhat the anxiety of individuals who are a few pounds overweight.

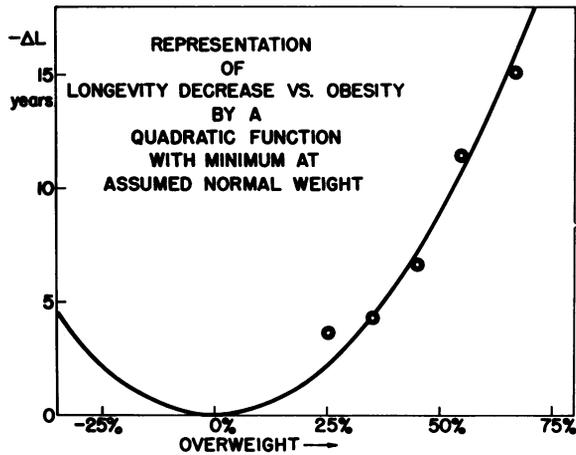


FIG. 2.—The best representation of longevity decrease by a quadratic function with minimum at assumed normal weight. The mean deviation for this function is one-third that for the best linear function.

* Contribution No. 2333.

¹ H. B. Jones, *Proc. Health Physics Soc.*, p. 114, 1956.

² E. B. Lewis, *Science*, 125, 465, 1957.

³ L. I. Dublin and H. H. Marks, *Mortality among Insured Overweights in Recent Years* (New York: Metropolitan Life Ins. Co., 1952).

⁴ E. Teller and A. Latter, *Life*, **44**, 64(1958); *Our Nuclear Future*, (New York: Criterion Books, Inc., 1958).

INTERRACIAL HYBRIDIZATION AND BREAKDOWN OF COADAPTED GENE COMPLEXES IN DROSOPHILA PAULISTORUM AND DROSOPHILA WILLISTONI

BY THEODOSIUS DOBZHANSKY AND OLGA PAVLOVSKY*

DEPARTMENT OF ZOÖLOGY, COLUMBIA UNIVERSITY, NEW YORK CITY

Communicated April, 1958

Certain local populations, or races, of the three sibling species, *Drosophila tropicalis*, *D. paulistorum*, and *D. willistoni* possess an interesting genetic structure.^{1, 2, 3} More than half the individuals in these populations are heterozygotes for the same inverted section in one of their chromosomes, and fewer than half are homozygous. In a panmictic population at equilibrium, such a state of affairs is possible only if greater proportions of the homozygotes than of the heterozygotes are removed by a differential mortality. Natural selection then establishes a situation known as "balanced polymorphism"; the Mendelian population in which it occurs may possess a high fitness, since the hybrid vigor (heterosis) in the heterozygotes compensates for the low adaptive value of the homozygotes.⁴

In *D. tropicalis*, a population in which 70 per cent of the individuals were heterozygous for a certain inversion, was encountered at Lancetilla, Honduras; elsewhere in Central America and in the West Indies the same chromosomal inversion had frequencies below 50 per cent, while in South American populations it was rare or altogether absent.^{1, 3} In *D. paulistorum*, the sample from Urubamba, Peru, contained significantly more than 50 per cent of heterozygotes, while in two other localities on the eastern slope of the Andes in Peru the heterozygotes amounted to less than 50 per cent.³ We have no evidence to show whether in these species the excesses of the heterozygotes are widespread or occur only in some small populations of isolated localities. In *D. willistoni* the situation is a little clearer. In at least three localities in northeastern Brazil the population samples contained more than 50 per cent of heterozygotes for a certain inversion (J, in the third chromosome), and in one of these localities samples taken on two successive years both showed this condition.³ Elsewhere in South and Central America this chromosomal inversion is heterozygous in 50 or less per cent of the individuals, but another inversion (E, in the right limb of the second chromosome) reaches frequencies higher than 50 per cent of the heterozygotes in the Brazilian state of Ceará.³

The experiments reported below were designed to elucidate the nature of the genetic difference between populations in which the incidence of the heterozygotes is above and below 50 per cent. Our working hypothesis has been that this difference is quantitative rather than qualitative. Under balanced polymorphism, the incidence of the homo- and heterozygotes in a population at equilibrium is deter-