If any one should repeat my work or extend it to other metals which I did not test I suggest that the following precautions be observed: Use wire coiled in large coils instead of wound on spools. If spool wire is used it should first be made smaller by a draw-plate and so straightened. Use fine wire, say .25 to .30 mm in diameter, so that after the cooling which necessarily ensues when a wire is stretched it may regain the air temperature before the lengthening is measured and before the elastic after-effect has a chance to appear. It was found that if the lengthenings were measured about twelve seconds after the application of the loads the measurements were not perceptibly vitiated either by the adiabatic cooling or by the elastic after-effect. Of course the wire should be long and one must always guard against error arising from changes in tower-temperature.

Since it is clear from the above that the ratio of stress to strain in a wire is a variable, it follows that the modulus of elasticity is also a variable, and so if we wish to speak of *the* modulus we should mean the modulus of the unstrained body. How to calculate this is shown in the original articles from which the above figures are taken.

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THE QUANTITATIVE THEORY OF SEX

THE quantitative theory of sex was first derived by the present writer in 1912 (preliminary note 1911), in essentially the same form as it stands today, from his experiments on intersexuality in the gipsy moth. (The term intersexuality was only used since 1915.) The theory claims that in both sexes determiners for femaleness and maleness are present, the relative quantities of which are balanced in such a way that one or the other has the upper hand in the respective sexes. Which of them is to be present in the higher quantity is decided by the mechanism of the sex-chromosomes, the meaning of which is thus explained, namely: one of the determiners (for maleness in the Abraxas type, for femaleness in the Drosophila type) is situated within the X-chromosome, the others (those for femaleness in the Abraxas type, for maleness in the Drosophila type) outside the X-chromosomes (in 1912 we assumed in the autosomes). Therefore, the always identical determiners for one sex are confronted either with one or with two doses of the determiners of the other sex. If the relative quantities of both are balanced in such a way that FF>M<MM (Abraxas type) and MM>F<FF (Drosophila type), normal sex-determination is explained on a quantitative basis: as a quantitative relation between male and female determiners, as I prefer to say, or a balance, as English writers prefer to call it. Intersexuality then appears, if the normal quantitative relation is changed in favor of the other sex in a definite way. When this theory was first derived in 1912 more than one sex-factor for each sex were assumed, namely, at least two for sex and secondary sex characters, and it was pointed out that each might really mean a group of linked factors. This notion has been abandoned since 1914, because the experiments show only the presence of one gene and therefore no necessity arose to split this into a group of linked genes. In 1912, further, the notion of the relative quantities was clearly indicated by the equations FF>M<MM. However, to fit these notions into the general genetical views the quantity of the genes was expressed in terms of their effect and called potencies or valencies. It was proved that M is inherited in the X-chromosome and it was assumed that F was inherited in an autosome. Later work, however, proved that in the gypsy moth F is inherited maternally (which is an experimental fact, not a theory) and it was made highly probable that this means inheritance in the Y-chromosome. Later, it was shown (and corroborated by Schweitzer and Lent) that in addition to the main factor for femaleness in the Y-chromosome there are at least two more factors for femaleness in the autosomes, the relation being a parallel to the main factor for spotting in rodents and its different modifiers. The last step was taken when (since 1917) the quantitative relation (balance) of the sex-differentiators could be linked with the embryological facts regarding intersexuality and thus the quantitative explanation of the mechanism of sex-determination could be enlarged into a physiological theory of sex-differentiation. The results of this analysis were published in a considerable number of papers since 1911, including a book (1920, also translated into English by Dakin) in which the quantitative theory was applied to the whole sex-problem and another book ("Die Quantitative Grundlage von Vererbung und Artbildung") in which the theory was developed into a general theory of heredity. I am glad to say that many zoologists and botanists have since accepted my theory as a solution of the sex problem and have assisted in their further development, thus creating quite an extensive literature about the subject. One of the most noteworthy among these contributions, which, however, had escaped the writer's notice, when publishing his book, was Standfuss's discovery of what nowadays is called triploid intersexuality (1908, 1914). Standfuss had shown a long time ago that sexual intergrades, at that time called gynandromorphs, were obtained in back-crosses from species-crosses in moths. After Federley's work on the chromosomes in such species-

crosses and the present writer's quantitative theory of sex were published he realized (1914) that both taken together give the explanation for these triploid intersexes: an abnormal balance of F and M as a consequence of the triploidy.

During the years between 1912 and 1922 the Columbia group of Drosophila workers was solidly opposed to my theory of sex. The different quotations of my work in their writings, among them a rather detailed review by Morgan and Bridges (1919) make it clear that they regarded facts and theory as something more or less queer which did not fit into their general views. Then Bridges (1922) rediscovered triploid intersexuality for Drosophila and was forced by the facts to accept every single point of importance of my quantitative theory. Bridges first realized that intersexuality as well as normal sex-determination can only be understood as a balance between female and male genes, as I had claimed since 1912. He further found that the female ones are situated in the X-chromosomes and the male ones outside, just as I had expected it for the Drosophila type since 1912. He further found that the male genes are situated in different autosomes. Here a difference between the gipsy moth and Drosophila appears; not a difference of theory but of fact. In Drosophila no factor in the Y-chromosome has yet been found and no principal genes for male sex (M), the others acting as modifiers as in Lymantria. This difference, however, might disappear with further work on Drosophila. Bridges prefers in his first paper to assume more than one gene for each sex, just as I had done in my first paper and prefers to call them + and − modifiers. In his later papers, however, he seems to get away more or less from this rather unfortunate notion and adopts further details from my theory, as the definite quantitative values, the epistatic minimum, etc. Of course there are differences in fact between triploid intersexuality in Drosophila and diploid intersexuality in the gipsy moth. In both the quantitative relation is regulated by the number of X-chromosomes. In the gipsy moth, however, where intersexuality occurs within the normal mechanism of the chromosomes, the decisive factor is the absolute quantity of the gene. Considering the other side of the equation in the triploid the abnormal quantities of male determiners (M) is produced by the presence of three sets of chromosomes instead of two, therefore three quantities of the respective genes instead of two. In diploid intersexuality, however, the number of such genes (here F) is always identical, only their absolute quantity being abnormal as a consequence of crossing races with different hereditary quantities of these genes. The method of breaking the normal balance of the sex-determiners is therefore different in both cases, the effect is the same and therefore the theory of sex determination derived from the facts is also the same.

It is rather unfortunate that Bridges omitted completely thus far to make a frank statement to the end that he has accepted now all the essentials of the present writer's theory of sex after ten years of opposition. Probably he intended to make such a statement in a forthcoming extensive paper and assumed meanwhile that the writer's work is sufficiently known to make such a statement unnecessary. During the last years, however, quite a number of papers have appeared (*e.g.*, by Sharp, Bonnevie, Bateson) in which the triploids of Drosophila have been hailed as opening a new insight into the sex problem. For the better information of such writers the present note is intended.

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A NEW SPECIES OF MONILIA PATHOGENIC FOR MAN

A NEW species of monilia pathogenic for man has recently been identified by the writer. It was isolated from a case of clinical pulmonary tuberculosis. Small, hard, whitish granules were present in the sputum. It was from these granules that the organism was isolated. Tubercle bacilli were not found in the sputum nor did tuberculosis result from injection of the sputum into the peritoneal cavity of the guinea pig.

The organism grows abundantly on dextrose agar, the growth being creamy white with a smooth surface and composed of budding, yeast-like cells. In dextrose broth the organism develops both budding forms and mycelial threads, the latter of which are often long and branched. Asci are absent. Without preliminary acidification, milk is rendered alkaline, which is manifest in forty-eight hours. Acid and gas are produced in dextrose, levulose, maltose and galactose. Neither acid nor gas is present in saccharose, lactose, mannite, dulcite, raffinose, arabinose, adonite, dextrin, sorbite or inulin. Neither gelatin nor blood serum is liquefied. Indol is not produced. There is no pellicle formed on broth.

The organism was found to be pathogenic for rabbits and guinea pigs when injected intravenously, but not when injected intraperitoneally. It was pathogenic for white rats when injected intraperitoneally.

For means of identification the writer suggests the name Monilia richmondi for the organism described above.

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