

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, Bonnevill, 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. Asepsis is 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, dulcitol, raffinose, arabinose, adonitol, dextrin, sorbitol 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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