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CORRESPONDENCE

SIR,—The following are a few points which occur to me on reading Dr. Hughes' paper on Resistance in Rheumatism.

The basis of the whole paper seems to be invalidated by a confusion between hypersensitivity and resistance, as is shown by the example quoted of Koch's phenomenon and the tuberculin test, both of which are the result of hypersensitivity or allergy, and not necessarily connected with the degree of resistance. It is now established that hypersensitivity can occur independently of high resistance and *vice versa*, though admittedly they are often coincidences. Therefore, if the analogy between the effect of measles, pregnancy, etc., on the tuberculin

reaction and on rheumatism holds good, it is the hypersensitivity that is reduced and there is no evidence at all to show that the resistance is either raised or lowered. A similar argument applies in connection with the Leprolin reaction in leprosy. The characteristic destructive lesion of adult tuberculosis and the lesions of rheumatism and leprosy are generally regarded as being due not to the direct effect of the reaction between the resisting host and the invading organism but as the product of hypersensitivity on the part of the host. The effect of high resistance in the absence of allergy is shown by the healed calcified primary complex in the case of tuberculosis.

The conception of the lesion of tuberculosis or rheumatism as " designed " for the purpose of resisting infection is highly unscientific to say the least. In fact, the evidence provided by Rich's work is all the other way. Furthermore, " resistance " does not depend entirely or even mainly on a local cellular reaction, but also on a general humoral mechanism.

Hill and Martin's work on the suppression of anaphylaxis by various agents does not seem to have anything whatever to do with the problem at issue. Anaphylaxis is not now regarded as playing any part in the defence mechanism of the body, and is generally held to be an undesirable side-effect due to hypersensitivity.

The effect of malaria on G.P.I. is beside the point. It is generally held to be due to the effect of the high temperature on the organism, and the immunological reactions of the host are not directly involved.

What the author's thesis seems to amount to is this: measures designed to decrease hypersensitivity—that is to say, desensitisation—are successful in the treatment of rheumatism, and in this he is doubtless quite correct; but on the other hand he is guilty of a certain amount of loose thinking about the term resistance, as used in connection with rheumatism. The exact meaning of the term is vague, and no one has yet been able to demonstrate exactly what the resistance of the body is supposed to be directed against, and until a direct connection has been shown to exist between the lesion of rheumatism and a specific infecting organism the use of the term resistance is perhaps unsatisfactory.

I am, etc.,

I. R. S. GORDON.

BATH.
February 25, 1943.