

Zeitschrift: Bulletin der Schweizerischen Akademie der Medizinischen Wissenschaften = Bulletin de l'Académie suisse des sciences médicales = Bollettino dell' Accademia svizzera delle scienze mediche

Herausgeber: Schweizerische Akademie der Medizinischen Wissenschaften

Band: 8 (1952)

Heft: 1-2: Symposium über die Beeinflussung des reaktiven Geschehens durch Hypophyse und Nebennierenrinde = Symposium on the influence of the hypophysis and the adrenal cortex on biological reactions = Symposium sur l'influence de l'hypophyse et de la corticossurrénale dans les réactions biologiques

Artikel: The effect of cortisone on the histology of the tuberculin reaction

Autor: Gell, P.G.H. / Hinde, Isobel T.

DOI: <https://doi.org/10.5169/seals-307079>

Nutzungsbedingungen

Die ETH-Bibliothek ist die Anbieterin der digitalisierten Zeitschriften auf E-Periodica. Sie besitzt keine Urheberrechte an den Zeitschriften und ist nicht verantwortlich für deren Inhalte. Die Rechte liegen in der Regel bei den Herausgebern beziehungsweise den externen Rechteinhabern. Das Veröffentlichen von Bildern in Print- und Online-Publikationen sowie auf Social Media-Kanälen oder Webseiten ist nur mit vorheriger Genehmigung der Rechteinhaber erlaubt. [Mehr erfahren](#)

Conditions d'utilisation

L'ETH Library est le fournisseur des revues numérisées. Elle ne détient aucun droit d'auteur sur les revues et n'est pas responsable de leur contenu. En règle générale, les droits sont détenus par les éditeurs ou les détenteurs de droits externes. La reproduction d'images dans des publications imprimées ou en ligne ainsi que sur des canaux de médias sociaux ou des sites web n'est autorisée qu'avec l'accord préalable des détenteurs des droits. [En savoir plus](#)

Terms of use

The ETH Library is the provider of the digitised journals. It does not own any copyrights to the journals and is not responsible for their content. The rights usually lie with the publishers or the external rights holders. Publishing images in print and online publications, as well as on social media channels or websites, is only permitted with the prior consent of the rights holders. [Find out more](#)

Download PDF: 04.08.2025

ETH-Bibliothek Zürich, E-Periodica, <https://www.e-periodica.ch>

Department of Experimental Pathology, University of Birmingham

The Effect of Cortisone on the Histology of the Tuberculin Reaction

By **P. G. H. Gell and Isobel T. Hinde**¹

We have described in the Congress, which has just ended, our work on the histology of the tuberculin reaction. Our conclusion was that this reaction should not be compared with a simple inflammation, but rather with the changes which take place in lymphoid tissue of a sensitised animal after renewed contact with antigen. This work was supplemented by an investigation of the effects of cortisone on the histology of the tuberculin reaction.

Our material was taken from rabbits sensitised by the injection of dead tubercle bacilli in paraffin. Mild lesions (about 10 mm in diameter) were produced by the intradermal injection of 80 I.U. of Old Tuberculine, a dose too small to produce necrosis; these were biopsied at 4 and 22 hours. For comparison a small amount of crude brain phospholipid was injected to produce a non-progressive acute inflammatory reaction of similar size, also without gross necrosis.

Two pairs of animals were used. Four hours before the injection of tuberculin one of each pair was given 30 mg of cortisone (Merck) subcutaneously. Another injection of 10 mg was given after the 4 hour biopsy, perhaps unnecessarily.

Macroscopically treatment with cortisone produced an obvious reduction in the intensity of the tuberculin reactions as compared with those of the controls; this effect has been described by other workers. There was no obvious effect on the simple inflammatory reaction.

The histological changes are striking, although a quite obvious cellular infiltration with the characteristic focal, perivascular pattern of the tuberculin reaction occurs in the cortisone-treated animals; but it is much less intense. Detailed examination shows that the reduction is mainly due to a deficiency of mononuclear cells. There is also a reduction in the mononuclear component of the simple inflammatory reaction,

¹ In receipt of a whole-time grant from the Medical Research Council.

**PERCENTAGE OF MONONUCLEARS
in lesions in Tuberculin-sensitive rabbits**

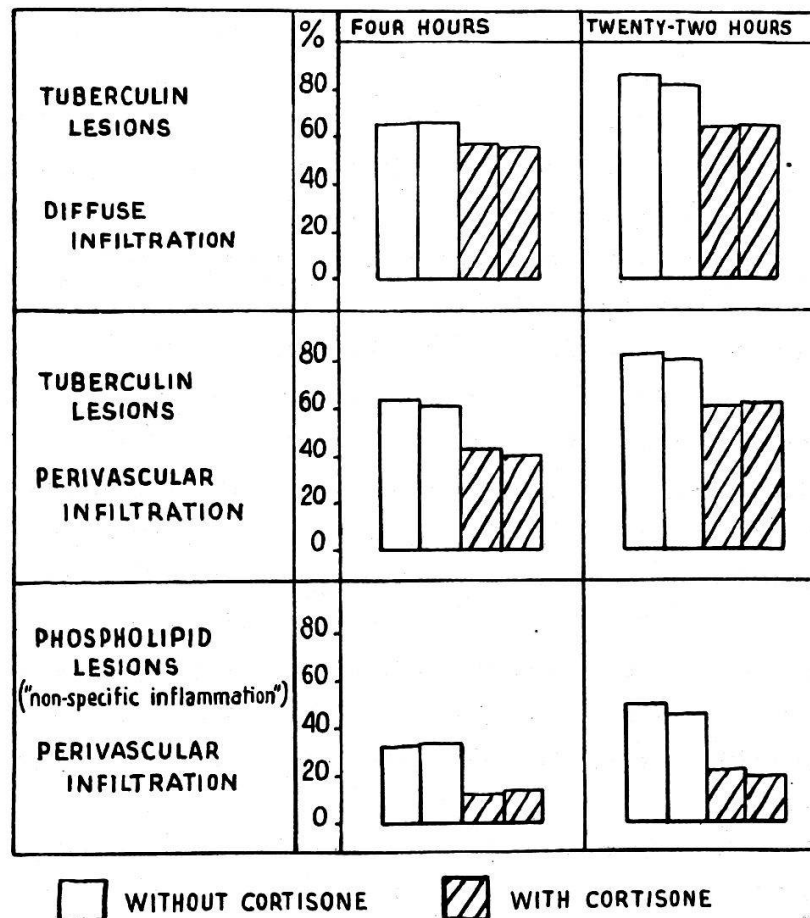


Fig. 1.

although as this is always subsidiary to the polymorphonuclear component it is not at all striking at first sight.

A quantitative estimate of the relative effect of cortisone on the two cell-types may be got from cell-counts made on comparable areas in sections from treated and untreated animals. Although the absolute figures must be treated with caution, since it is impossible to select strictly comparable areas and animals of identical sensitivity, the *proportion* of mononuclears present will show whether there has been a relative suppression of this type of cell. Counts were therefore made on two separate sites, the strip of diffuse infiltration in the superficial dermis, and the scattered foci of more intense infiltration round blood vessels. The ratios which are summarised in fig. 1 show that in every case there is a diminution in the proportion of mononuclears when treated and untreated animals are compared, which can be shown to be statistically significant.

This effect of cortisone, it must be noticed, is not equivalent just to a diminution in sensitivity. The animal pairs were of unequal sensitivity,

two being strongly and two moderately sensitive, one in each pair being treated with cortisone; but it is clear that the proportion of mononuclears is virtually identical in animals given the same treatment, although the total numbers of cells are less in the less sensitive animals.

It should also be noticed that the deficiency of mononuclears after cortisone is as striking in the non-specific inflammatory lesions as it is in the tuberculin lesions, showing that no specific anti-allergic effect of the hormone need be postulated here, the greater effect on the allergic lesion being merely because the mononuclear is the predominant cell.

One can analyse the effects of cortisone a little further, though our conclusions can only be tentative, as it is impossible to be certain where the individual reacting cells have come from. At four hours, small free mononuclear cells, very like blood monocytes, are common throughout the more vascular areas of the dermis in the normal tuberculin reaction especially immediately around small blood-vessels. In the reaction, modified by cortisone they are extremely rare, in fact the mononuclear reaction appears to consist almost entirely of a hyperplasia of the local reticulo-endothelial cells. Since these are most numerous round blood-vessels, hair follicles, etc., the characteristic multifocal pattern is preserved. The appearance of the cortisone-modified reaction suggests in fact that these are the cells which have produced what reaction there is, while it is the migration of monocytes from the blood-stream which has been largely suppressed, a not unreasonable supposition in view of the lymphopenia. There is reason to suppose that this explanation still holds at 22 hours, though it is now even more difficult to assign an origin to the developing cells.

We have suggested that the tuberculin reaction should be considered as an attempt to build up reticulo-endothelial tissue at the point of attack in the body. The effect of cortisone in interfering with such a build-up is quite consistent with its known ability to inhibit the reticulo-endothelial hypertrophy required for the production of anti-bodies.