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Letters to the Editor

The immunoregulatory abilities of polymorphonuclear neutrophils in multiple sclerosis

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EDITOR – In their very interesting work, Ziaber *et al.*[1] raised a question concerning involvement of polymorphonuclear neutrophils (PMNs) in the pathogenesis of multiple sclerosis (MS). Their results provide new and, from the point of view of recent concepts, rather unusual insights into MS pathogenesis.

We were pleased to read this. In our earlier experiments, we also found some evidence of abnormal PMN activation in MS. Moreover, these abnormalities appeared to be dependent on the interaction with autologous thrombocytes. We used two test systems: the nonadherent response of leukocytes elicited by calcium ionophore A23187; and, furthermore, we investigated the luminol-dependent chemiluminiscence.

Using the ionophore stimulation, we found that granulocytes preincubated with autologous thrombocytes (but not granulocytes alone) had increased nonadherent response in healthy controls. This phenomenon was less expressed in MS patients and absent in MS patients taking corticosteroids. We speculated that these results may reflect a thrombocyte-dependent alteration of adhesive membrane properties during the early phase of nonspecific PMN activation.[2] In the chemiluminiscence studies,[3] the addition of autologous thrombocytes markedly depressed both the spontaneous and the zymosan-stimulated granulocyte chemiluminiscence in MS patients and in patients with some other neurological diseases. In healthy controls, this was expressed less pronouncedly. These findings might indicate an activation of thrombocyte suppressive or scavenger function in MS and some other neurological diseases.

Although the PMNs seem not to be directly involved in the demyelination process within the central nervous system, they might play an important part in the complex interactions during vascular and hematogenic processes associated with immunoactivation in MS. In our opinion, this level of MS immunopathogenesis deserves greater and more intensive attention.

References

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Reply to M. Mayer

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EDITOR – I agree with Dr Mayer that PMN cells, especially in the blood of MS patients, are not only 'the silent observers'. In my study, I have described the enhanced extracellular markers expression on PMNs that might suggest priming of PMNs of the peripheral blood. In the course of clinically active MS, the role of PMNs in MS has not yet been shown. Our study does not show it either, thus there is still an unsolved problem as to whether these cells play an important role in the course of MS and how they are activated.

Dr Mayer suggests, on the basis of his study, that the changed reactivity of PMNs in the course of MS is due to thrombocyte-dependent alteration of the membrane properties during the early phase of nonspecific PMNs activation. This cannot be excluded. It is worthwhile to express that, in both studies, Dr Mayer draws conclusions based on *in vitro* studies. Furthermore, PMNs were isolated from patients during different stages of MS activity. In MS patients without corticosteroid treatment, rather heterogeneous results were obtained, which appears to reflect the fluctuation in the disease activity. In my opinion, the study would have been more interesting if the study groups had consisted of MS patients in the same stage of MS activity.

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