mined whether the astrogliosis is a reaction of the astrocyte to the axons or if it is inherent to the glial cell. As regards the relationship between the astrogliosis and myelination, the critical question is not so much the cause of the astrocytic abnormality but whether the condition itself interferes with myelination.

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[1] Meier, C. & Bischoff, A. *Nature* **267**, 177 (1977).
[2] Skoff, R. P. *Nature* **264**, 560–562 (1976).
[3] deF. Webster, H., Reier, P., Matthieu, J. & Quarles, R. *J. Neuropath. exp. Neur.* **35**, 309 (1976).
[4] Blakemore, W. F. *J. neurol. Sci.* **20**, 73–83 (1973).
[5] Adams, R. D. & Kubik, C. S. *Am. J. Med.* **12**, 510 (1962).
[6] Perier, O. & Gregoire A. *Brain* **88**, 937 (1965).
[7] Prineas, J. *Human Path.***6**, 531 (1975).
[8] Oppenheimer, D. R. in *Greenfield's Neuropathology*, 470 (1976).

Nature of stromatoporoids

KAZMIERCZAK[1] argues that the spherulitic microstructure of stromatoporoids, together with the spherulitic infilling of astrorhizal canals in specimens described from the Upper Devonian of south-eastern Poland, indicate that they have been formed by the calcification of coccoid blue-green algal cells. This suggestion, that stromatoporoids are calcified cyanophyte colonies, is novel and stimulating but it is open to two principal objections. First, it fails to consider alternative explanations of the spherulites; secondly, it overlooks a variety of additional complex morphological features of stromatoporoids.

Stromatoporoids have also been compared with coelenterates[2,3] and sponges[4] and representatives of both groups have skeletons composed of aragonitic spherulites. The similarity of this to the granular or melanospheric microfabric of stromatoporoids has been detailed by Stearn[5]. Several non-skeletal origins of the spherule-like infillings of the astrorhizae described by Kazmierczak[1] are possible, including sedimentation of fine peloidal material, cementation, and recrystallisation. However, Kazmierczak does not deal with any of these points but instead assumes that calcification of coccoid cells must be the origin of skeleton and infilling alike.

The characteristic skeletal features of stromatoporoids such as laminae, pillars and canals together with the not uncommon cyst plates, basal layers and external epithecal coverings, appear to have no parallels in cyanophyte colonies. The permineralised cyanophyte colonies[6] cited by Kazmierczak do not resemble stromatoporoids.

Kazmierczak is essentially proposing that stromatoporoids are skeletal stromatolites[7] of the thrombolitic variety[8]. Stromatoporoids and stromatolites, although superficially similar, differ in external morphology, internal structure, and geological distribution. The complex and varied internal morphology and limited geological range of stromatoporoids, together with their tendency toward discrete forms, strongly suggest that they are higher organisms than cyanophytes.

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[1] Kazmierczak, J. *Nature* **264**, 49–51 (1976).
[2] Lecompte, M. in *Treatise on Invertebrate Paleontology*. Part F (ed. Moore, R. C.) 107–144 (University of Kansas Press, Lawrence, 1956).
[3] Kazmierczak, J. *Acta palaeont. pol.* **26**, 1–146 (1971).
[4] Stearn, C. W. *Lethaia* **5**, 369–388 (1972).
[5] Stearn, C. W. *Lethaia* **8**, 89–100 (1975).
[6] Horodyski, R. J. & Vonder Haar, S. P. *J. sedim. Petrol.* **45**, 894–906 (1975).
[7] Riding, R. in *Fossil Algae* (ed. Flügel, E.) 57–60 (Springer, Berlin, 1977).
[8] Aitken, J. D. *J. sedim. Petrol.* 37, 1163–1178 (1967).

Do antigen-specific helper factors in rabbit lack V-region Ig determinants?

TAUSSIG, Finch, and Kelus[1] proposed that antigen-specific helper factors derived from T cells in rabbit lack V-region Ig determinants. They further argued that T-cell surface receptor would also lack V-region Ig determinants since the V regions of both the helper factors and T-cell receptor would very likely be the same. Their proposal was based on the finding that sheep red blood cell (SRBC) specific helper factors derived from rabbit carrying an allotypic marker at the a locus in the V region of its immunoglobulins is not adsorbed by anti-allotype antibody (anti-al, Table 2 in ref. 1) conjugated to Sepharose. Since no adsorption occured the authors interpreted it as an indication that the allotypic marker was not present in the SRBC-specific helper factors. In fact, Table 2 in ref. 1 shows that the helper activity is partly adsorbed by anti-al-Sepharose; the number of plaque forming colonies (PFCs) is reduced from $3{,}200 \pm 365$ to $2{,}450 \pm 280$ as a result of adsorption (Table 2, ref. 1). Since nothing is known about the relationship between PFC and quantity of helper factor used, it is difficult to say anything about the percentage of factor adsorbed by the immunoadsorbent. If the quantity of factor used in the control giving $3{,}200 \pm 365$ PFC is far greater than the optimum required (minimum quantity required to give maximum PFC) then the reduction in PFC after passing through the immunoadsorbent observed may be quite significant and represent a substantial reduction in helper activity. But, the authors have not studied the dependence of PFC on quantity of helper factor and not made any attempt to quantitate the factor before and after passing through the immunoadsorbent. It seems to me that the crude data presented do not allow the proposal the authors have made.

If indeed the data show a lack of adsorption of the helper factor by anti-al-Sepharose, then the lack of adsorption can be explained in an alternate way also. Since the helper factor has the capacity to bind antigen and since the factor was generated by incubating lymphocytes with antigen, it is conceivable that the factor is complexed with antigen fragments. These antigen fragments, though non-immunogenic and incapable of sensitising the bone marrow cells, may be large enough to sterically block the allotypic (al) determinant and thus prevent recognition by anti-al antibody. The authors, thus, have not ruled out the possibility that V-region immunoglobulin determinants are present in antigen-specific helper factors and receptor molecules of T helper cells. Whether or not the antigen-recognising sites of T helper cell receptor molecules and the helper factors are controlled by immunoglobulin V genes in rabbit, thus, remains an open question.

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[1] Taussig, M. J., Finch, A.P. & Kelus, A. S. *Nature* **264**, 776–778 (1976)

TAUSSIG *et al.* REPLY—Dr Sundharadas has raised certain points concerning removal of the rabbit antigen-specific T-cell factor on immunoadsorbents carrying anti-immunoglobulin (Ig) reagents. The difference in numbers of PFCs which he points out is not significant (at the 5% level). The capacity of the anti-al allotype adsorbent was more than sufficient, on past experience, to have removed the factor had the latter carried the al allotypic marker. As stated in the paper, the method was sensitive to a 50% reduction in factor activity, based on titration. The question is really not whether al-carrying molecules could have escaped detection, but rather whether a proportion of factor molecules greater than 50% could have Ig V regions yet be negative for a allotype, as are about 10% of normal rabbit Ig molecules. This cannot be ruled out, as discussed in our paper. Thus, at the very least our results show that the factor molecules do not carry the Ig allotype in the same proportion as circulating Ig. The further point about possible blocking of the factor binding site and steric hindrance of the allotype we consider unlikely. Evidence against it is that the factor can be absorbed by antigen, indicating the availability of its binding site.

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