2 hemocyte types in question are already present in the hemolymph.

Steps in hematopoiesis imply, according to Rizki<sup>18,19</sup>, the transformation of prohematocytes into 'crystal cells' and plasmatocytes, and the transformation of the latter into 'podocytes' and finally 'lamellocytes'. However, in our experience spherule cells (= 'crystal cells') transform directly into coagulocytes (= 'lamellocytes'), while plasmatocytes which by the emission of pseudopods may turn into 'podocytes', never end up as coagulocytes (= 'lamellocytes'). We have further shown<sup>29</sup> that the lymph gland which grows progressively like an imaginal disc, releases at the end of larval life a single type of cell, viz. the oenocytoids. Thus, spherule cells more likely stem from embryonic mesoderm directly as seems to be the case with prohematocytes and plasmatocytes<sup>7,9,34,35</sup>. Or, alternatively, they might branch off from plasmatocytes at a later stage. Spherule cells appear to be implicated in the process of coagulation<sup>31</sup>, in the encapsulation of parasites<sup>25</sup>, and in the formation of melanotic tumours<sup>21</sup>. Moreover, they are capable of phagocytosis, as are all other hemocytes which presumably stem from embryonic mesoderm. We found the oenocytoids of the lymph gland to be the only element incapable of phagocytizing heat-killed, stained yeast cells. In any case it sems likely that the essential function of spherule cells is the stocking of compounds highly charged with tyrosine, as indicated by the Millian reaction in Rizki's<sup>20</sup> observations and by the Glenner and Lillie<sup>30</sup> reaction in our own observations (figure 1, L). Significantly, spherule cells in D. melanogaster<sup>20,33</sup> and in D. hydei alike, disappear abruptly at the approach of metamorphosis. This could mean that they contribute to the sclerotization of the pupal case. Biochemical analyses of hemolymph have shown that tyrosine derivatives, in particular tyrosine-o-phosphate in D. melanogaster<sup>36</sup> and  $\beta$ -glucosyl-o-tyrosine in D. busckii<sup>37</sup>, increase steadily through larval life, but diminish considerably just before pupation. This suggests that spherule cells function as a reservoir of tyrosine.

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- A. Weismann, Z. wiss. Zool. 14, 187 (1864).
- E. Metchnikoff, Arb. zool. Inst. Univ. Wien 5, 141 (1887).
- H. Viallanes, Annls Sci. nat. 14, 1348 61882).
- C. Pérez, C.r. Soc. Biol. 56, 781 (1904).
- A. C. Hollande, Archs Zool. exp. gén. 6, 283 (1911). J. C. Jones, Am. Zool. 2, 209 (1962).
- J.C. Jones, The circulatory system of Insects, Charles C. Thomas, Springfield, Ill. 1977.
- J.W. Arnold, in: Physiology of Insecta, p.201. Ed. M. Rockstein. Academic Press, New York and London 1974.
- 10 C.D. Price and N.A. Ratcliffe, Z. Zellforsch. 147, 537 (1974).
- M. Rooseboom, Archs néerl. Zool. 2, 432 (1937).
- D. Zachary and J. A. Hoffmann, Z. Zellforsch. 141, 55 (1973).
- B. Åkesson, Ark. Zool. 6, 203 (1953).
- R. Dennell, Proc. R. Soc. Lond. B 134, 79 (1947).
- J.C. Jones, J. Morph. 99, 233 (1956).
- J. C. Jones, J. Insect Physiol. 13, 645 (1967) 16
- 17 J. M. Whitten, J. Insect Physiol. 10, 447 (1964)
- 18 M.T.M. Rizki, in: Physiology of Insect Development, p.91. Ed. F. L. Campbell. Chicago University Press 1956.
- M.T.M. Rizki, J. Morph. 100, 437 (1957).
- M.T.M. Rizki, J. Morph. 100, 459 (1957).
- M.T.M. Rizki, J. Morph. 106, 147 (1960).
- M.T.M. Rizki, Am. Zool. 2, 247 (1962).
- M.T.M. Rizki, in: The Genetics and Biology of Drosophila, p. 397. Ed. M. Ashburner and T.R.F. Wright. Academic Press, London, New York and San Francisco 1978.

- A. J. Nappi, Ann. ent. Soc. Am. 63, 1217 (1970).
- A.J. Nappi and F.A. Streams, J. Insect Physiol. 15, 1551 (1969).
- E. Gateff, Science 200, 1448 (1978).
- E. Gateff, in: The Genetics and Biology of *Drosophila*, p. 181. M. Ashburner and T.R.F. Wright. Academic Press, London, New York and San Francisco 1978.
- M.T.M. Rizki, J. exp. Zool. 123, 397 (1953)
- Ž. Srdić and H. Gloor, Revue suisse Zool. 85, 11 (1978).
- S.W. Thompson, Selected Histochemical and Histopathological Methods, Charles C. Thomas Pub. Springfield, Illinois (1966).
- C. Gregoire, in: Physiology of Insecta, p.309. Ed. M. Rockstein. Academic Press, New York and London 1974.
- A.C.S. Crossley, in: Advances in Insect Physiology, p. 117. Ed. J.E. Treherne, M.J. Berridge and V.B. Wigglesworth. Academic Press, London and New York 1975.
- M.T.M. Rizki and R.M. Rizki, J. biophys. biochem. Cytol. 5, 235 (1959).
- M. Demerec, Biology of Drosophila, John Wiley, New York 1950.
- J.C. Jones, in: Regulation of Hematopoiesis, vol. 1, p.7. Ed. A.S. Gordon. App. Century Crofts, New York 1970.
- H.K. Mitchell and K.D. Lunan, Archs biochem. Biophys. 106, 219 (1964).
- P.S. Chen, H.K. Mitchell and M. Neuweg, Insect Biochem, 8, 279 (1978).

## Cerebrospinal fluid immune complexes in multiple sclerosis

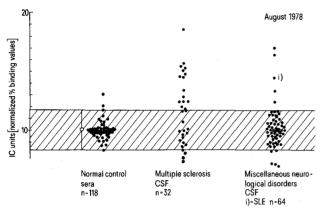
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Summary. Using a Clq binding test, immune complexes have been detected in one half of cerebrospinal fluid samples from patients with multiple sclerosis. These results provide additional evidence for the participation of an immune reaction in the disease process.

Several groups have reported on the presence of immune complexes (IC) in blood sera of patients with multiple sclerosis (MS) 1-3. Using the C1q binding test (C1q BT)4, we have examined cerebrospinal fluid (CSF) samples from 32 MS patients taken during an active phase of their disease as determined by a standardized clinical documentation system<sup>5</sup>. For the IC assay, CSF samples were mixed with one volume of normal serum (itself negative in the Clq BT). Amounts of IC were expressed in IC units, obtained

by dividing the percent binding values of the patient's sample by the arithmetic mean percent binding value of a larger pool (n = 118) of negative control sera from healthy individuals, and multiplying the results  $\times 10^6$ . Immune complexes were detected in 15 CSF samples, i.e. in 46.9% (figure). Positive results were also obtained in 6/64 control CSF from miscellaneous neurological disorders including several encephalitis cases (all IC -ve). Among the positive control CSF, 1 case of SLE with chorea is noteworthy. Whereas no correlations were found between the presence of CSF-IC and CSF-IgG or CSF IgG/albumin ratio, the CSF cell count was significantly higher in IC positive CSF samples (cell count in IC +ve CSF samples:  $39.9\pm52.5$ ; cell count in IC – ve CSF samples:  $14.8\pm18.0$ ; p < 0.01). Among 14 cases in which CSF and serum was examined on the same day, concordant results were obtained in 6 cases



Immune complexes in CSF of patients with mutiple sclerosis and in a control group from patients with miscellaneous neurological diseases

only. No connections between presence of IC in serum or CSF and clinical data such as duration, severity, or activity of disease<sup>6</sup> have become apparent; follow-up studies are needed to show such relations more clearly.

While no information is yet available concerning the nature of the antigen involved, we consider the presence of IC in MS-CSF as evidence for long-term antigenic stimulation within the central nervous system, i.e. at the site of tissue lesion, where local immunoglobulin production has been documented<sup>7</sup>. Whether such IC may have pathogenic significance, possibly suggested by the demonstration of IgG deposits in MS brain<sup>8</sup>, remains to be established.

- T.G. Tachovsky, H. Koprowski, R.P. Lisak, A.N. Theofilopoulos and F. J. Dixon, Lancet 2, 997 (1976).
- J.M. Goust, F. Chenais, J.E. Carnes, C.G. Hames, H.H. Fudenberg and E.C. Hogan, Neurology 28, 421 (1978).
- H. Jans, C. Jersild, E. Taaning, E. Dybkjaer, E. Fog and A. Heltberg, Protides Biol. Fluids 26 (1978).
- R.H. Zubler and P.H. Lambert, in: In Vitro Methods in Cellmediated and Tumor Immunity, p.565. Ed. B.R. Bloom and J.R. David. Academic Press, New York 1976.
- U. Patzold and W. Weinrich, Nervenarzt 46, 550 (1975). H. Deicher, H. Meyer zu Schwabedissen, W. Liman, B. Baruth, U. Patzold and P. Haller, in: Progress in Multiple Sclerosis Research International Symposium. Ed. H. Bauer, Springer, Berlin-Heidelberg-New York, in press.
- M. Sandberg-Wollheim, Thesis, University of Lund, 1975.
- B. F. Tavolato, J. neur. Sci. 24, 1 (1975).

## Conversion of glycine max seed agglutinins from nonspecific to anti-(A + B)

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Summary. The seeds of glycine max contain agglutinins which are typically nonspecific in their reactivity. Our investigations show that the phytagglutinins in GM can be converted from nonspecific to anti-(A + B) after the lectin is absorbed with horse red cells. The anti-A and anti-B fractions can be further separated by suitably absorbing the lectin with human red cells. The lectin absorbed with horse red cells or with group-0 human red cells shows an A-stressed activity.

Agglutinins in the seeds of glycine max (GM) are known to react nonspecifically with the red cells of several animal species including man and horse<sup>1</sup>. In this paper it has been demonstrated that the nonspecific glycine max lectin can be converted into anti-(A+B) after it is absorbed by the red cells of horse. An attempt has also been made further to isolate anti-A and anti-B fractions of the lectin by selective absorption with human red cells.

Methods. The seed extract was prepared in isotonic saline solution after Dunsford and Bowley<sup>2</sup>. The extract was stored in sterilized vials at  $-20\,^{\circ}\text{C}$ . 0.1% sodium azide was added as preservation. Absorption of the seed extract was carried out following the technique described by Moore et al.3. The absorbed lectin was tested soon after the final absorption was completed.

Observations. Table 1 shows the reactivity of GM lectin with 0, A, B and AB red cells. The lectin gave uniformly strong reaction with all types of red cells and behaved as a typical nonspecific phytagglutinin.

Reactivity of the GM lectin after its absorption with pooled human red cells is shown in table 2. Absorption of the GM seed agglutinins was carried out by mixing the lectin overnight with packed red cells of horse. It is interesting to note that all 6 samples of the absorbed lectin gave specifically negative reactions with pooled group-0 red cells. The absorbed lectin was further screened against 65 human red cell samples (table 3). It showed distinctly stronger reactivity (+ + to + + +) with A cells than with B cells (w + to + +) + + +) indicating that the absorbed lectin was probably Astressed. All 25 group-0 samples gave consistently negative reaction with the absorbed lectin.

A confirmation of anti-(A+B) activity in the GM seed extract was sought by absorbing the lectin separately with human 0, A, B and AB red cells. 2 fractions could be

Table 1. Reactions of glycine max seed agglutinins with human pooled red cells

Lectin	Pooled red cells				
	0	Α	В	AB	
Glycine max	+++	+++	+++	+++	

Table 2. Reactions of glycine max seed agglutinins absorbed\* with human red cells (pooled)

Absorbed lectin	Human pooled red cells				
	0	A	В	AB	
H <sub>1</sub> GM	_	+++	+++	+++	
H <sub>2</sub> GM		+++	+++	+++	
H <sub>3</sub> GM	_	+++	+++	+++	
H <sub>4</sub> GM	_	+++	+++	+++	
H <sub>5</sub> GM	_	+++	+ + +	+++	
H <sub>6</sub> GM	_	+++	+++	+++	

<sup>\*</sup>Absorption of GM seed agglutinins was carried out separately with the packed red cells of 6 horses (H<sub>1</sub>, H<sub>2</sub>, H<sub>3</sub> ... H<sub>6</sub>).