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Alternative, Non-immunoglobulin Origin of Amyloid Fibrils

By N-terminal amino-acid sequence analysis, Glenner *et al.*¹ have shown that amyloid fibrils are very similar to the variable part of an immunoglobulin kappa light chain (Bence-Jones protein Ker), and they suggested that amyloid is constituted by immunoglobulin light chains.

We have performed Edman degradation² on a 5,000 molecular weight subunit of two different amyloid preparations, one from the liver of a patient (T. H.) with juvenile rheumatoid arthritis, the other from the spleen of a patient (J. L.) with ankylosing spondylitis^{3,4}. After denaturation of isolated amyloid fibrils by guanidine or strong alkali respectively, the material was fractionated on 'Sephadex G-100'. Similar elution patterns and identical amino-terminal sequences were obtained with the material from the two denaturation procedures. The first 13-14 amino-acid residues of the 5,000 m.w. subunits of the two different amyloid preparations are shown in Table 1. They are very similar, but certain differences were observed. The sequences of both amyloids are quite different from immunoglobulin chains and indicate that amyloid fibrils in rheumatoid arthritis and ankylosing spondylitis, which are typical examples of secondary amyloidosis, may have a different origin than immunoglobulin polypeptide chains. The most noticeable observation was the high content of phenylalanine in the amino-terminal region of both amyloid proteins. We believe this to be a novel sequence of human proteins.

Table 1 N-Terminal Sequences of Amyloid Proteins

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
T. H.	Arg	Ser	Phe	Phe	<u>Ser</u>	Phe	Leu	Gly	Glu	Ala	Phe	Asp	Gly	Ala
J. L.	Arg	Phe	Phe	<u>Phe</u>	(Phe)	Leu	Gly	Glu	Ala	Phe	Asx	Gly	Ala	
	1	2	3	4	5	6	7	8	9	10	11	12	13	

Our data are in agreement with preliminary evidence of Benditt *et al.*⁵, who demonstrated that the amino-acid composition of some amyloid preparations (amyloid secondary to chronic inflammatory diseases) clearly differed from immunoglobulin polypeptide chains.

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Induction of the Feline Oncornavirus Associated Cell Membrane Antigen in Human Cells

Using indirect immunofluorescence we recently demonstrated a feline oncornavirus associated cell membrane antigen (FOCMA) in cultured feline lymphoma cells that produce feline leukaemia virus (FeLV)^{1,2}. Using electron microscopy and ferritin-labelled immunoglobulin, Oshiro *et al.* obtained similar results³. We found that cats with regressing feline sarcoma virus (FeSV)-induced tumours had high titres of FOCMA-specific antibody⁴, and that the presence of antibody in neonatal kittens was associated with resistance to tumour development². In the latter case, the antibody was apparently passively obtained from immune dams.

We investigated whether the FOCMA antigen could be induced *in vitro* by infecting cultured cells with FeLV or FeSV, especially cells from a species other than cats, such as man. The successful infection of human fibroblasts with FeLV has been reported⁴. It was also important to see if FOCMA was expressed *in vivo* in other species, such as marmosets, that are susceptible to tumour induction by FeSV⁵. We report evidence which indicates that FOCMA is induced in human lymphoblasts following infection by FeLV and expressed in marmoset monkeys given transplants of FeSV-induced tumours.

One to 45-week-old marmosets were given 0.1 g of live tissue from serially transplanted marmoset sarcomas originally induced with cell-free FeSV. As can be seen in Table 1, all of 7 such monkeys developed antibody titres that varied from 8 to 1,024 when assayed with a feline target cell. Sera from uninoculated control marmosets were negative. For assaying antibody on FeLV-infected cat lymphoma cells (FL74) the procedure was the same as previously described^{1,2}, except that fluorescein conjugated rabbit anti-rhesus monkey globulin (Sylvania Co., Millburn, New Jersey) was used in place of rabbit anti-cat globulin. Absorption of positive marmoset sera with normal uninfected cat fibroblasts gave no reduction in titre when assayed on FL74 cells. Since an otherwise identical uninfected cat lymphoblast target cell was not available, however, and since certain sera from adult rhesus monkeys have been found to have "natural" antibody against cat cells (M. E., unpublished observations) it seemed necessary to check the sera on *in vitro* infected and otherwise identical uninfected lymphoblasts.

A human lymphoblast culture derived from histopathologically normal tissue taken from a Burkitt lymphoma patient (PESS) was used. The culture is negative for the Epstein-Barr virus associated antigens routinely detected by immunofluorescence (G. K., unpublished observations). The culture was supplied by Dr S. Golub (Department of Surgery, University of California, Los Angeles). We believe this is the first report of FeLV replication in human lymphoblasts, but Hampar *et al.* have reported infecting the Burkitt lymphoma derived HR 1 K and Raji lines with FeSV⁶. For infection, cell-free FeLV concentrated from 30 ml. of FL74 culture supernatant (10⁶ cells/ml.) was added to approximately 10 million PESS cells. The virus concentration and infection procedures have been described⁷.

Using feline sera known to be highly positive for FOCMA, distinct membrane immunofluorescence was observed in the FeLV-exposed PESS line when examined at 30-120 days post-infection, but no reaction was observed in uninfected PESS cells (Table 2). Similar results were obtained with seven sera from FeSV-sarcoma bearing marmosets (Table 1). Sera from normal marmosets and normal cats were negative for both uninfected and FeLV-infected PESS cells. Also, the titres of FOCMA-antibody obtained on PESS-FeLV were either identical to or slightly less than the titres obtained on FL74 cells. The only noticeable difference between the fluorescence obtained with the PESS-FeLV culture and that obtained with the FL74 culture was that 95-100% of the FL74 cells were