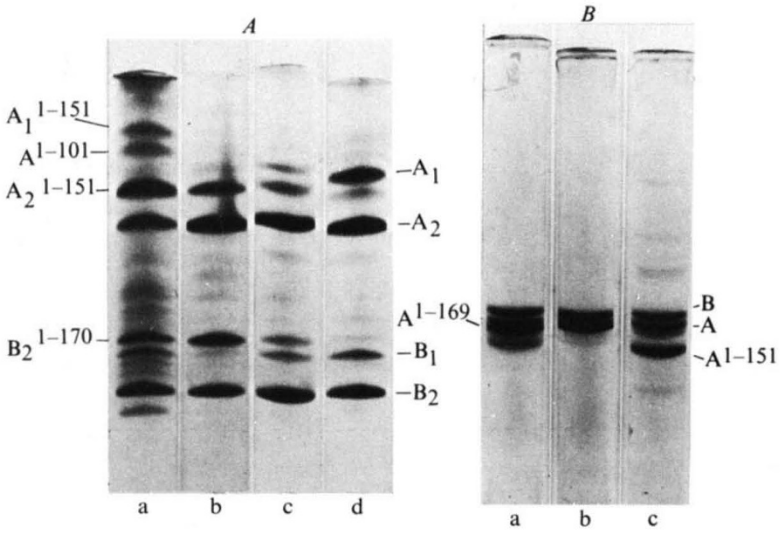

Fig. 3 *A*, Isoelectric focusing gels (*p*H 5–8) in 6 M urea of α-crystallin from: *a*, calf lens nucleus; *b*, 3-month-old foetus; *c*, 6-month-old foetus; *d*, calf lens cortex. *B*, SDS gel electrophoresis of α-crystallin from: *a*, 3-month-old foetus; *b*, calf lens cortex; *c*, calf lens nucleus.

This indicates that the formation of these chains continues in the adult nucleus, which is devoid of biosynthetic activity, and thus demonstrates the postsynthetic origin of these chains. Further proof comes from the fact that $A^{1\text{-}151}$ and $B^{1\text{-}170}$ can be produced *in vitro* simply by prolonged dialysis of α-crystallin against 0.1 M Tris-HCl buffer, *p*H 7.3 (ref. 15).

Our data thus show that the A_2 and B_2 chains are subjected to two types of postsynthetic modifications: deamidation and C-terminal degradation, as summarised in Fig. 1. The deamidation products A_1 and B_1 only appear in the later stages of foetal life and reach their highest level in the old bovine cortex, where a probable further deamidation product also becomes prevalent: B_0 (ref. 10). Postsynthetic deamidations have been demonstrated in other proteins, and can be obtained *in vitro* in proteins and synthetic peptides[16]. This deamidation process is non-enzymatic and its rate depends on the character of neighbouring residues[16].

The degradation of α-crystallin chains is apparently a postsynthetic process which starts early in embryonic life. The number of degraded chains is, however, clearly largest in the old bovine nucleus[10]. It seems, therefore, justified to consider these degradations as an ageing process. It seems that the chains are preferentially cleft at the sites indicated in Fig. 1. A stepwise breakdown, degrading the A chain successively to 169, 168, 151 and 101 residues, is suggested by the very high level of $A_2^{1\text{-}169}$ and the absence of $A^{1\text{-}101}$ in the embryonic lens, and the low level of $A^{1\text{-}168}$ and high level of $A^{1\text{-}101}$ in the old bovine nucleus[15].

Several features make it unlikely that the observed degradations are the result of enzymatic processes. The appearance of $A^{1\text{-}101}$ is a very slow process, being most pronounced in the nucleus, which implies that a putative enzyme becomes active only very late after its synthesis. If the enzyme were an endopeptidase it should have a specificity (compare Fig. 1) unlike any known endopeptidase. If it were an exopeptidase it should have carboxypeptidase C-like activity to remove proline residues, and be delayed at the sites indicated in Fig. 1. The fact that degradations can be caused by prolonged dialysis of purified α-crystallin would require that the possible enzyme has the same molecular weight as α-crystallin or that the degradation is an autolytic process. Also the action of lysosomal enzymes cannot be involved, since no lysosomes seem to be present in the nucleus of the lens[17]. In conclusion we propose that the observed degraded chains are the result of non-enzymatic breaks of susceptible bonds.

The knowledge obtained from the age-related phenomena in eye lens proteins may contribute to a better understanding of ageing in general; although it can be questioned whether the processes taking place in the ageing lens are representative of ageing in other types of cells. In addition, the knowledge of normal ageing of lens proteins is of great interest to the study of those forms of cataract which are considered to be the result of precocious ageing[18].

This work was supported by the Netherlands Foundation for Chemical Research (SON) and the Netherlands Organization for Pure Research (ZWO).

FRANS S. M. VAN KLEEF
WILFRIED W. DE JONG
HERMAN J. HOENDERS

Department of Biochemistry,
University of Nijmegen,
Nijmegen, The Netherlands

Received August 22; accepted September 19, 1975.

1 Schmike, R. T., and Katunuma, N. (ed.), *Intracellular Protein Turnover* (Academic, New York and London, 1975).
2 Goldberg, A. L., and Dice, J. F., *A. Rev. Biochem.*, **43**, 835–869 (1974).
3 Young, R. W., and Fulhorst, H. W., *Invest. Ophthalmol.*, **5**, 288–297 (1966).
4 van Kamp, G. J., Schats, L. H. M., and Hoenders, H. J., *Biochim. biophys. Acta*, **295**, 166–173 (1973).
5 van Kleef, F. S. M., and Hoenders, H. J., *Eur. J. Biochem.*, **40**, 549–554 (1973).
6 Stauffer, J., Rothschild, C., Wandel, T., and Spector, A., *Invest. Ophthalmol.*, **13**, 135–146 (1974).
7 van der Ouderaa, F. J., de Jong, W. W., and Bloemendal, H., *Eur. J. Biochem.*, **39**, 207–222 (1973).
8 van der Ouderaa, F. J., de Jong, W. W., Hilderink, A., and Bloemendal, H., *Eur. J. Biochem.*, **49**, 157–168 (1974).
9 van Kleef, F. S. M., Nijzink-Maas, M. J. C. M., and Hoenders, H. J., *Eur. J. Biochem.*, **48**, 563–570 (1974).
10 van Kleef, F. S. M., Willems-Thijssen, W., and Hoenders, H. J., (to be published).
11 Schoenmakers, J. G. G., and Bloemendal, H., *Nature*, **220**, 790–791 (1968).
12 Palmer, W. G., and Papaconstantinou, J., *Proc. natn. Acad. Sci. U.S.A.*, **64**, 404–410 (1969).
13 van Kamp, G. J., Struyker Boudier, H. A. J., and Hoenders, H. J., *Comp. Biochem. Physiol.*, **49B**, 445–456 (1974).
14 de Jong, W. W., van Kleef, F. S. M., and Bloemendal, H., *Eur. J. Biochem.*, **48**, 271–276 (1974).
15 van Kleef, F. S. M., thesis, Univ. Nijmegen (1975).
16 Robinson, A. B., and Rudd, C. J., in *Current Topics in Cellular Regulation*, **8** (edit. by Horecker, B. L., and Stadtman, E. R.) 247–295 (Dekker and Nordeman, Amsterdam, 1974).
17 Kuwabara, T., *Expl Eye Res.*, **20**, 427–443 (1975).
18 Bellows, J. G., and Bellows, R. T., in *Cataract and Abnormalities of the Lens* (edit. by Bellows, J. G.), 303 (Grune and Stratton, New York, 1975).

Erratum

In the article "Discrimination between eukaryotic and prokaryotic, and formylated and non-formylated, initiator tRNAs by eukaryotic initiation factor EIF–3" by I. G. Wool and R. S. Ranu (*Nature*, **257**, 616; 1975), the first four lines should read

PROKARYOTIC and eukaryotic cells contain two methionine accepting species of transfer RNA, $tRNA_f^{Met}$ and $tRNA_m^{Met}$ (refs 1–3). The formulated Met-$tRNA_f$ (fMet-$tRNA_f$) in prokaryotes[4] and non-formylated Met-$tRNA_f$ from . . .

and not as printed.

©1975 Nature Publishing Group