Comment

Misunderstanding or misrepresentation?

'Reticulocyte lipoxygenase, ingensin, and ATP-dependent proteolysis' by Shoichi Ishiura, Tanihiro Yoshimoto and Claude A. Villee [(1986) FEBS Lett. 201, 87–93]

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The authors purport to study the relationship between lipoxygenase, ATP-dependent proteolysis and 'ingensin', a fatty acid-dependent proteolytic system described by them. The thrust of the article is to show that lipoxygenase is not directly connected with ATP-dependent proteolysis. Such a claim has never been made. As a matter of fact, it was shown by us [1] that SHAM has no effect on the proteolysis of human serum albumin and that lipoxygenase has no direct influence on ATP-dependent proteolysis. Our claim has been that lipoxygenase triggers the ATP- and ubiquitin-dependent proteolytic attack on susceptible intact mitochondria. Of course, such an action cannot be studied with casein as substrate as the authors have done. Moreover, they have used phenylhydrazine to produce reticulocytosis despite the fact that they cite our report in which we have shown that phenylhydrazine anemia is not suitable for the study of biological degradation of mitochondria [2]. Phenylhydrazine damages mitochondria and gives rise to denatured and aggregated proteins which are substrates of both ATP-dependent and -independent proteolysis. Therefore, the triggering action of lipoxygenase is exhibited only to a slight degree.

Since lipoxygenase does not take part in the proteolytic system, comparisons carried out by the authors between the action of inhibitors on lipoxygenase and ATP-dependent proteolysis are beside the point of our work.

One must take exception with the way the authors deal with the literature. Just a few examples may suffice.

The statements that we had studied ATP-dependent proteolysis of hemoglobin, amino acid analog-containing abnormal proteins, casein or pulse-labelled endogenous proteins are incorrect; for our aim to study the breakdown of mitochondria, such experiments would have been senseless. The assertion, citing us, that ATP-dependent degradation catalyzes 1–2% of intracellular protein breakdown is also incorrect; we found about 90% to be ATP-dependent. The authors say in the introduction that our work "has not been confirmed by other investigators", without citing a single reference to back up the harsh criticism. Our recent work [3–6] is not cited, nor that of Haas and Bright [7]. The statement of the authors that

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"In mammalian cells, the ATP-dependent proteolysis of endogenous and exogenous proteins was demonstrated only in reticulocyte extracts and in liver mitochondria" is contradicted by both older reviews [8,9], and newer work in which ATP- and ubiquitin-dependent proteolysis was demonstrated in tumor cells, fibroblasts, kidney, liver and plant cells [10–15]. The authors state that "attempts to purify ATP-dependent protease(s) were unsuccessful", disregarding recent work [16] which appeared in February 1986. As far as 'ingensin' is concerned, it is barely mentioned and the only documentation referring to it consists of an SDS electrophoretic gel. No evidence is given for its role in the maturation of reticulocytes.

REFERENCES

[1] Dubiel, W., Müller, M. and Rapoport, S. (1981) Biochem. Int. 3, 165–171.
[2] Rapoport, S. and Dubiel, W. (1984) Biomed. Biochim. Acta 43, 23–27.
[3] Rapoport, S., Dubiel, W. and Müller, M. (1985) FEBS Lett. 180, 249–252.
[4] Dubiel, W., Müller, M. and Rapoport, S. (1986) FEBS Lett. 194, 50–55.
[5] Rapoport, S., Schmidt, J. and Prehn, S. (1985) FEBS Lett. 183, 370–374.
[6] Rapoport, S., Schmidt, J. and Prehn, S. (1986) FEBS Lett. 198, 109–112.
[7] Haas, A.L. and Bright, P.M. (1985) J. Biol. Chem. 260, 12464–12473.
[8] Hershko, A. and Ciechanover, A. (1982) Annu. Rev. Biochem. 51, 335–364.
[9] Goldberg, A.L. and St. John, A.C. (1976) Annu. Rev. Biochem. 45, 747–803.
[10] Ciechanover, A., Finley, D. and Varshavsky, A. (1984) Cell 37, 57–66.
[11] Hershko, A., Eyton, E., Ciechanover, A. and Haas, A.L. (1982) J. Biol. Chem. 257, 13964–13970.
[12] Hough, R. and Rechsteiner, M. (1984) Proc. Natl. Acad. Sci. USA 81, 90–94.
[13] Haas, A.L. and Bright, P.M. (1985) J. Biol. Chem. 260, 12464–12473.
[14] Haas, A.L., Murphy, K.E. and Bright, P.M. (1985) J. Biol. Chem. 260, 4694–4703.
[15] Vierstra, R.D., Langan, S.M. and Haas, A.L. (1985) J. Biol. Chem. 260, 12015–12021.
[16] Hough, R., Pratt, G. and Rechsteiner, M. (1986) J. Biol. Chem. 261, 2400–2408.