FEBS Letters 466 (2000) 394–395

Errata

FEBS 22705

Erratum to: **Stimulation of angiogenesis through cathepsin B inactivation of the tissue inhibitors of matrix metalloproteinases** (FEBS 22347)

[*FEBS Letters* 455 (1999) 286–290][1]

Georgios Kostoulas[a], Angela Lang[a], Hideaki Nagase[b], Antonio Baici[a,*]

[a] *University Hospital, Department of Rheumatology, CH-8091 Zurich, Switzerland*
[b] *Department of Biochemistry and Molecular Biology, University of Kansas Medical Center, Kansas City, KS 66160, USA*

Three errors in concentration units were made during the typesetting of the Materials and Methods section, *2.4. Degradation of TIMPs by cathepsin B*. In three places the character μ was wrongly converted to m, so that the section should have been printed as follows (the corrections have been made in bold):

2.4. Degradation of TIMPs by cathepsin B

The mature form of cathepsin B was affinity-purified from human kidney [18] and was free of other enzymes as ascertained by electrophoresis, specificity tests with substrates and inhibitors, and N-terminal sequencing. Cathepsin B (containing the pro-form and the single-chain form) was also prepared from conditioned media of dedifferentiated human articular chondrocytes by gel chromatography [12] followed by ion exchange chromatography on DEAE-Sephacel (Pharmacia). Human recombinant, non-glycosylated TIMP-1 whose Asn-30 and Asn-78 were mutated to Ala was expressed in CHO K1 cells and purified on MacroPrep 50Q anion exchange resin (Bio-Rad), Green A Dyematrex (Amicon) and Sephacryl S-200 (Pharmacia). Recombinant human, glycosylated TIMP-1 was expressed in CHO K1 cells and purified using anti-(human TIMP-1) affinity chromatography [19]. Human TIMP-2 was purified from the medium of uterine cervical fibroblasts [20]. The incubation solutions in 50 mM sodium acetate buffer, 2 mM dithiothreitol, 2 mM EDTA, pH 5.50, contained cathepsin B (5 **μg/ml** final concentration) and recombinant wild-type or non-glycosylated TIMP-1 (final concentrations in different experiments 100–160 μg/ml), or the same cathepsin B concentration and TIMP-2 (final concentrations in different experiments 25–57 **μg/ml**). Incubation was performed at 30°C, reaction was stopped at time intervals by making the solution 2 mM with iodoacetic acid, and the progressive disappearance of the original protein band followed by SDS-PAGE. After 5 h of incubation the original proteins were completely digested giving a mixture of peptides. Control experiments included the addition of the cathepsin B-specific inactivator CA-074 at a final concentration of 1 **μM**. MALDI-TOF spectra of TIMP-1 and TIMP-2 peptides were accumulated using a Voyager Elite instrumentation (PerSeptive Biosystems, Framingham, MA, USA) with α-cyano-4-hydroxy-cinnamic acid (5 mg/ml in 50% acetonitrile and 0.1% trifluoroacetic acid in water) as the matrix. Samples were analyzed in pulsed extraction reflectron mode using an acceleration voltage of 20 kV, a pulse delay time of 75 ns, a grid voltage of 55% and a guide voltage of 0.05%. Peptide sequences matching the determined molecular masses were calculated with the software PAWS (Protein Analysis WorkSheet) of ProteoMetrics (http://www.proteometrics.com).

*Corresponding author. Center of Experimental Rheumatology, University of Zurich, Winterthurerstrasse 190, CH-8057 Zurich, Switzerland. Fax: (41) (1) 635 6805. E-mail: baici@ruz.unizh.ch

[1] PII: S0014-5793(99)00897-2

FEBS 22936

Erratum to: **Sphingosylphosphorylcholine induces cytosolic Ca^{2+} elevation in endothelial cells in situ and causes endothelium-dependent relaxation through nitric oxide production in bovine coronary artery** (FEBS 22521)

[*FEBS Letters 457 (1999) 375–380*][1]

Kimiko Mogami[a], Yoichi Mizukami[a], Natsuko Todoroki-Ikeda[a], Masato Ohmura[a,b], Kazuki Yoshida[a], Saori Miwa[a], Masunori Matsuzaki[b], Masako Matsuda[c], Sei Kobayashi[a,*]

[a]*First Department of Physiology, School of Medicine, Yamaguchi University, 1-1-1 Minamikogushi, Ube 755-8505, Japan*
[b]*Second Department of Medicine, School of Medicine, Yamaguchi University, 1-1-1 Minamikogushi, Ube 755-8505, Japan*
[c]*School of Allied Health Sciences, Yamaguchi University, 1-1-1 Minamikogushi, Ube 755-8505, Japan*

The correct affiliations of Sei Kobayashi and Masato Ohmura is given above.

*Corresponding author. Fax: (81) (836) 22-2348. E-mail: seikoba@po.cc.yamaguchi-u.ac.jp

[1] PII: S0014-5793(99)01076-5

Febs 23082

Erratum to: **Hormone-activated nuclear receptors inhibit the stimulation of the JNK and ERK signalling pathways in endothelial cells** (FEBS 22697)

[*FEBS Letters 459 (1999) 272–276*][1]

María Victoria González[a], José Manuel González-Sancho[a], Carme Caelles[b], Alberto Munoz[a,*], Benilde Jiménez[a]

[a]*Instituto de Investigaciones Biomédicas 'Alberto Sols', Consejo Superior de Investigaciones Científicas, Universidad Autónoma de Madrid, Arturo Duperier 4, E-28029 Madrid, Spain*
[b]*Facultad de Farmacia, Universidad de Barcelona, E-08028 Barcelona, Spain*

In the original publication of this paper, the first three figures were printed in the wrong positions. Whilst the legends were correct, Fig. 1 was printed as Fig. 2, Fig. 2 was printed as Fig. 3 and Fig. 3 was printed as Fig. 1. The Publisher apologises for this mistake to author and readers alike.

*Corresponding author. Fax: (34)-91-5854587. E-mail: amunoz@biomed.iib.uam.es

[1] PII: S0014-5793(99)01257-0