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

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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, nonglycosylated 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).

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FEBS 22936

Erratum to: Sphingosylphosphorylcholine induces cytosolic Ca²⁺ 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

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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

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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.

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