



Erratum

Pre-B cell colony-enhancing factor inhibits neutrophil apoptosis in experimental inflammation and clinical sepsis

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During the preparation of this manuscript for publication, errors were introduced into the panel labels of Figure 6. The correct figure appears below. We regret this error.

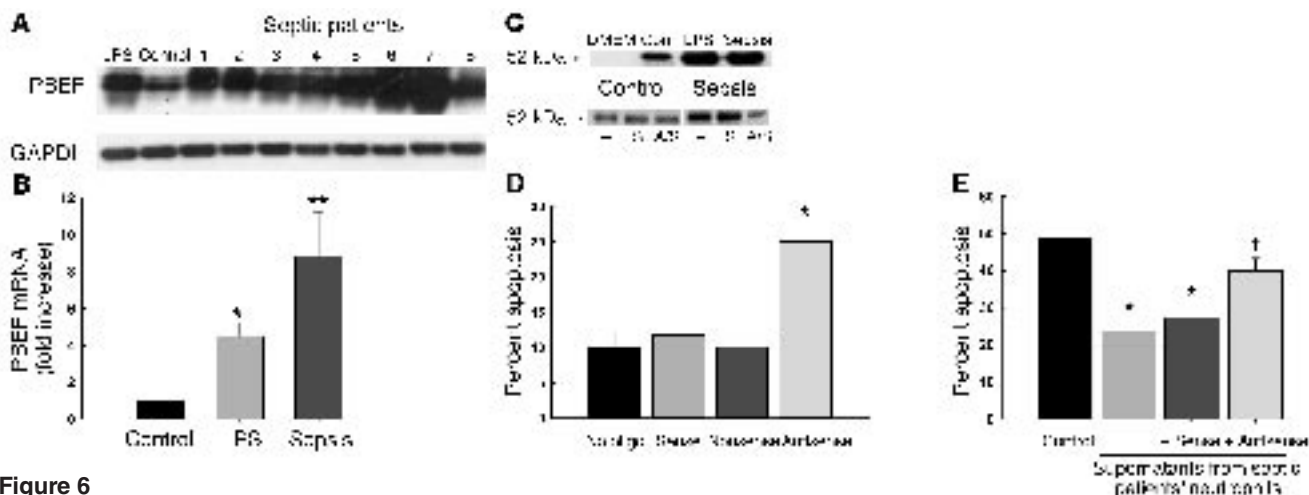


Figure 6 PBEF is expressed and is biologically active in neutrophils harvested from critically ill septic patients. (A) PBEF mRNA in neutrophils from eight critically ill septic patients was expressed at higher levels than in control (Con) or LPS-stimulated neutrophils. Blots were reprobed with GAPDH to confirm comparability of loading. (B) Expression of PBEF mRNA transcripts in septic and LPS-treated neutrophils was evaluated by real-time PCR, normalizing expression to that for GAPDH. Expression was induced by LPS (*P < 0.05 versus unstimulated cells) and even more in septic neutrophils (**P < 0.05 versus both LPS-stimulated cells and unstimulated cells). (C) Immunoreactive PBEF was detectable by Western blot in supernatants from LPS-treated and septic neutrophils following 21 hours of in vitro culture in serum-free medium; antisense pretreatment blocked the secretion of PBEF. DMEM denotes medium only; studies were repeated three times, and a representative blot is shown. S, sense; A/S, antisense. (D) Neutrophils from 16 septic critically ill patients were incubated for 5 hours with PBEF antisense or the sense or nonsense controls, and apoptosis was evaluated 21 hours later. Antisense treated cells, but not controls, showed increased rates of apoptosis (*P = 0.002 versus no oligonucleotide [no oligo]; ANOVA). (E) Supernatants from control PMN had minimal effects on the apoptosis of resting PMN (black bar). In contrast, supernatants from septic PMN or septic PMN incubated with PBEF sense oligonucleotides significantly inhibited the apoptosis of control PMN (*P < 0.05), whereas supernatants from antisense-treated septic PMNs induced significantly less inhibition (†P < 0.05 versus sense or no oligonucleotide; P = NS versus controls, n = 5).

Erratum

In This Issue

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In the June 1, 2004, “In this Issue” section, a figure was inadvertently included with the paragraph titled “Thyrotropin receptor can be so insensitive”, regarding the article by Takao Ando and colleagues. This figure should have been included with the paragraph titled “RAGE against an immune response”, regarding the paper by Peter Nawroth and colleagues. We apologize for any confusion this may have caused. The online version of this section has been corrected.



Corrigendum

Cathepsin B inactivation attenuates hepatic injury and fibrosis during cholestasis

Ali Canbay, Maria Eugenia Guicciardi, Hajime Higuchi, Ariel Feldstein, Steven F. Bronk, Robert Rydzewski, Makiko Tanai, and Gregory J. Gores

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The authors wish to correct errors and provide further clarification regarding this manuscript. Figure 2a was reproduced from a previous publication (Canbay et al. *Gastroenterology*. **123**:1323–1330 [2002]). An appropriate photomicrograph from concurrent control mice is depicted below and should replace the original.

In addition, although wild-type and cathepsin B^{-/-} mice originated from the same litter, they were subsequently bred as homozygotes for this study and therefore were not concurrent littermates as was indicated in the study.

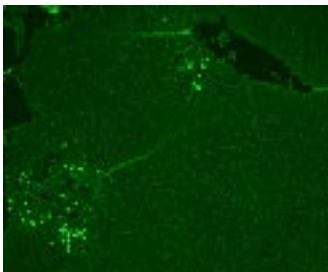


Figure 2a
Photomicrograph of TUNEL assay in three-day bile duct ligated wild-type mice.

Corrigendum

Hepatic expansion of a virus-specific regulatory CD8⁺ T cell population in chronic hepatitis C virus infection

Daniele Accapezzato, Vittorio Francavilla, Marino Paroli, Marco Casciaro, Lucia Valeria Chircu, Agostino Cividini, Sergio Abrignani, Mario U. Mondelli, and Vincenzo Barnaba

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Figure 7 contains an error generated during the revision of this manuscript for publication. The original values in the column “CD8⁺ LILs α -perf.” were incorrectly reported. The correct version appears below.

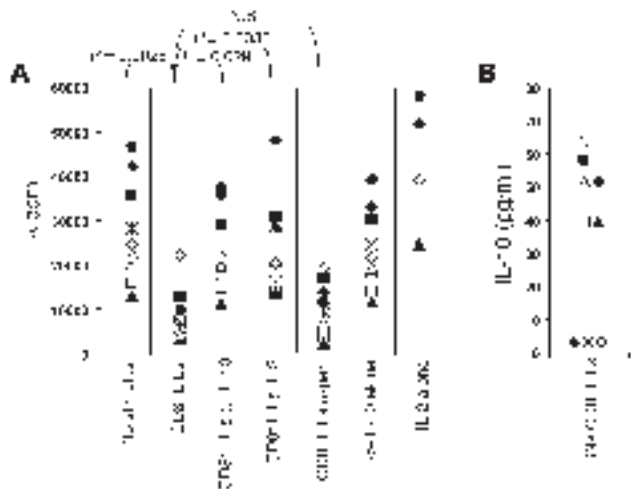


Figure 7
Hepatic IL-10-producing CD8⁺ T cells perform a regulatory function. (A) Highly purified CD8⁺ LILs pooled from two to three biopsies were cocultured with PBMCs plus soluble mAb's to CD3 and CD28 in the presence or absence of anti-IL-10 (α -IL-10), mAb to perforin (α -perf.), or IL-2. Control cocultures were prepared in the presence of either IgG1 or IgG2b isotype, which did not produce any interference with suppressive function (data not shown). Control cultures in which PBMCs were stimulated with mAb's to CD3 and CD28 in the presence of IL-2 or anti-IL-10 but in the absence of CD8⁺ LILs were also prepared. Each symbol represents an individual experiment performed with single LIL pool derived from two biopsies. In all experiments, 1 μ Ci [³H]thymidine was added to the cultures after 6 days, and the radioactivity incorporated by cells was determined after 18 hours. The cpm values were calculated after subtraction of background (Δ cpm). Statistical analysis was performed by Student's *t* test for paired data. (B) The production of IL-10 was determined in the supernatant pool (not conditioned with mAb to IL-10) from cocultures indicated by symbols.