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CORRECTIONS

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CORRECTIONS

Miguel Aste-Amezaga, Xiaojing Ma, Alexandrina Sartori, and Giorgio Trinchieri. Molecular Mechanisms of the Induction of IL-12 and Its Inhibition by IL-10. *The Journal of Immunology* 1998;160:5936–5944.

In the top half of Fig. 2*A*, the authors inadvertently reproduced the same experiment depicted in a previous publication by their group. The data in that panel were controls for the data presented in Fig. 2*B*, and the original source of these data (*J. Exp. Med. 178:1041*) was clearly referred to in the *Results* section. The time course indicated in Fig. 2*A* refers to the p35 date (*bottom half*); the correct time course for the p40 data is 1, 4, 5, and 15 h.

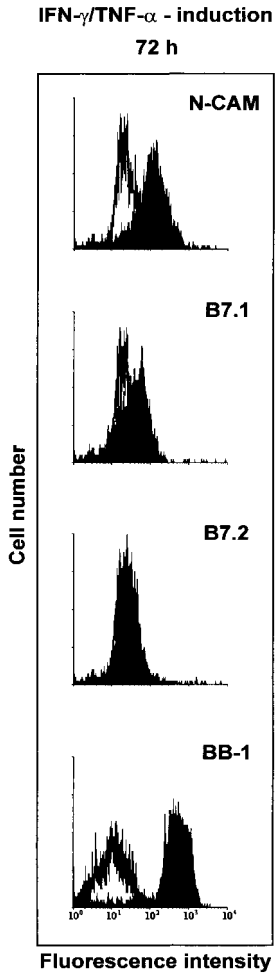
Yuichi Endo, Minoru Takahashi, Miki Nakao, Hidetoshi Saiga, Hideharu Sekine, Misao Matsushita, Masaru Nonaka, and Teizo Fujita. Two Lineages of Mannose-Binding Lectin-Associated Serine Protease (MASP) in Vertebrates. *The Journal of Immunology* 1998;161:4924–4930.

There were errors in the length and restriction map of the region between exons II and III of the human MASP-1 gene in Fig. 3*A*. The correct figure is shown below.

Lüder Behrens, Martin Kerschensteiner, Thomas Misgeld, Norbert Goebels, Hartmut Wekerle, and Reinhard Hohlfeld. Human Muscle Cells Express a Functional Costimulatory Molecule Distinct from B7.1 (CD80) and B7.2 (CD86) In Vitro and in Inflammatory Lesions. *The Journal of Immunology* 1998;161:5943–5951.

The figures representing the FACS data contain several inadvertent exchanges of FACS panels. A new figure (presented on the following page) was reconstructed from the original data. This figure demonstrates that the myoblasts derived from the original donor expressed BB-1, but not B7.1 or B7.2. Functional experiments (T cell proliferation tests) supported the FACS results. As most of the FACS experiments reported in the paper were based on myoblast lines from a single donor, we subsequently screened a large panel of human muscle-derived cell lines. In these experiments, only a minority of cultures inconstantly expressed BB-1, indicating phenotypic heterogeneity of myoblasts or a genetic polymorphism. In contrast, as shown in our paper and recently by another group (Murata, K., and M. C. Dalakas. 1999. Expression of the costimulatory molecule BB-1, the ligands CTLA-4 and CD28, and their mRNA in inflammatory myopathies. *Am. J. Pathol. 155:453*) most, if not all, muscle specimens from patients with inflammatory myopathy contain muscle fibers which express BB-1 in the absence of B7.1 and B7.2.

 0022-1767/00/$02.00



Selective induction of BB-1 on human myoblasts treated with IFN-γ and TNF-α for 72 h. Purity of the myoblasts is documented by NCAM expression (*upper panel*). The myoblasts uniformly express BB-1 but are negative for B7.1 and B7.2. Open curves represent appropriate isotype control.

Philip M. Wallace, John F. MacMaster, Katherine A. Rouleau, T. Joseph Brown, James K. Loy, Karen L. Donaldson, and Alan F. Wahl. Regulation of Inflammatory Responses by Oncostatin M. *The Journal of Immunology* 1999;162: 5547–5555.

Figs. 3 and 4 of this article are reversed. The filled and open circles within the legends of Figs. 3 and 4 denoting control and OM-treated animals, are also reversed.

Martin K. Wild, Wolfgang Strittmatter, Siegfried Matzku, Burkhart Schraven, and Stefan C. Meuer. Tumor Therapy with Bispecific Antibody: The Targeting and Triggering Steps Can Be Separated Employing a CD2-Based Strategy. *The Journal of Immunology* 1999;163:2064–2072.

The concentrations in Figs. 7 and 8 are incorrect and should be given in μg/ml.

Mads Hald Andersen, Jordi Espuny Bonfill, Anne Neisig, Gemma Arsequell, Ib Søndergaard, Jacques Neefjes, Jesper Zeuthen, Tim Elliott, and John S. Haurum. Phosphorylated Peptides Can Be Transported by TAP Molecules, Presented by Class I MHC Molecules, and Recognized by Phosphopeptide-Specific CTL. *The Journal of Immunology* 1999;163: 3812–3818.

In the original article, one name was omitted from the author list. Gregorio Valencia should be the sixth author.

Yi Luo, Clare Lloyd, Jose-Carlos Gutierrez-Ramos, and Martin E. Dorf. Chemokine Amplification in Mesangial Cells. *The Journal of Immunology* 1999;163:3985–3992.

A breeding error was found in the colony of CXCR2-deficient mice. Mesangial cells derived from mice of this colony were used to generate the data presented in Table II and Fig. 8*B*. All other results were reported accurately.

Jingwu Xu, Ali Ahmad, Mario D'Addario, Laurent Knafo, James F. Jones, U. Prasad, R. Dolcetti, E. Vaccher, and José Menezes. Analysis and Significance of Anti-Latent Membrane Protein-1 Antibodies in the Sera of Patients with EBV-Associated Diseases. *The Journal of Immunology* 2000;164:2815–2822.

The first line in the Abstract should read: "The latent membrane protein-1 (LMP-1) is an EBV-encoded type III integral membrane protein with oncogenic potential that is expressed most consistently in various EBV-associated malignancies."

Ivan Babic, Annette Schallhorn, Frederik P. Lindberg, and Frank R. Jirik. SHPS-1 Induces Aggregation of Ba/F3 Pro-B Cells Via an Interaction with CD47. *The Journal of Immunology* 2000;164:3652–3658.

Fig. 3*A* should be deleted, and the letter "*B*" should also be deleted from the figure.

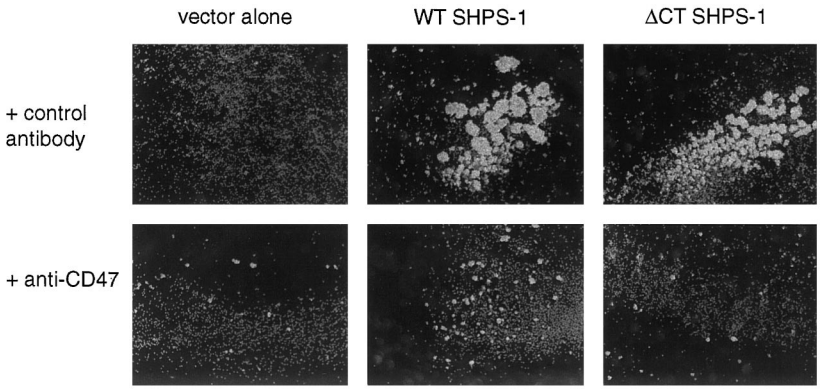


FIGURE 3. Anti-CD47 Ab (mIAP301) inhibits the aggregation of Ba/F3 cells expressing WT SHPS-1 and ΔCT SHPS-1. Anti-CD47 or an isotype control Ab was added at 50 μg/ml to a volume of 200 μl that contained 1×10^6 Ba/F3 cells transfected with either vector alone, SHPS-1, or ΔCT SHPS-1. Photographs were taken after incubation for 16 h.

Grant R. Stenton, Moo-Kyung Kim, Osamu Nohara, Chin-Fu Chen, Nadir Hirji, Fiona L. Wills, Mark Gilchrist, Pyoung-Han Hwang, Jong-Gu Park, Warren Finlay, Richard L. Jones, A. Dean Befus, and Alan D. Schreiber. Aerosolized Syk Antisense Suppresses Syk Expression, Mediator Release from Macrophages, and Pulmonary Inflammation. *The Journal of Immunology* 2000;164:3790–3797.

In the original footnote 1, a grant source was omitted. Footnote 1 should read: "This work was supported by the Alberta Lung Association, the Medical Research Council of Canada (A.D.B.), the National Institutes of Health (Grants HL-27068 and AI/HL-22193 to A.D.S.), and a Postdoctoral Fellowship from the Canadian Lung Association/Medical Research Council of Canada/Glaxo Wellcome (to G.R.S.)."
