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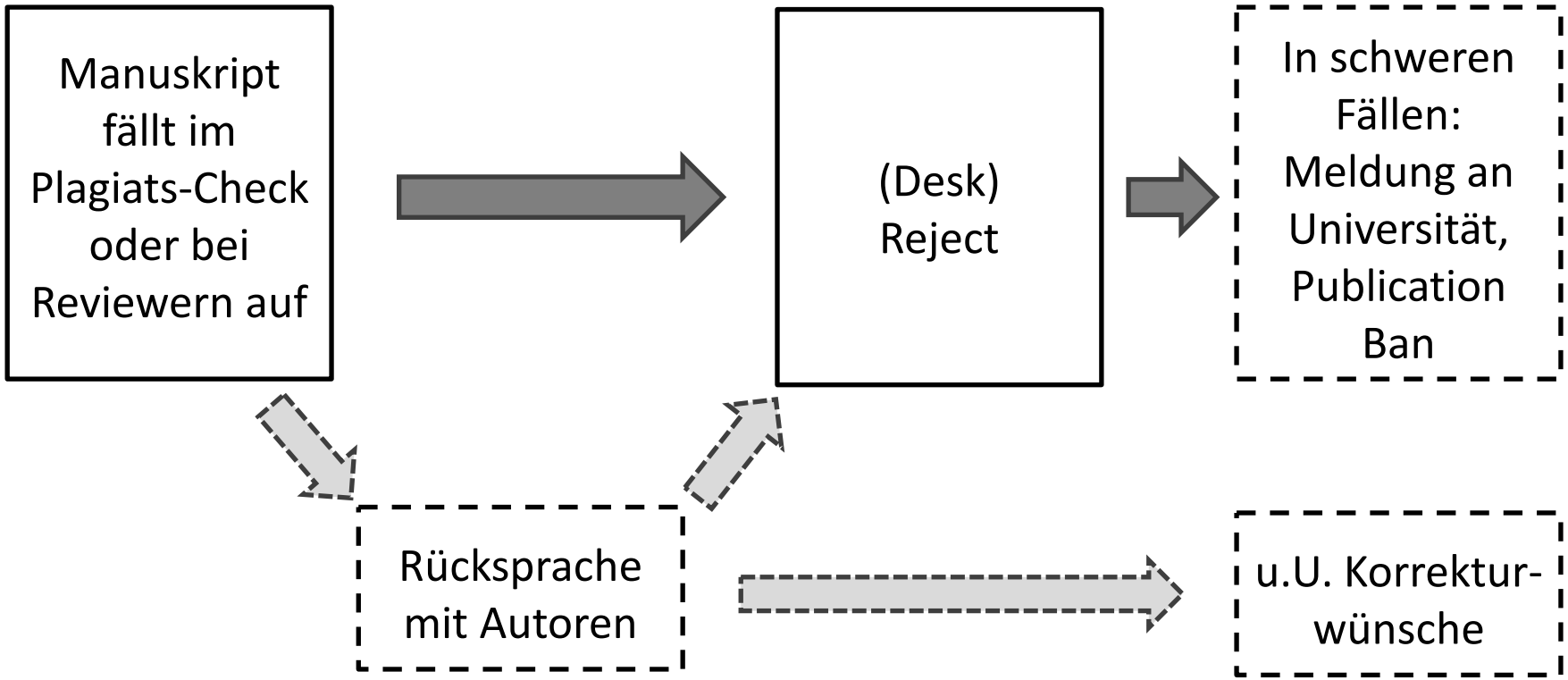
Felicitas Heßelmann (Deutsches Zentrum für Hochschul- und Wissenschaftsforschung)

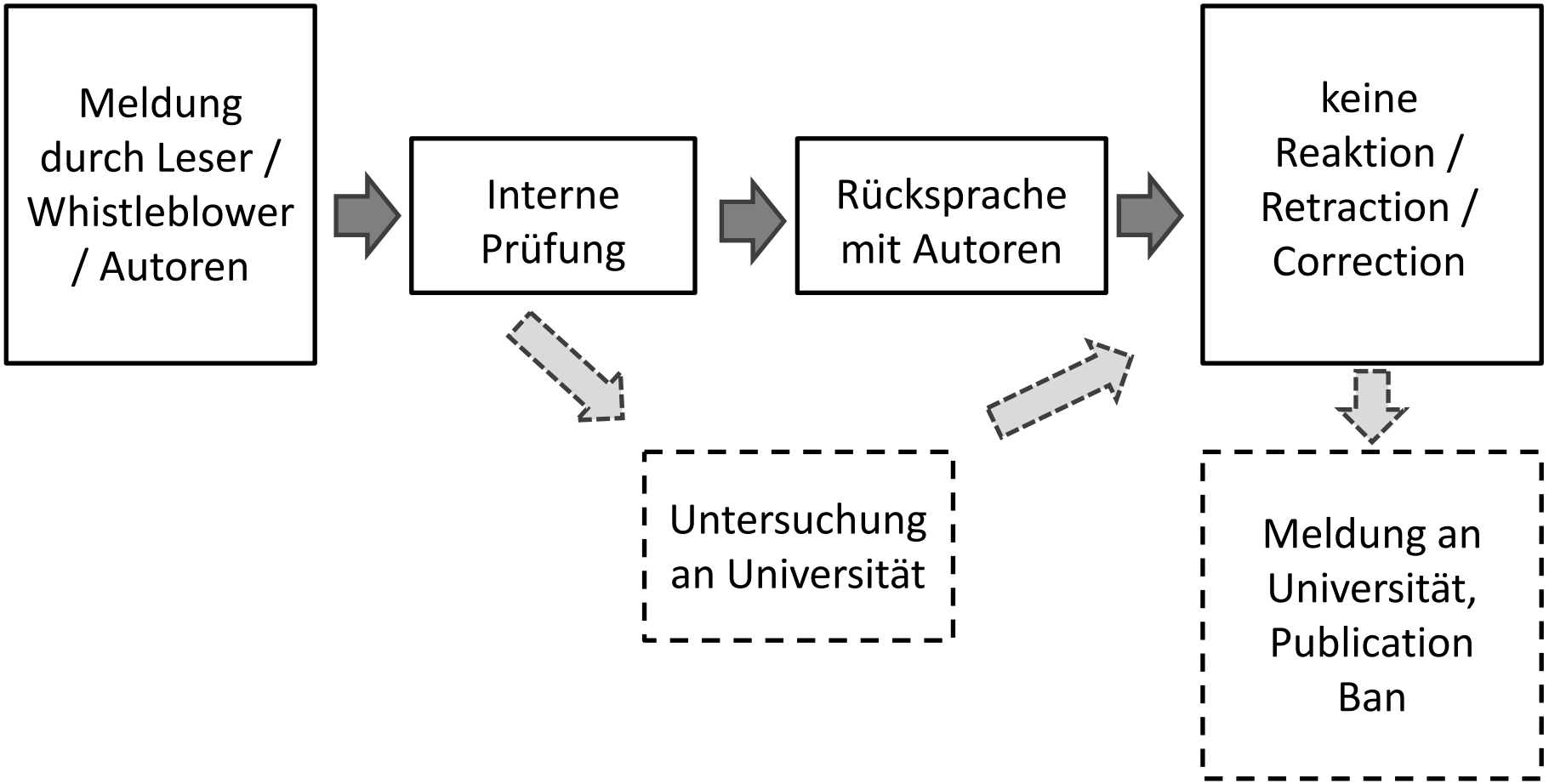
Tag der guten Wissenschaftlichen Praxis, Freie Universität Berlin, 14.06.17

1. Prozesse bei Zeitschriften
    - im Peer Review
    - in publizierten Artikeln
    - Retractions
2. Prozesse bei Universitäten
3. Weitere Ansprechpartner und Informationsangebote

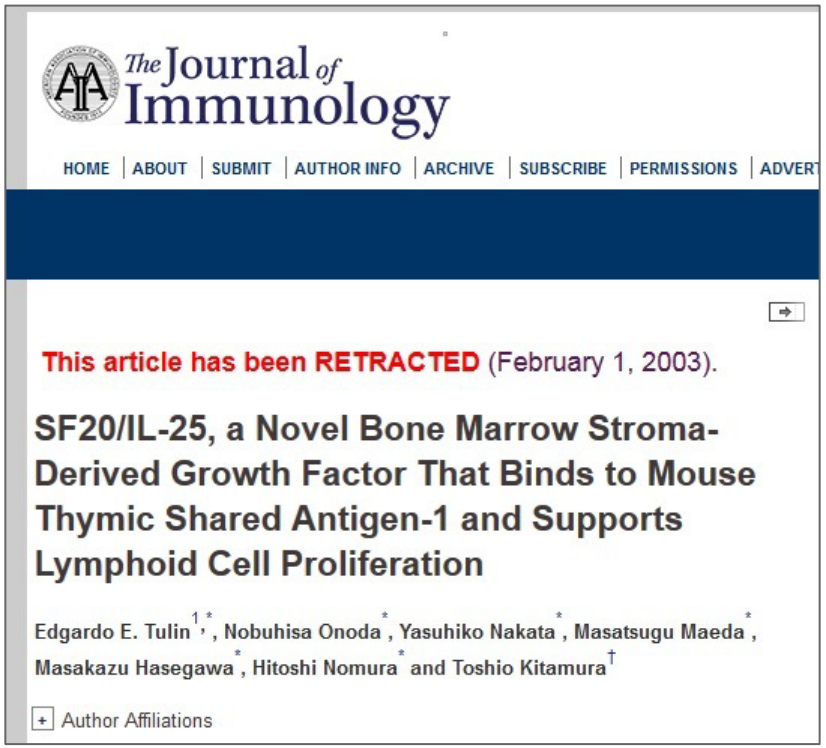
1a Verdachtsfälle im Peer-Review



1b Verdachtsfälle in Publikationen



1c Retractions



The Journal of Immunology

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This article has been RETRACTED (February 1, 2003).

SF20/IL-25, a Novel Bone Marrow Stroma-Derived Growth Factor That Binds to Mouse Thymic Shared Antigen-1 and Supports Lymphoid Cell Proliferation

Edgardo E. Tulin[1,*], Nobuhisa Onoda[*], Yasuhiko Nakata[*], Masatsugu Maeda[*], Masakazu Hasegawa[*], Hitoshi Nomura[*] and Toshio Kitamura[†]

Author Affiliations

SF20/IL-25, a Novel Bone Marrow Stroma-Derived Growth Factor That Binds to Mouse Thymic Shared Antigen-1 and Supports Lymphoid Cell Proliferation

Edgardo E. Tulin,[1,*] Nobuhisa Onoda,[*] Yasuhiko Nakata,[*] Masatsugu Maeda,[*] Masakazu Hasegawa,[*] Hitoshi Nomura,[*] and Toshio Kitamura[†]

Using a forward genetic approach and phenotype-based complementation screening to search for factors that stimulate cell proliferation, we have isolated a novel secreted bone marrow stroma-derived growth factor, which we termed SF20/IL-25. This protein signals cells to proliferate via its receptor, which we have identified as mouse thymic shared Ag-1 (TSA-1). Enforced expression of TSA-1 in IL-3-dependent Ba/F3 cells that do not express endogenous TSA-1 rendered cells to proliferate in a dose-dependent manner when stimulated with SF20/IL-25. FDCP2, a factor-dependent hemopoietic cell line that expresses endogenous TSA-1, could also be stimulated to proliferate with SF20/IL-25. Binding of SF20 to TSA-1 was blocked by anti-TSA-1 Ab and SF20-induced proliferation of TSA-1-expressing cells was inhibited by anti-TSA-1. In vitro assay revealed that SF20/IL-25 has no detectable myelopoietic activity but supports proliferation of cells in the lymphoid lineage. *The Journal of Immunology*, 2001, 167: 6338–6347.

RETRACTED February 2003

Thymic shared Ag-1 (TSA-1),[2] also called stem cell Ag-2, is a small Cys-rich cell surface protein and is a member of the Ly-6 family of hemopoietic proteins (1). It is anchored in the cell membrane by a C-terminal GPI moiety, a posttranslational modification common to each member of the Ly-6 family of proteins. Although the biologic roles for these molecules are not well understood, there is mounting evidence that Ly-6 proteins participate in intracellular adhesion and cell signaling (2–5). Lymphoid tissue distribution of mouse TSA-1 revealed that, in addition to expression in bone marrow cells, TSA-1 is also present on the developing thymocytes. Activation of TSA-1 expression in a lymphoid precursor cell line [illegible] an important function of TSA-1 in thymopoiesis.

Studies on the functional characterization of TSA-1 largely restricted to T cell activation, developed so far have been conducted using anti-TSA-1 mAbs. In the mouse, anti-TSA-1 Abs have been reported to inhibit T cell activation (9, 10). In fact, it was reported that the addition of anti-TSA-1 mAb to fetal thymus organ culture inhibited the development of double positive ($CD4^+CD8^+$) thymocytes and $TCR\alpha\beta^+$ mature thymocytes (2). No natural ligand has yet been reported for TSA-1. Using a forward genetic approach and phenotype-based complementation screening to search for stromal cell-derived factors that support cell proliferation (11), we have identified a novel secreted bone marrow stroma-derived growth factor, which we termed SF20/IL-25, that binds to mouse TSA-1 and stimulates cell proliferation. In this work, we report on the expression cloning of SF20/IL-25 and the subsequent identification of murine TSA-1 as its receptor, and show that this ligand-receptor interaction functions in [illegible] lymphoid cell proliferation. Although it has been shown that TSA-1 plays a role in T cell development in the thymus, it is unclear how TSA-1 functions in lymphocyte development. Identification of SF20/IL-25 as a ligand of mouse TSA-1 will provide a better understanding of the physiologic roles of these proteins in the regulation of lymphocyte [illegible].

Materials and Methods

Cytokines and cell lines

Recombinant murine (m)IL-3 was purchased from Upstate Biotechnology (Lake Placid, NY) and rmIL-2 was from R&D Systems (Minneapolis, MN). Anti-FLAG BioM2 Ab was purchased from Sigma-Aldrich (St. Louis, MO). Anti-TSA-1 monoclonal Ab (MTS35) was purchased from BD PharMingen (San Diego, CA). A retrovirus packaging cell line, Plat-E (12), was maintained in DMEM containing 10% (v/v) FCS and selection reagents (8 μg/ml blasticidin and 0.8 μg/ml puromycin; Sigma-Aldrich). The cells were transferred into DMEM/10% FCS without selection reagents 2 days before transfection. A murine pro-B cell line, BaF3, was cultured in RPMI 1640 medium containing 10% FCS in the presence of 1 ng/ml IL-3. A murine factor-dependent cell line, FDCP2, and mast cell line, MC9, were cultured in RPMI 1640 medium containing 10% FCS and 10 ng/ml IL-3. A murine IL-2-dependent T cell line, CTLL-2, was cultured in RPMI 1640 medium containing 10% FCS and 5 ng/ml IL-2. The mouse bone marrow-derived stromal cell lines, ST2, ST0, and PA6, and spleen-derived stroma CF-1 were cultured in DMEM/F-12 medium containing 10% FCS. The mouse bone marrow-derived stroma cell lines MS5 and MS10 were cultured in α-MEM (Life Technologies, Rockville, MD) containing 10% FCS. COS7 cells were maintained in DMEM/10% FCS.

Expression cloning of SF20

Ba/F3 mutagenesis, establishment of ST2 stroma-dependent mutants, and preparation of cDNA library from ST2 cells were performed as previously described (11). Production of retrovirus stocks from the cDNA library and infection of MS10 cells, a bone marrow stroma that does not support proliferation of SB2-33 mutants, were essentially the same as previously reported. To search for the factor that stimulates proliferation of SB2-33 cells, 12,000 independent clones from the ST2 cell cDNA library were screened using subdivided pools (120 clones per pool). After first screening, one pool (no. 6) was identified to support proliferation of SB2-33 cells. This pool contained 120 independent clones and was further subdivided

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[2] Abbreviations used in this paper: TSA-1, thymic shared Ag-1; m, murine; EST, expressed sequence tag; MTC, multiple tissue cDNA.

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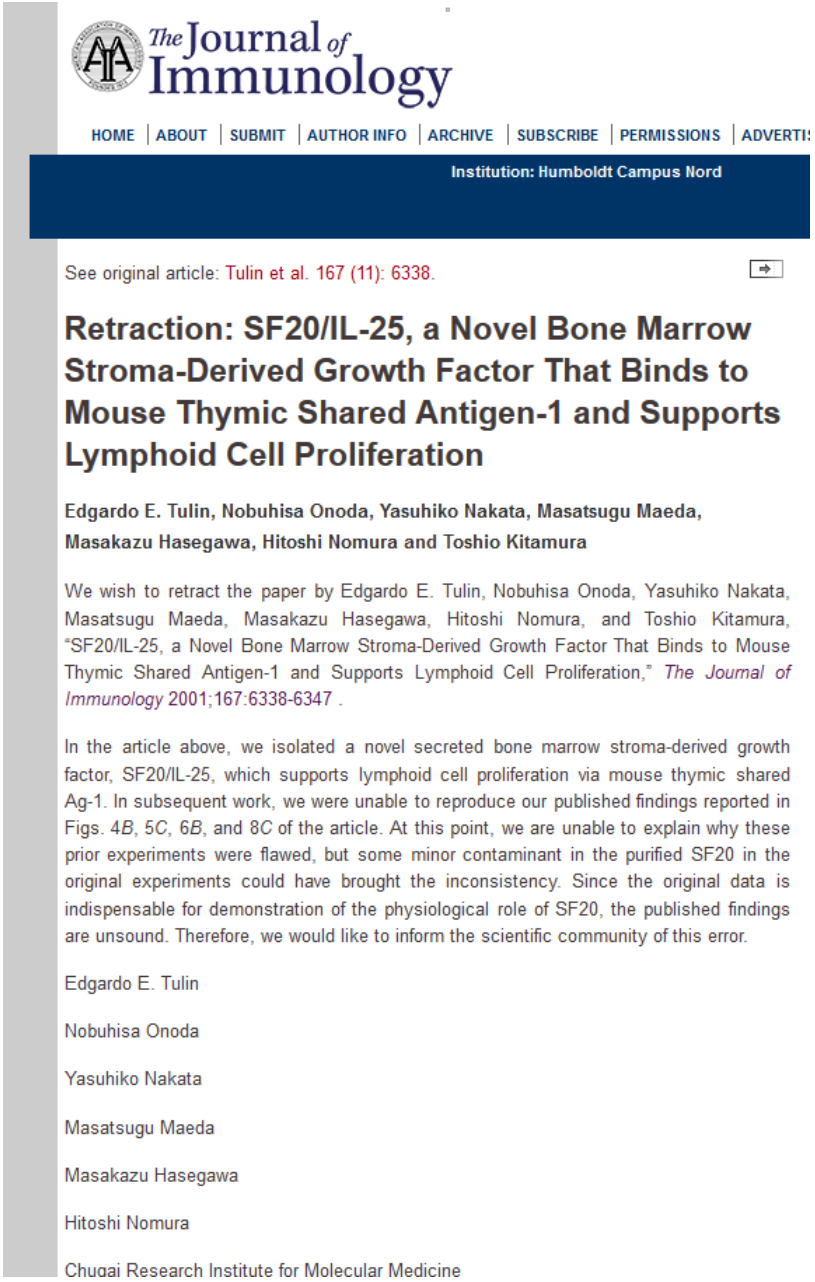

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Institution: Humboldt Campus Nord

See original article: Tulin et al. 167 (11): 6338.

Retraction: SF20/IL-25, a Novel Bone Marrow Stroma-Derived Growth Factor That Binds to Mouse Thymic Shared Antigen-1 and Supports Lymphoid Cell Proliferation

Edgardo E. Tulin, Nobuhisa Onoda, Yasuhiko Nakata, Masatsugu Maeda, Masakazu Hasegawa, Hitoshi Nomura and Toshio Kitamura

We wish to retract the paper by Edgardo E. Tulin, Nobuhisa Onoda, Yasuhiko Nakata, Masatsugu Maeda, Masakazu Hasegawa, Hitoshi Nomura, and Toshio Kitamura, "SF20/IL-25, a Novel Bone Marrow Stroma-Derived Growth Factor That Binds to Mouse Thymic Shared Antigen-1 and Supports Lymphoid Cell Proliferation," *The Journal of Immunology* 2001;167:6338-6347 .

In the article above, we isolated a novel secreted bone marrow stroma-derived growth factor, SF20/IL-25, which supports lymphoid cell proliferation via mouse thymic shared Ag-1. In subsequent work, we were unable to reproduce our published findings reported in Figs. 4*B*, 5*C*, 6*B*, and 8*C* of the article. At this point, we are unable to explain why these prior experiments were flawed, but some minor contaminant in the purified SF20 in the original experiments could have brought the inconsistency. Since the original data is indispensable for demonstration of the physiological role of SF20, the published findings are unsound. Therefore, we would like to inform the scientific community of this error.

Edgardo E. Tulin

Nobuhisa Onoda

Yasuhiko Nakata

Masatsugu Maeda

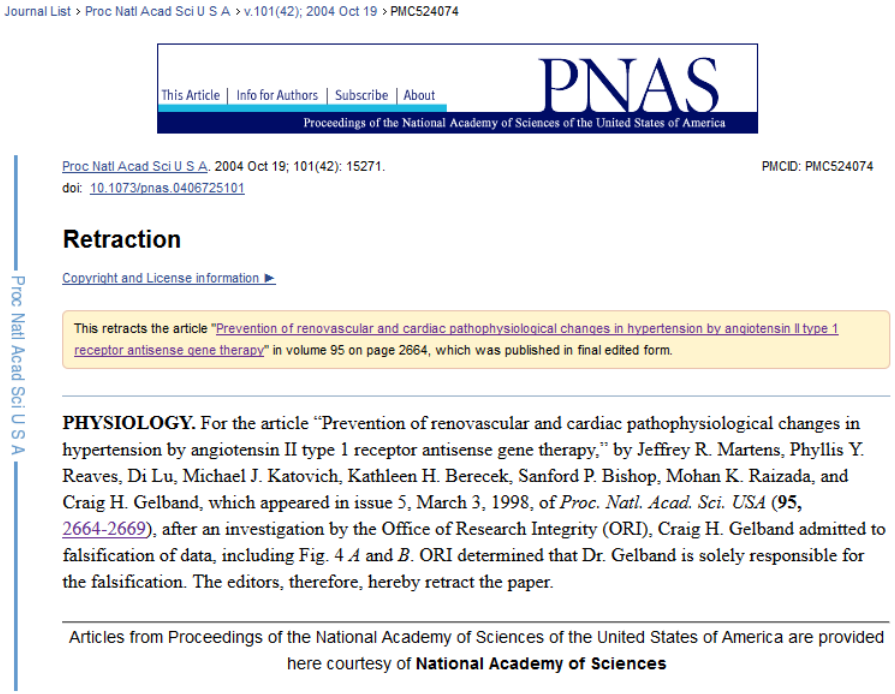
Masakazu Hasegawa

Hitoshi Nomura

Chugai Research Institute for Molecular Medicine

"**We wish to retract the paper** […]

In the article above, we isolated a novel secreted bone marrow stroma-derived growth factor, SF20/IL-25, which supports lymphoid cell proliferation via mouse thymic shared Ag-1. **In subsequent work, we were unable to reproduce our published findings** reported in Figs. 4*B*, 5*C*, 6*B*, and 8*C* of the article. At this point, we are unable to explain why these prior experiments were flawed, **but some minor contaminant in the purified SF20 in the original experiments could have brought the inconsistency.** Since the original data is indispensable for demonstration of the physiological role of SF20, the published findings are unsound. **Therefore, we would like to inform the scientific community of this error.**"



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Proc Natl Acad Sci U S A. 2004 Oct 19; 101(42): 15271.
doi: 10.1073/pnas.0406725101

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Retraction

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This retracts the article "Prevention of renovascular and cardiac pathophysiological changes in hypertension by angiotensin II type 1 receptor antisense gene therapy" in volume 95 on page 2664, which was published in final edited form.

PHYSIOLOGY. For the article "Prevention of renovascular and cardiac pathophysiological changes in hypertension by angiotensin II type 1 receptor antisense gene therapy," by Jeffrey R. Martens, Phyllis Y. Reaves, Di Lu, Michael J. Katovich, Kathleen H. Berecek, Sanford P. Bishop, Mohan K. Raizada, and Craig H. Gelband, which appeared in issue 5, March 3, 1998, of *Proc. Natl. Acad. Sci. USA* (**95,** 2664-2669), after an investigation by the Office of Research Integrity (ORI), Craig H. Gelband admitted to falsification of data, including Fig. 4 *A* and *B*. ORI determined that Dr. Gelband is solely responsible for the falsification. The editors, therefore, hereby retract the paper.

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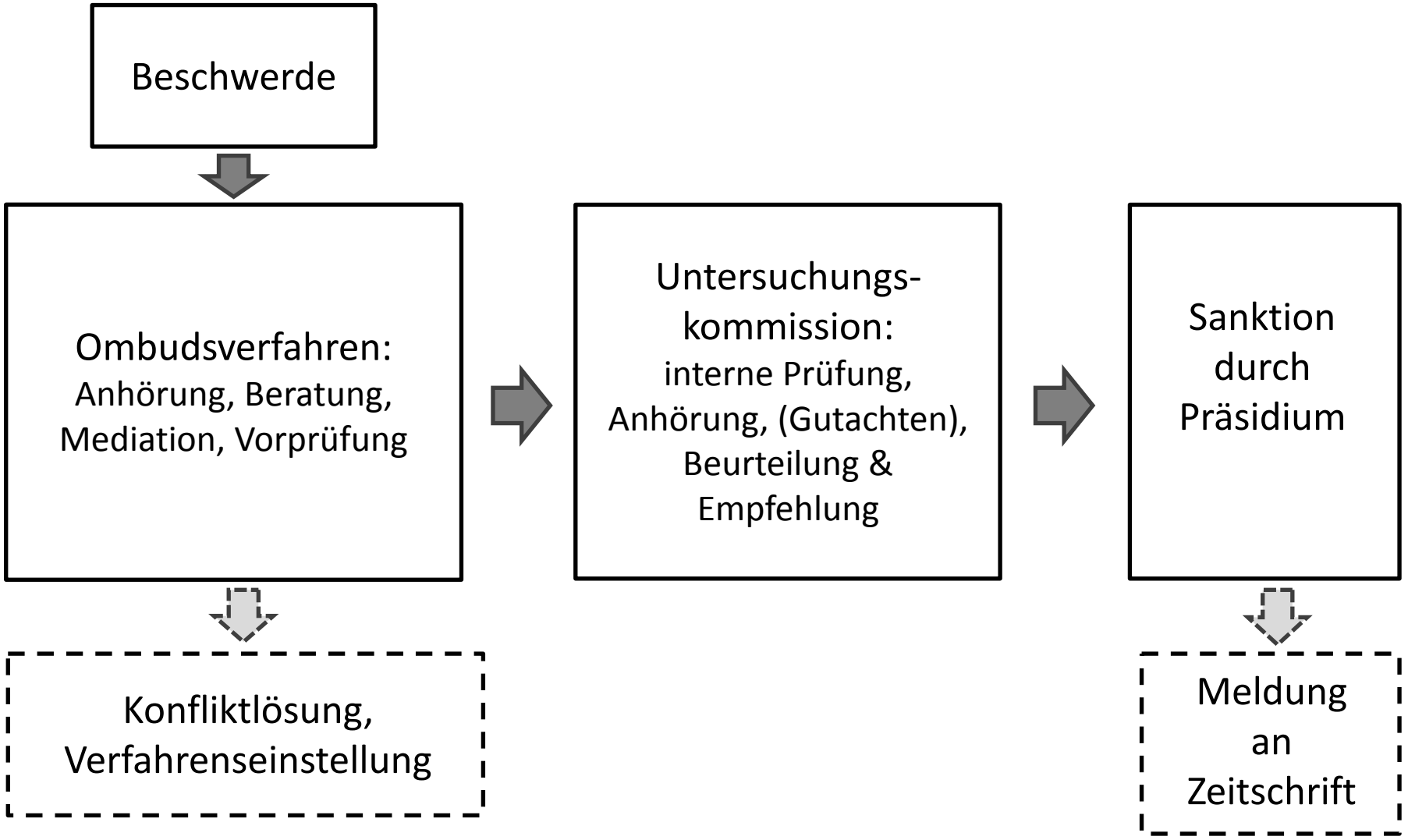
Proc Natl Acad Sci U S A

"For the article […] **after an investigation by the Office of Research Integrity (ORI), Craig H. Gelband admitted to falsification of data,** including Fig. 4 A and B. **ORI determined that Dr. Gelband is solely responsible for the falsification. The editors, therefore, hereby retract the paper.**"

Proceedings of the National Academy of Sciences of the United States of America. Retraction, 2004; 101(42):15271. doi:10.1073/pnas.0406725101.

2. Verdachtsfälle in Universitäten



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http://www.fu-berlin.de/forschung/service/foerderung/dokumentensammlung/ombud/index.html

(Vertrauenspersonen der Fachbereiche, Ehrenkodex der FU)

Ombudsman für die Wissenschaft

Prof. Dr. Stephan Rixen (Sprecher)

E-Mail: geschaeftsstelle(at)ombuds-wissenschaft.de

http://www.ombudsman-fuer-die-wissenschaft.de/

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Projekt „Refairenz“ (Uni Konstanz)

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(Materialien zum Thema Plagiat)

Vielen Dank!

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