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Yes. Elsevier has a longstanding policy of helping authors comply with the NIH public access policy. In May 2005 Elsevier began depositing author manuscripts to PubMed Central (PMC) on behalf of authors who were reporting NIH funded research. Today, we deposit over 1,000 manuscripts a month to PubMed Central.

Q2. I have seen a list of journals on PubMed Central – do I have to publish in these titles to comply with the NIH policy?

No. The NIH maintains a list of journals on PubMed Central that have an agreement for entire journals to appear on PubMed Central. This list should not be mistaken as a list of journals where authors must publish to comply with the NIH policy. We have requested that the NIH clarify this point, as it has resulted in considerable confusion for authors and editors.

Q3. What do you deposit to PubMed Central, what happens after you deposit, and when will the manuscript appear on PubMed Central?

Elsevier has agreed with NIH that it will deposit on behalf of NIH authors the final peer-reviewed manuscript files in the NIH Manuscript Submission system. (see C.1.II.c of the NIH FAQ document available at <http://publicaccess.nih.gov/faq.htm>). Authors will receive an email message from PMC, requesting them to verify and approve the final peer-reviewed manuscript personally. Elsevier currently deposits the manuscript at the time of publication, and PMC will make it publicly available after 12 months.

Q4. The policy requires that the manuscript be deposited upon acceptance. Does Elsevier provide this service?

Elsevier is revising our production system so that as of July we will be depositing the author manuscript upon manuscript acceptance. We have been in consultation with the NIH regarding this change and they have confirmed that this will be consistent with the NIH public access policy. Authors, whose manuscripts are accepted in May or June 2008 and wish to deposit their manuscript upon acceptance to PubMed Central, may do so. Elsevier will subsequently redeposit the manuscript upon publication.

Q5. How will I get the PMC-ID number?

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1: Am J Pathol, 2007 Sep;171(3):928-37. Epub 2007 Aug 9.

Neutrophil elastase converts human immature dendritic cells into transforming growth factor-beta1-secreting cells and reduces allostimulatory ability.

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During microbial infection, neutrophils (polymorphonuclear leukocytes; PMNs) activate dendritic cells (DCs). However, early reports illustrated that neutrophil-derived mediators may suppress responses to mitogens. In the present study, we investigated the mechanism used by PMNs to modulate the immunostimulatory ability of DCs. Autologous syngeneic PMNs decreased T-cell proliferation induced by allogeneic DCs. Culture supernatant (CS) derived from PMNs also decreased allostimulation ability of immature DCs and increased the expression of transforming growth factor (TGF)-beta1 on DCs. A TGF-beta1 monoclonal antibody, a CD40 monoclonal antibody, or a serine protease inhibitor reversed the effect of PMN CS on DC allostimulatory ability. Furthermore, elastase reproduced the inhibitory effect of PMN CS on DC allostimulatory ability and the TGF-beta1 production. The role of elastase was confirmed by examining PMN CS from two patients with cyclic neutropenia, a disease due to mutations in the neutrophil elastase gene. These PMN CS samples had reduced elastase activity and were unable to increase DC TGF-beta1 production. Moreover, elastase and PMN CS induced IkappaBalpha degradation in DCs. We conclude that PMNs decrease DC allostimulatory ability via production of elastase leading to a switch of immature DCs into TGF-beta1-secreting cells.

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