Clinical Research Seminar April 18, 2012

Understanding and Complying

The NIH Public Access Policy

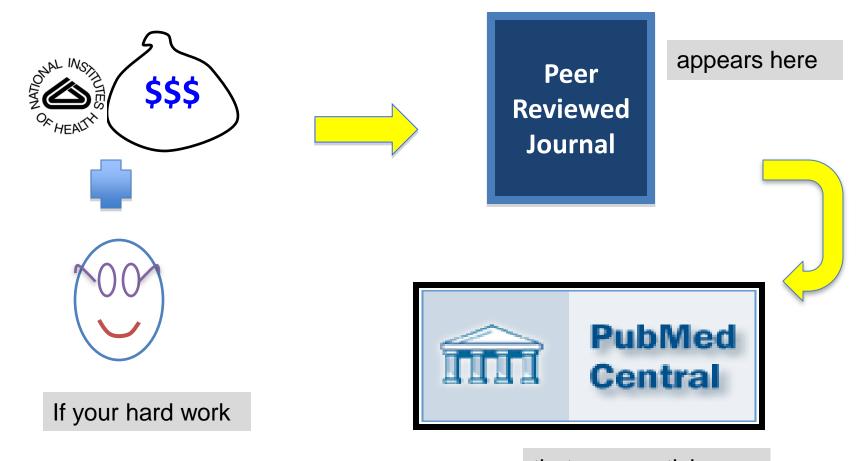


The Law says...

The NIH Public Access Policy requires that all investigators funded by the NIH, submit or have submitted for them to the National Library of Medicine's PubMed Central (PMC), an electronic version of their final, peer-reviewed manuscripts upon acceptance for publication, to be made publicly available no later than 12 months after the official date of publication.

Division G, Title II, Section 218, PL 110-161 (Consolidated Appropriations Act, 2008)

In a nutshell...



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Works assigned a PMCID Over two million full-text works

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Help

Displ

Cancer Res. 2009 Jul 15;69(14):5673-80. Epub 2009 Jun 23.

Overexpression of DPAGT1 leads to aberrant N-glycosylation of E-cadherin and cellular discohesion in oral cancer.

Nita-Lazar M, Noonan V, Rebustini I, Walker J, Menko AS, Kukuruzinska MA.

Department of Molecular and Cell Biology, Boston University Medical Center, Boston, Massachusetts 02118, USA.

Abstract

Cancer cells are frequently characterized by aberrant increases in protein N-glycosylation and by disruption of Ecadherin-mediated adherens junctions. The relationship between altered N-glycosylation and loss of E-cadherin adhesion in cancer, however, remains unclear. Previously, we reported that complex N-glycans on the extracellular domains of E-cadherin inhibited the formation of mature adherens junctions. Here, we examined whether dysregulated N-glycosylation was one of the underlying causes for cellular discohesion in oral cancer. We show that dense cultures of human salivary epidermoid carcinoma A253 cells exhibited elevated expression of DPAGT1, the gene that initiates protein N-glycosylation. Overexpression of DPAGT1 correlated with the production of E-cadherin-bearing complex N-glycans in nascent adherens junctions. Partial inhibition of DPAGT1 with small interfering RNA reduced the complex N-glycans of E-cadherin and increased the abundance of alphacatenin and stabilizing proteins in adherens junctions. This was associated with the assembly of functional tight junctions. The inverse relationship between DPAGT1 expression and intercellular adhesion was a feature of oral squamous cell carcinoma. Oral squamous cell carcinomas displayed overexpression of DPAGT1 that correlated with diminished localization of E-cadherin and alpha-catenin at the sites of adherens junctions. Our studies show for the first time that DPAGT1 is an upstream regulator of E-cadherin N-glycosylation status and adherens junction composition and suggest that dysregulation of DPAGT1 causes disturbances in intercellular adhesion in oral cancer.

MID: 19549906 PubMed - indexed for MEDLINE] PMCID: PMC277119

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Research Support, N.I.H., Extramural

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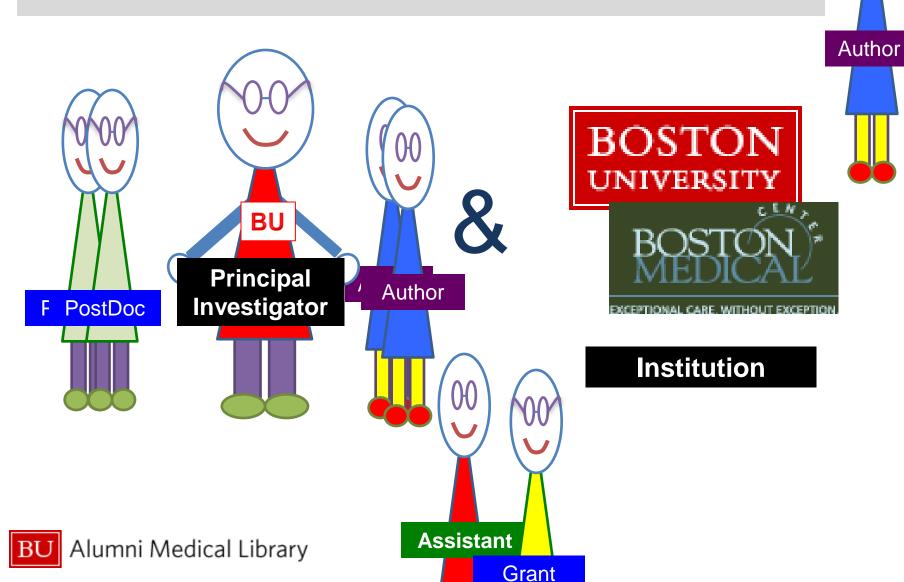
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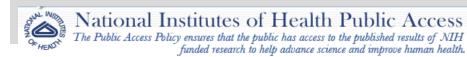
2. Manuscript *Citation*

How to comply?

Part I

Manuscript *Submission*

How to Submit? \rightarrow 4 methods



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- 1. Determine Applicability
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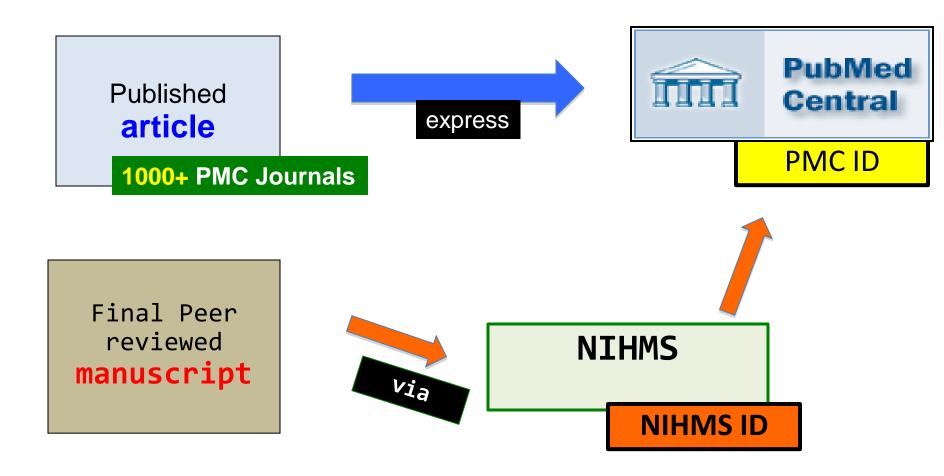
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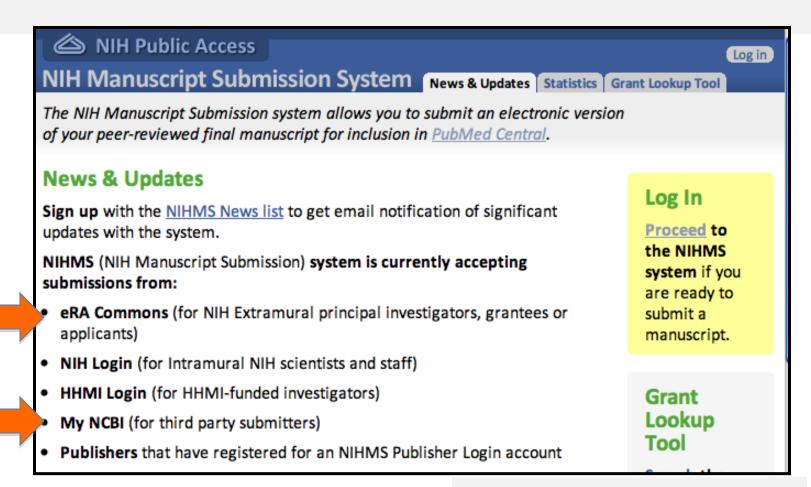
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What to PMC & How to PMC



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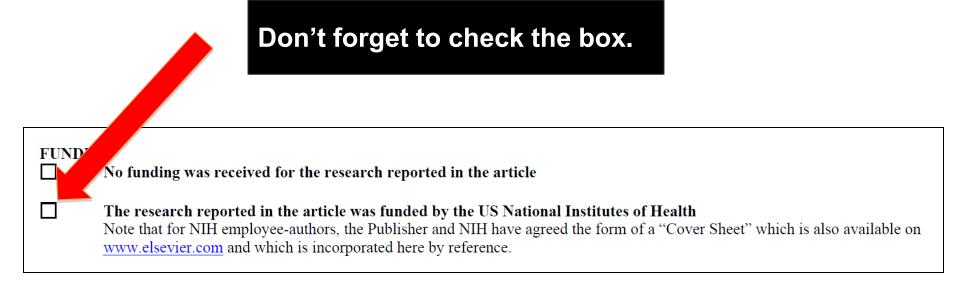
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The policy ONLY applies to...

YES

Peer Reviewed Journal Article

NO



How to comply?

Part II

Manuscript *Citation*

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But, submittal to PMC should be made upon ACCEPTANCE of publications.

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PI needs PMCID

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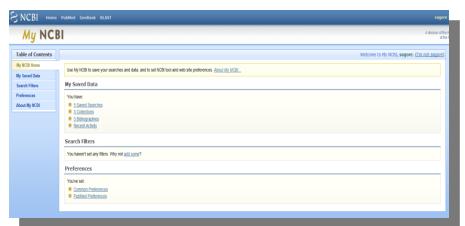
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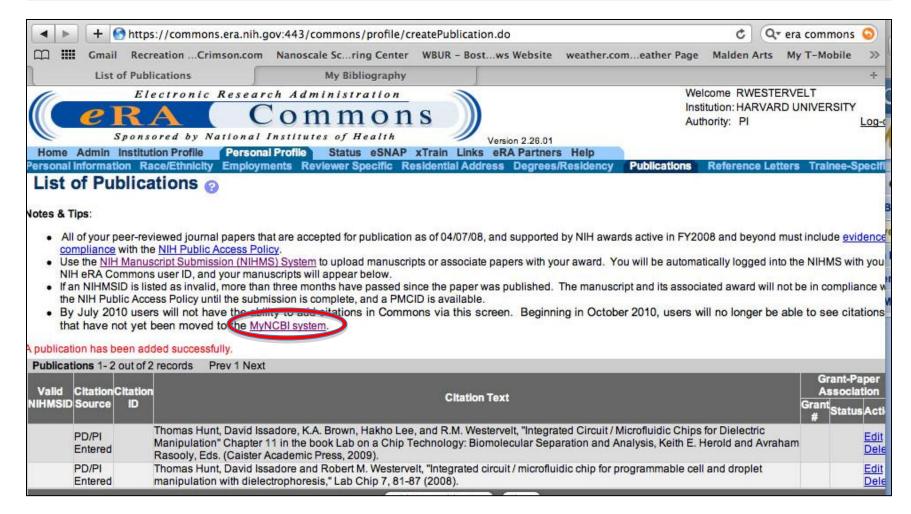


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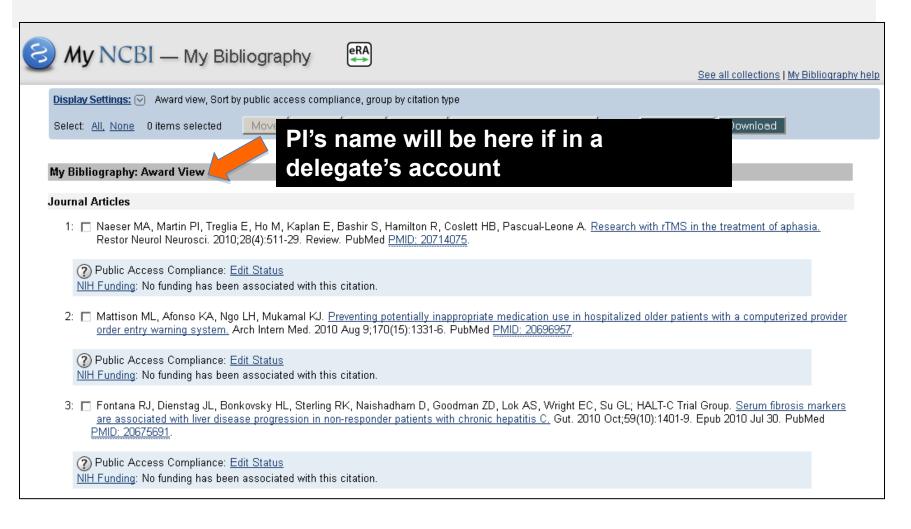


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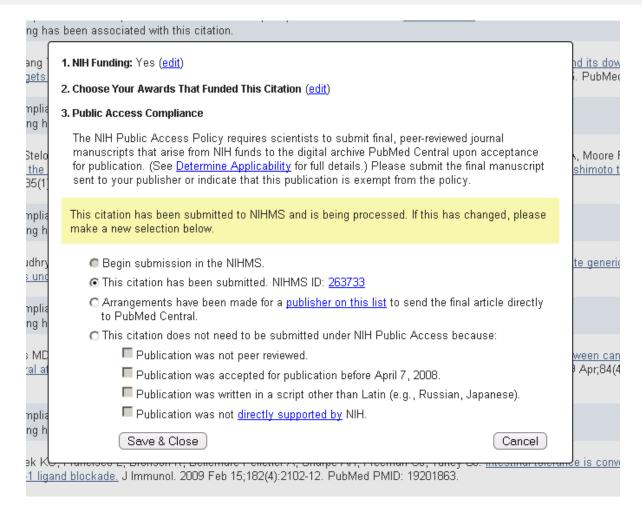
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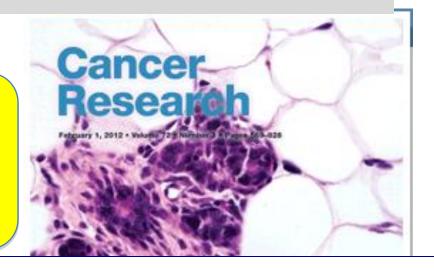
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QUIZ TIME

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NIH Grantees

Author

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Biochem Biophys Res Comm in. 2011 Jul 1;410(2) 333-8. Epub 2011 Jun 6.

Podocan-like protein: a novel small leucine-rich repeat matrix protein in bone.

Mochida Y, Kaku M, Yoshida K, Katafuchi M, Atsawasuwan P, Yamauchi M.

Department of Periodontology and Oral Biology, Boston University, Henry M. Goldman School of Dental Medicine, 700 Albany Street, Boston, MA 02118, USA. mochida@bu.edu

Abstract

Recently, significant attention has been drawn to the biology of small leucine-rich repeat proteoglycans (SLRPs) due to their multiple functionalities in various cell types and tissues. Here, we characterize a novel SLRP member, "Podocan-like (Podnl) protein" identified by a bioinformatics approach. The Podnl protein has a signal peptide, a unique cysteine-rich N-terminal cluster, 21 leucine-rich repeat (LRR) motifs, and one putative N-glycosylation site. This protein is structurally similar to podocan in SLRPs. The gene was highly expressed in mineralized tissues and in osteoblastic cells and the high expression level was observed at and after matrix mineralization in vitro. Podnl was enriched in newly formed bones based on immunohistochemical analysis. When Podnl was transfected into osteoblastic cells, the protein with N-glycosylation was detected mainly in the cultured medium, indicating that Podnl is a secreted N-glycosylated protein. The endogenous Podnl protein was also present in bone matrix. These data provide a new insight into our understanding of the emerging SLRP functions in bone formation.

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PMID: 21672516 PubMed - indexed for MEDLINE PMCID: PMC3159388 [Available on 2012/7/1]

Publication Types, MeSH Terms, Substances, Grant Support

Publication Types

Research Support, N.I.H., Extramural

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http://publicaccess.nih.gov/FAQ.htm

PMC Journals (=Method A)

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Do they change?
How about Method C & D?
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- → Any papers originated from the grant (which pays 2% of salary) is subject to the Policy.
- → All other research (98% of PI's salary is funded) is NOT.

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Alert from NIH

C Ornell et al.

Antiviral Therapy 14:523-531

Original article

HIV type-1 clade C resistance genotypes in treatmentnaive patients and after first virological failure in a large community antiretroviral therapy programme

Catherine Orrell*, Rochelle P Walensky^{23,4}, Elena Losina^{23,4,6}, Jennifer Pitt¹, Kenneth A Freedberg^{2,3,6,6} and Robin Wood¹

Desmand Tutu HIV Foundation, Institute of Infectious Disease and Molecular Medicine, Faculty of Health Sciences, University of Cape Town, Cape Town, South Africa

*Division of General Medicine and Infectious Disease, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA *Division of General Medicine and Infectious Disease, Massachusetts General Hospital, Harvard Center for AIDS Research, Boston, MA, USA *Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

*Department of Orthopedic Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA *Department of Biostatistics and Epidemiology, Boston University School of Public Health, Boston, MA, USA

repartment or anomalous are aproximately section of the section of

*Corresponding author: E-mail: catherine.orrell@hiv-research.org.za

Background: This study aimed to evaluate HIV type-1 (HIV-1) drug resistance pretreatment and in those failtreatment-experienced patients, 95 (86%) individuals had therapy-limiting NNRII mutations including K103N (55%).

Let me know if you want to receive updated info.

ade C virus should be of concern nmes, as the presence of this muta lility to all NRTIs except AZT, the choice of NRTIs for second-lim

this study is the 4-6 month window d samples in the South African antimme. Some individuals might have ks of their last suppressed viral load days of their first raised viral load, virological breakthrough to time of serefore be an underestimate.

e reverse transcriptase enzyme after TI-containing first-line therapy folis predictable and similar to that of istance to antiretrovirals that require tation, followed by slower developto drugs with a higher genetic barrier as the thymidine analogues.

e treatment been commenced within all virological breakthrough in the tood group in this study, the likeliting TAMs might have been reduced, orease in the efficacy of the recycled are in second-line therapy. Identificates the study of the recycled are in second-line the second-line all suggest clinical value to regular to identify virological failure soon ontrast to a recently published model to unexpected emergence of the K65R bstantial proportion of the cohort, the introduced cautiously with careful fect on the emergence of resistance.

This study suggests that at present it is not crucial, in the context of the South African National ART programme, to have routine access to genotypes at baseline, as the vast majority of treatment-naive samples continue to be wild type. By contrast, the development of extensive resistance in those failing first-line therapy suggests that viral load monitoring is crucial and there could be a role for individual genotypes in those failing first-line therapy, particularly if second-line therapy is likely to be compromised by resistance to first-line therapy. Increased availability of low-cost assays for identifying resistance in patients in South Africa would be clinically valuable.

Acknowledgements

Many thanks go to Monica Vogt and Felicity Cope, who carefully stored and retrieved samples for us for many years. We would also like to thank the staff at Toga Laboratories that stored and retrieved the more recent samples.

This work was supported by the National Institute of Allergy and Infectious Diseases (R01 Al058736, K24 Al062476, P30 Al060354) and the Doris Duke Charitable Foundation Clinical Scientist Development Award.

Discio- statement

CO and RW are partially supported through the NIH CIPRA-SA grant. In addition, CO receives partial funding from the PEPFAR grant. All other authors declare no competing interests.

Additional file

The additional file 'Supplementary figures' can be accessed at www.intmedpress.com

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Thank you!

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Sylvia Baedorf Kassis, MPH

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