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Peter (P.W.) Andrews

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Department of Biochemistry and
Cell Biology
State University of New York
at Stony Brook
Stony Brook, New York, USA

Anders (A.H.) Lund

Biotech Research and Innovation Centre
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Carlos Martínez-A

Department of Immunology and Oncology
National Center for Biotechnology
Campus Universidad Autonoma
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Correspondence regarding production may be sent to:

Biochemical and Biophysical Research Communications, Elsevier Inc.

525 B Street, Suite 1800, San Diego, California 92101-4495, USA
Telephone +1 (619) 699-6857, Fax +1 (619) 699-6859, E-mail bbr@elsevier.com



0006-291X(20141205)455:1-2;1-I

Cover photo. Epigenomic relatedness of *IDH/SDH/FH* mutant- versus non-mutant- tumors, as shown by PCA plots and heatmap of DNA methylation profiles. Samples included here are: (1) *IDH*-mutant versus -wildtype cholangiocarcinoma (GSE49656, $n = 32$; GSE32286, $n = 50$), *IDH*-mutant versus -wildtype glioma (GSE36278, $n = 136$; GSE48461, $n = 56$; GSE32286, $n = 62$) and *IDH*-mutant versus -wildtype chondrosarcoma (GSE40853, $n = 51$); (2) *SDHx*- versus kinase-mutant GIST (GSE34387, $n = 69$) and *SDHx*- versus kinase-mutant paraganglioma/pheochromocytoma (GSE43293, $n = 22$); and (3) multiple normal associated tissue lineages ($n = 19$). Variables included here are the CpG methylation β -values, as measured by Infinium 450 K array, of the top 10 K differentially-methylated CpG targets between *IDH/SDH/FH* mutant- and non-mutant tumor groups (statistical calculations and graphics performed with QluCore Omics Explorer software). In general, *IDH/SDH/FH* mutant tumors of diverse histological types and embryonic lineages show significantly greater global DNA hypermethylation than non-mutant counterparts. See J.J. Waterfall et al., The role of mutation of metabolism-related genes in genomic hypermethylation, *Biochem. Biophys. Res. Commun.* 455 (2014), 16–23 (Figure 1, this issue).