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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).