NSUN5

Tumors have aberrant proteomes that often do not match their corresponding transcriptome profiles. One possible cause of this discrepancy is the existence of aberrant RNA modification landscapes in the so-called epitranscriptome.

Janin et al. reported that human glioma cells undergo DNA methylation-associated epigenetic silencing of NSUN5, a candidate RNA methyltransferase for 5-methylcytosine. In this setting, NSUN5 exhibits tumor-suppressor characteristics in vivo glioma models. They also found that NSUN5 loss generates an unmethylated status at the C3782 position of 28S rRNA that drives an overall depletion of protein synthesis, and leads to the emergence of an adaptive translational program for survival under conditions of cellular stress. Interestingly, NSUN5 epigenetic inactivation also renders these gliomas sensitive to bioactivatable substrates of the stress-related enzyme NQO1. Most importantly, NSUN5 epigenetic inactivation is a hallmark of glioma patients with long-term survival for this otherwise devastating disease [1)].

1)
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