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Poster Submissions

Poster Title

Pan-cancer analysis of RNA editing suggest how RNA editing changes in 3' UTR regulate oncogenes/tumor suppressors

Authors and their Affiliation

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Please describe the extent of your work in this research

I did all the work.

Abstract Submission

- RNAediting_GSI2015_abstract.docx

Would you like your abstract to be considered for an oral presentation (students and post docs only)?

Yes

Pan-cancer analysis of RNA editing suggest how RNA editing changes in 3' UTR regulate oncogenes/tumor suppressors

Liye Zhang[1], Stefano Monti[1]
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Abstracts:

RNA editing is a post-transcriptional modification targeting various cellular RNAs. Despite the large number of RNA editing sites characterized in humans, altered RNA editing is still a largely unexplored source of oncogenic transcriptome variations. Cancer is driven by various genetic alterations, yet very few studies have analyzed the role of RNA editing in cancer. Here, we first characterize the RNA editing sites in patient samples from lymphoma, neuroblastoma and head and neck cancers. We show that A-to-I RNA editing sites are highly conserved across most samples of the same tissue type and that most A-to-I RNA editing sites identified in cancer samples are also detectable in normal tissues, suggesting that cancer cells do not acquire novel (somatic) A-I RNA editing sites. Next, we identify the significant changes in RNA editing levels of known sites between tumor and paired normals across 14 cancer types (627 tumor-normal RNA-Seq samples pairs) from The Cancer Genome Atlas (TCGA) project and show that the complexity of RNA editing regulation cannot be adequately captured by the activity of ADAR family genes alone. Our pan-cancer analysis confirms previous results on individual tumor types with limited sample size and suggests that changes of RNA editing levels in coding and 3' UTR regions could be a general mechanism to promote tumor growth. We also propose a model explaining how altered RNA editing levels affect microRNA-mediated expression regulation of oncogenes and tumor suppressors. The catalogue of editing sites we compiled, comprising sites with significant differential editing level between tumors and normals, and those recurring across multiple cancer types, can serve as a useful resource to guide future functional studies.