Comment re: Ran-GTP Control of Tumor Cell Mitosis

In Response:

We thank Simula and colleagues for commenting on our recent article (1), and for reporting their findings of increased Ran expression in a group of patients with histopathologic features of destructive celiac disease. Although it will be necessary to confirm these results in a much larger patient population, the idea that Ran up-regulation may occur in epithelial cells chronically exposed to inflammatory conditions, and prone to full-blown malignant transformation, is generally in keeping with our own conclusions (1), identifying Ran as a mediator of aberrant cell division, potentially contributing to aneuploidy and genomic instability.

However, it is important to caution against premature generalization from the initial observations of Simula and colleagues. In particular, our results, along with the data of a follow up study (2), emphasize that increased expression not only of Ran, but also of Ran-GTP effector molecules, for instance survivin, is critical to globally exploit this pathway for deregulated cell division. It is not inconceivable that survivin or other Ran-GTP effector molecules, for instance Aurora A or TPX2, may also become up-regulated during chronic inflammatory responses, in vivo. However, in the absence of these data, a potential causative role of Ran in mediating aberrant cell division during the evolution of celiac disease remains experimentally unproven.

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Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

References


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