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CONFIDENTIAL: request to review Scientific Reports manuscript SREP-15-21934

1 message

scientificreports@nature.com <scientificreports@nature.com>
Reply-To: scientificreports@nature.com
To: scguo@ucsd.edu

Sat, Aug 1, 2015 at 11:50 PM

Dear Dr. Guo,

A manuscript has been submitted to Scientific Reports, which we were hoping you would be interested in reviewing. The manuscript comes from Prof Di et al. and is entitled "Epidemiological correlation between cancer risk and tumor genomic mutation rate confirms the predominant contribution of somatic mutation"; the abstract is appended below.

Scientific Reports is an online multidisciplinary publication which is committed to providing a rapid and fair review process. We would hope to receive your comments within 7 days if you are able to review the manuscript. However if you would like to assist us, but require a few extra days to review the manuscript, please do not hesitate to contact us.

To respond to our request, please use the following link:

<http://mts-srep.nature.com/cgi-bin/main.plex?el=A3CG1Cfp7A1Bzpu2J5A9ftdnNHBj54gPL1KJhLmmPLa7wZ>

From there, simply follow the link to manuscript SREP-15-21934, where you will be able to view general manuscript information followed by options to accept or decline our request.

If you are unable to help on this occasion, we would appreciate any suggestions for alternative reviewers - perhaps someone in your own laboratory might be suitably qualified?

Many thanks in advance for your help; I look forward to hearing from you. Please do not hesitate to contact me by replying to this e-mail if you have any questions.

Best regards,

Jiucun Wang
Editorial Board Member
Scientific Reports

Dapeng Hao, Li Wang, and Li-jun Di

Cancer is believed to be a result of accumulated mutations. However, this concept has not been fully confirmed owing to the impossibility of tracking down the ancestral somatic cell with mutation accumulation before it gives rise to a detectable tumor. We sought to verify the concept by exploring the correlation between cancer risk and "consensus" mutation rate in bulk tumor of different tissues. We collected a comprehensive list of "consensus" mutation rates revealed by bulk tumor sequencing of 53 studies, and investigated its correlation with cancer risk to mirror the correlation between mutation rate in somatic cells and cancer risk. This revealed a 1:1 relationship between mutation rate and cancer risk in 41 different cancer types based on the sequencing data of 5,542 patients. The correlation was extremely robust even against the variation of estimations of mutation rate. Moreover, the correlation establishes a baseline to evaluate the effect of non-mutagenic carcinogens on cancer risk. Since the mutations obtained from bulk tumor sequencing are largely inherited from somatic mutations of an ancestral cell that gives rise to tumor, our mathematic modeling provides the evidence to reinforce that cancer risk is predominantly determined by the first rate-limiting mutation.

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