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LETTERS TO THE EDITOR

nm23H1 expression and its role in the evolution of non-gastrointestinal malignancies

Shailendra Kapoor

Shailendra Kapoor, 26-Hoffman Estates, Schaumburg, IL 60197, United States
Author contributions: Kapoor S wrote the draft of the letter.
Correspondence to: Shailendra Kapoor, MD, Physician, 26-Hoffman Estates, Schaumburg, IL 60197, United States. shailendrakapoor@yahoo.com
Telephone: +1-847-8857234 Fax: +1-847-5678907
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Abstract

The role of *nm23H1* genetic instability is not limited to gastrointestinal malignancies. A similar close relationship exists between *nm23H1* genetic instability and other non gastrointestinal systemic malignancies. For instance, in oral malignant melanomas with lymphoid metastasis, the *nm23H1* expression is significantly lower in contrast to tumors with no lymphoid metastasis. Similarly, increased metastasis is seen in non small cell lung cancers following down regulation of *nm23H1* in conjunction with KAI-1 down regulation. There is an inverse relationship between tumor stage and metastasis and *nm23H1* expression in individuals with prostate carcinomas and a similar relationship exists between microsatellite instability of the *nm23H1* gene and ovarian carcinogenesis. For instance, nearly 70.5% of stage Ⅰ-Ⅱ ovarian tumors express *nm23H1* in sharp contrast to only 25% of stage Ⅲ-Ⅳ ovarian tumors. As is clearly evident, *nm23H1* has a major role in gastrointestinal and non-gastrointestinal carcinogenesis. The coming few years will hopefully see the development of new strategies by virtue of which we can alter *nm23H1* expression and thus decrease the risk of metastasis in malignant tumors.

Key words: *nm23H1*; Non small cell lung cancers; Prostate carcinomas; Nasopharyngeal carcinomas

Peer reviewer: Chi-Hin Cho, Chair and Professor, Departmentof Pharmacology, The University of Hong Kong, 21 Sassoon Road, Hong Kong, China

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TO THE EDITOR

The recent article by Yang *et al*[1] about the relationship between gastrointestinal malignancies and *nm23H1* genetic instability is highly interesting. Interestingly, the role of *nm23H1* genetic instability is not limited to gastrointestinal malignancies. In fact, a similar close and intricate relationship exists between *nm23H1* genetic instability and other non gastrointestinal systemic malignancies.

For instance, Korabiowska *et al*[2] have shown that the *nm23H1* expression is significantly lower in oral malignant melanomas with lymphoid metastasis than in tumors with no lymphoid metastasis. Similarly, increased metastasis is seen in non small cell lung cancers following down regulation of *nm23H1* in conjunction with KAI-1 down regulation[3]. Similarly, there is an inverse relationship between tumor stage and metastasis and *nm23H1* expression in individuals with prostate carcinomas[4]. A similar relationship exists between microsatellite instability of the *nm23H1* gene and ovarian carcinogenesis. For instance, nearly 70.5% of stage Ⅰ-Ⅱ ovarian tumors express *nm23H1* in sharp contrast to only 25% of stage Ⅲ-Ⅳ ovarian tumors[5].

Similarly, *nm23H1* expression may be used to determine response to treatment. For instance, following radiotherapy, the five-year survival rate in patients with nasopharyngeal carcinomas and high expression of *nm23H1* is 53.2% in comparison to only 22.7% in individuals with low expression of *nm23H1*[6]. In fact, transfer of *nm23H1 via* adeno viruses is rapidly emerging as a potential therapeutic tool to prevent metastasis. For instance, this method has been shown to decrease metastasis in implantation models of ovarian cancer[7].

As is clearly evident, *nm23H1* plays a major role in gastrointestinal and non-gastrointestinal carcinogenesis. The coming few years will hopefully see the development of new strategies by virtue of which we can alter *nm23H1* expression and thus decrease the risk of metastasis in malignant tumors.

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