



DNA methylation and folate metabolism in gastric cancer

Shun-Shi Zhu, Shu-Dong Xiao, Zhi-Ping Chen, Yao Shi, Jing-Yuan Fang, Rong-Rong Li, Joel B Mason

Shun-Shi Zhu, Shanghai Second Medical University, Shanghai Ninth Hospital, Shanghai 200011, China

Shu-Dong Xiao, Zhi-Ping Chen, Yao Shi, Jing-Yuan Fang, Rong-Rong Li, Shanghai Second Medical University, Shanghai Renji Hospital, Shanghai 200001, China

Joel B Mason, Human Nutrition Research Center at Tufts University, United States

Author contributions: All authors contributed equally to the work.

Supported by National Natural Science Foundation, No. 39370332

Correspondence to: Dr. Shun-Shi Zhu, Shanghai Second Medical University, Shanghai Ninth Hospital, Shanghai 200011, China
Telephone: +86-21-63138341
Fax: +86-21-63136856

Received: May 25, 2000
Revised: June 24, 2000
Accepted: July 10, 2000
Published online: September 15, 2000

Abstract

AIM: To investigate DNA methylation status in gastric cancer and its relationship with folate metabolism.

METHODS: Serum before operation, the gastric mucosa from the lesion, and the surrounding area in patients with gastric cancer and the remote normal appearing mucosa of the resected stomach were collected respectively. The serum folate, mucosal tissue folate, S-adenosylmethionine (SAM), S-adenosylhomocysteine (SAH), and the DNA methylation levels were determined.

RESULTS: The tissue folate was significantly lower than that in ulcers, especially in the surrounding and normal mucosa (0.38 ± 0.13 , 0.50 ± 0.17 vs 0.53 ± 0.50 , 0.79 ± 0.82 ng/mg protein, $P < 0.01$), and it decreased gradually in the lesion areas. The DNA methylation status showed similar decreasing trend in cancers compared with the methylation increasing trend in ulcers. The SAM level ascended in the lesion areas with a higher concentration in cancer mucosa (63.5 ± 43.0 vs 25.9 ± 11.9 nmol/g tissue, $P < 0.01$). The accumulation of SAH in the surrounding and normal mucosa of cancers was observed (17.3 ± 24.6 , 15.5 ± 8.6 vs 14.6 ± 4.2 , 10.0 ± 1.9 nmol/g tissue, $P < 0.05-0.01$). There were significantly negative correlations between tissue folate and the SAM and SAH levels in the three areas.

CONCLUSION: Patients with gastric cancer have the regional folate deficiency in the stomach mucosa, although the serum folate level remains normal. This disturbs the local SAM and SAH metabolism with accumulation of SAH and DNA hypomethylation which has been known as an important molecular mechanism for carcinogenesis. Folic acid can modulate DNA methylation status by its effect in one carbon group metabolism and thus affect the process of the carcinogenesis. Therefore, this may be an access for the prevention of gastric cancer.

Key words: Stomach neoplasms; DNA methylation; Folic acid/metabolism; Gastric mucosa; Stomach neoplasms/prevention and control

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Zhu SS, Xiao SD, Chen ZP, Shi Y, Fang JY, Li RR, Mason JB. DNA methylation and folate metabolism in gastric cancer. *World J Gastroenterology* 2000; 6(Suppl 3): 28 Available from: URL: <http://www.wjgnet.com/1007-9327/full/v6/iSuppl3/28.htm> DOI: <http://dx.doi.org/10.3748/wjg.v6.iSuppl3.28>

E- Editor: Hu S



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