CORRESPONDENCE

Re: Urokinase and the Urokinase Receptor: Association With In Vitro Invasiveness of Human Bladder Cancer Cell Lines

In their May 1997 report, Hudson and McReynolds stated, “To our knowledge, this is the first study demonstrating that bladder tumor cells express the urokinase receptor...” (1). I would respectfully point out that expression of the mRNA encoding the urinary plasminogen activator receptor by human transitional carcinoma cell lines and the regulation of it and other plasminogen activator-related proteins (urokinase, tissue plasminogen activator, plasminogen activator inhibitor 1 [PAI-1], and plasminogen activator inhibitor 2 [PAI-2]) by the cytokine-transforming growth factor-B1 and transforming growth factor-α, have been reported previously (2).

WILLIAM A. SEE

References


Note

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Response

Dr. See has pointed out that Xu and See (1) published a paper on cytokine regulation of plasminogen activator-related proteins in which the expression of the messenger RNA for urinary plasminogen activator receptor in transitional cell carcinoma cell lines was reported. Omission of Drs. Xu and See’s work in our report (2) was by no means deliberate. We were unaware of their article, which was not found on a standard MEDLINE subject search at the time that our manuscript was submitted. For completeness, we also add that we were unaware that Drs. Xu and See had presented these same data at the annual meeting of the American Urological Association in April 1995, which were published as an abstract in the Journal of Urology (3).

Dr. See has cited only a part of the sentence from our report, and we believe that it lost some of its meaning when taken out of context. The full sentence reads, “To our knowledge, this is the first study demonstrating that bladder tumor cells express the urokinase receptor and that both receptor expression and urokinase expression are required for bladder tumor cell invasion in vitro.” Those who have read our report realize that the paper primarily emphasizes the functional relationship between concomitant expression of urokinase and its receptor and the ability of bladder cancer cells to invade an artificial basement membrane. The importance of our work is in the functional significance of the receptor, not merely its presence. A preliminary report of our work on the detection of the urokinase receptor in human bladder cancer was also published in an abstract form and presented at the annual meeting of the American Association for Cancer Research in March 1995 (4).

M’Liss A. Hudson

Dietary Fat and Breast Cancer

A recent review (1) and an article (2), both appearing in the Journal, suggest that the misclassification of fat intake as measured by food-frequency questionnaires (FFQs) is the reason that cohort studies consistently have “been unable” to detect the hypothesized relation of dietary fat intake with breast cancer risk. We wish to respond briefly to these arguments and critiques of our pooled analysis of seven cohort studies of the relationship between dietary fat and breast cancer (3).

Both Wynder et al. (1) and Prentice (2) make much of the data from studies that use doubly labeled water to estimate energy expenditure; these data have been interpreted to show that energy expenditure is generally underestimated by individuals and more severely underestimated by obese individuals. Some underestimation of energy intake by FFQs is undisputed, but it has not been established that obese individuals exhibit bias in reporting the fat composition of their diet (i.e., fat intake adjusted for energy intake). Prentice (2) references one analysis in which reporting of protein (not fat) intake was correlated with obesity even after adjustment for reported energy intake (4). Other analyses, such as the analysis by Lissner and Lindross (5), have concluded that the degree of underreporting of energy intake is not associated with the fat composition of the diet.

Prentice’s argument (2) depends on his postulated measurement-error model, which forces the validity of reporting of fat composition of diet to depend on body mass index (BMI). It should be noted that this model is theoretical, and Prentice offers no data to support it. Nonetheless, Prentice’s model makes a testable prediction, “…the estimated probabilities that a person reporting a 20%-calories-from-fat diet is in the lowest, middle, and highest BMI tertile are...16.2%, 14.4%, and 69.3%, respectively, for the FFQ’’