

CORRECTION *The Journal of Cell Biology*

Vol. 83, No. 2, Pt. 2, November 1979

The following Abstract should have appeared on page 479a of the Abstracts, The American Society for Cell Biology, Nineteenth Annual Meeting, 4-8 November 1979, Toronto, Ontario, Canada:

22928 HISTOCHEMICAL DETECTION OF HIGH γ -GLUTAMYL TRANSFERASE ACTIVITY IN HUMAN EPITHELIAL TUMORS. Silvio Fiala and Edgar C. Trout*. Cell Physiology Laboratory, VA Medical Center Martinsburg, WV 25401 and Shepherd College, Shepherdstown, WV 25443.

γ -glutamyl transferase (E.C. 2.3.2.2., GGT) is a good Marker in chemically induced carcinogenesis in rat liver and dimethyl hydrazine-induced cancers of the colon (Fiala et al, J. Natl. Cancer Inst. 48, 1393, 1972, Arch. Geschwulstforsch., 47, 117, 1977). The similarity between experimental carcinogenesis in rat colon to human colon cancers led us to investigate, using the histochemical method of Rutenburg et al (J. Histochem. Cytochem. 17, 517, 1969), whether human adenocarcinomas of the colon will be distinguished from the normal adjacent tissue by high GGT activity. This was, indeed, the case (Fiala et al, The Lancet, May 26, 1979). In extending a systematic search for GGT as a potential Marker in cases of various human cancers of epithelial origin, we detected histochemically extremely high GGT activity in histologically established cancers of the oesophagus, tongue, urinary bladder, Ca of the larynx. In all these cases the cancerous tissue and foci were clearly distinguished by high GGT activity from GGT-negative adjacent normal tissue. In normal human prostate only the epithelium lining the glandular cavities was GGT(+) but GGT(+) reaction was found also in the secret inside the lumen of the glands. In Ca of the prostate, GGT(+) reaction was conspicuously increased in the randomly arranged malignant acini. Where the alveolar pattern prevailed, the highest activity was in the lumen-oriented layers, suggesting that in normal and also in malignantly changed prostate a great portion of the enzyme formed intracellularly is excreted in the soluble form. Our observations were possible by cooperation of the Department of Surgery, VA Medical Center, Martinsburg, and were supported by the VA Dept. of Research and by a Grant CA-14084 from the National Cancer Institute, USPHS.
