CELL BIOLOGY

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Purification of S-Methyl-L-Methionine:Homocysteine Methyltransferase in *Triticum aestivum* (Gramineae). James E. Bryan and Betty D. Allamong, Department of Biology, Ball State University, Muncie, Indiana 47306.——Purification procedures for S-methyl-L-methionine:homocysteine methyltransferase (EC 2.1.1.10) were investigated. Centrifugation studies at varying speeds, up to 105,000 xg, were performed and the majority of the activity was determined to be in the soluble fraction. Ammonium sulfate fractionation and gel filtration were employed to obtain a final 88-fold purification. The purity of the enzyme was checked using polyacrylamide gel electrophoresis. The molecular weight of the enzyme, was determined by gel filtration to be in the range of 35,000-42,000.

Endogenous Virus from Mouse L-Cells. R. J. Boyd, C. W. Godzeski, and V. C. Spurling, The Lilly Research Laboratories, Indianapolis, Indiana 46206.——The fact that mouse cells and tissues all carry tumor virus potential has been well publicized since 1958, but researchers do not seem to heed the implicit warnings. Mouse cell lines may express virus and/or virus-like particles as a result of relatively minor environmental changes. Our laboratory has found that mouse L-cells contain intracisternal type A particles and intracytoplasmic type A particles (possible precursor to the type B virus) when the serum content of the 199 medium was lowered to 2% from 5% or when $NaHCO_3$ was increased from 1.68 gms/L to 2.2 gms/L. Published reports imply that more drastic measures, e.g., halogenated pyrimidines, high serum levels, radiation, or cytotoxic agents, were required to induce endogenous virus release. This may not be so. Cautious handling of mouse cells and tissues is emphasized and the routine ultrastructural examination of such cells highly recommended.

Fatty Acid Composition of Microsomal and Soluble Fractions of Mammary Adenocarcinomas in Mice. Gloria M. K. Raines and Alice S. Bennett, Ball State University, Muncie, Indiana 47306.——It has been suggested that membrane characteristics associated with carcinomas could be related to an altered molecular structure of lipids in the plasma membrane. The microsomal and soluble fractions of the cell are major sites of *de novo* synthesis and elongation/desaturation of fatty acids. It was the purpose of this study to compare the fatty acid composition of microsomal and soluble fractions isolated from mammary adenocarcinomas with that of normal mammary tissue and to determine if deviations found in the plasma membrane isolated from tumors could be observed at these subcellular levels. Electron micrographs of tumor

cells were compared to those of normal. Microsomal and soluble fractions were isolated by differential centrifugation from mammary adenocarcinomas and from normal mammary tissue of Strain A female mice. Activities of nicotinamide adenine dinucleotide, reduced (NADH) dependent cytochrome C reductase and nicotinamide adenine dinucleotide phosphate, reduced (NADPH) dependent cytochrome C reductase in these fractions were determined. The fatty acids were extracted, methylated, and methyl esters identified and quantified using gas liquid chromatography. Polar and nonpolar GLC columns, silver nitrate thin-layer chromatography, hydrogenation, and spiking were used to confirm the identity of some fatty acids. Electron micrographs revealed that the endoplasmic reticulum of neoplastic cells was more discontinuous and fragmented than that of normal cells. The fatty acid composition of the microsomal fraction did not reflect the altered characteristics evident in the plasma membrane. Generally, the fatty acid composition of the microsomal and soluble fractions was similar in tumor and normal tissue, but there was a greater percentage of $C_{24:1}$ and $C_{22:0}$ fatty acids in tumors. An increase in the level of palmitic acid and of long chain fatty acids, as reported in studies on the plasma membrane isolated from tumors, was not evident in either of the microsomal or soluble fractions. There was evidence of greater utilization of NADPH in the reduction of cytochrome C reductase in tumors. This may result in a decreased availability of NADPH for fatty acid synthetase and lipogenesis.