American Society for Biochemistry and Molecular Biology
Meeting June 3-7, 1990

ABSTRACT MUST BE RECEIVED AT
SOCIETY OFFICE BY TUESDAY,
(NUARY 23, 1990.

Mail to your Society of membership
ASBMB or AAI
CIS, AAVI and SMI members send to AAI
9650 Rockville Pike
Bethesda, MD 20814

1990 ASBMB/AAI (FASEB) ABSTRACT FORM June 3-7, 90

DO NOT FOLD THIS FORM

IMPORTANT

1. Prepare abstract carefully on this form only.
2. Also fill out:
   a. Topic category selection
   b. Mailing address of first author
   c. Signature block for member's signature
3. Return to your society of membership by January 23, 1990.
   a. The original typed abstract form
   b. Three photocopies
   c. Abstract handling fee ($25)
   d. Program confirmation card
4. See over for complete instructions.

(PRESENTATION PREFERENCE (Check one)

☐ Oral ☐ Poster ☒ Indifferent

Final decision regarding presentation format is at the discretion of the programing society.

SELECT CATEGORY NUMBERS & TITLES
(See Topic Category Lists)

1. 1040-2 Structure of Euk...
2. 1047-2 Oncogenes
3. 1031-2 Molecular Genet...

LACTATE FORMATION AND MITOCHONDRIAL DNA STRUCTURE IN MOUS CELLS PROGRESSING TO MALIGNANCY. Durwood B. Ray, Tan M. Lam, an David H. Jones, Department of Biochemistry, Oral Roberts University Scho of Medicine, Tulsa, Oklahoma 74171.

One well recognized change in tumorigenic progression is a shift fro respiratory metabolism towards increased glycolysis. There are sever reports of mitochondrial DNA (mito-DNA) mutations in cancer cells, b how these are related to the observed respiratory changes is not know To assess this we have undertaken a comparison of lactate production an mito-DNA structural analysis among serially derived genetically relate cells that represent different stages of tumorigenic progression. The cel are: 1) normal NIH Swiss embryonic cells (mortal); 2) non-tumorigeni NIH/3T3 cells (immortal); 3) tumorigenic ras oncogene-transforme NIH/3T3 cells; and 4) highly tumorigenic ras oncogenic-transformed T1- cells (derived from a tumor produced in a NIH Swiss mouse injected wit the ras oncogene-transformed NIH/3T3 cells). The rate of lacta formation in the T1-A cells is 19.3% ± 1.9 (SEM) greater than in NIH/3T cells, as determined at various cell densities. This indicates a significa shift from respiratory metabolism towards glycolysis in T1-A cel compared to NIH/3T3 cells. Southern blot analysis of the mito-DNA isolate from all four cell types using 10 restriction enzymes revealed no RFLI among these DNAs. Thus despite a significant metabolic alteration, ther are no large deletions or DNA rearrangements in the mito-DNAs among th cells of this tumorigenic progression model. Whether smaller mito-DN mutations or mitochondrially related nuclear DNA mutations are respon sible will require further study. These studies may help define a new cla of cancer related genes that will add another piece to the cancer puzzle.

Blue lines are printer's cut lines, do not type on or outside of these lines