## 1964 #24

## EFFECTS OF ULTRAVIOLET RADIATION ON TRITIATED THYMIDINE UPTAKE IN FERTILIZED EGGS OF <u>Echinarachnius</u> parma

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Unfertilized eggs of Echinarachnius parma exposed to ultraviolet and subsequently fertilized have the same rate of  $H^3$  TDR uptake as non-radiated controls between fertilization and 31 min post-fertilization. The rate of incorporation becomes significantly more rapid after this point is the radiated population, which however has a 24 min cleavage delay. If the zygotes are irradiated 55 min post-fertilization, the immediately following  $H^3$  TDR uptake peak (70 min) and cleavage (83 min) are not delayed. The following or second cleavage occurred at 154 min (radiated) and min (non-radiated) with the peak  $H^3$  TDR incorporation at 145 min and 125 min respectively. The is a delay of 21 min for cleavage for 20 min for peak  $H^3$  TDR uptake, this latter being the synthesis for the third cleavage. In these experiments the DNA synthetic period in immediate preparation for a subsequent cleavage was not delayed by the irradiation even though a delay in cleavage occurred. This suggests that the ultraviolet was not affecting the DNA synthesis directly but was deleterious to other processes in preparation for the cell division. The increased rate of  $H^3$  TDD uptake in eggs radiated pre-fertilization cannot be clarified at present.

The ultraviolet dose of  $5.72 \times 10^{-3} \text{ ergs/cm}^2$  from a polychromatic Hanovia high pressure mercury arc was such that recovery would occur spontaneously in the dark and the ensuing de-velopment was normal in all aspects. Photorecovery in these phenomena was not studied.

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GAS EXCHANGE IN BIOLOGICAL SYSTEMS

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There is a bimodal distribution of  $CO_2$  tensions in biological forms.  $PCO_2$ s are generally less than 5 mmHg and  $[HCO_3^-]$  less than 10 meq/L in aquatic gas exchangers.  $PCO_2$ s are generally greater than 15 mmHg and  $[HCO_3^-]$  greater than 20 meq/L in terrestrial gas exchangers. This distribution may be explained by considering the quantitative relationship between  $O_2$  and  $CO_3$  tensions in body fluids in the steady state.

A general relationship may be derived for the relationship between  $PaO_2$  and  $PaCO_2$  as folllows:

$$PaO_2 = P_{I_{O_2}} - K_1 - \left[\frac{a_{CO_2}}{a_{O_2}} \times \frac{1}{R} (PaCO_2 - P_{I_{CO_2}} - K_2)\right]$$

where  $PaO_2$  : arterial  $O_2$  tension  $P_{I_{O_2}}$  : ambient  $O_2$  tension

R	:	respiratory exchange ratio
° co <sub>2</sub>	:	solubility coefficient CO2
•°02	:	solubility coefficient O2
$PaCO_2$	:	arterial CO <sub>2</sub> tension
P <sub>ICO2</sub>	:	ambient CO <sub>2</sub> tension
к1	:	concentration gradient arterial blood - mean expired water/air ${ m O}_2$ tension
к <sub>2</sub>	:	concentration gradient arterial blood - mean expired water/air $CO_2$ tension

This general relationship may be utilized to explain a wide variety of biological phenomena. For example the mode of gas transport may be characterized in a given system in terms of the parameters  $K_1$  and  $K_2$  as follows:

	Mode of gas exchange
K <sub>1</sub> zero; K <sub>2</sub> zero	simple passive diffusion
K <sub>1</sub> positive; K <sub>2</sub> positive quantitatively consistent	diffusion limitation v-p imbalance in gas exchange unit
K <sub>1</sub> negative; K <sub>2</sub> negative	counter current exchange
K <sub>1</sub> positive; K <sub>2</sub> zero	"active transport" of O2
K <sub>1</sub> zero; K <sub>2</sub> positive	"active transport" of CO2

The bimodal distribution of PCO<sub>2</sub> is based on the difference between  $\frac{a_{\rm CO_2}}{a_{\rm O_2}}$  in air as compared to water. In air this ratio is 1 and PO<sub>2</sub>s of 100 mmHg result in PCO<sub>2</sub>s that are greater than 10 mmHg. In water, however,  $\frac{{}^{a}CO_{2}}{{}^{a}O_{2}} > 20$  and PO<sub>2</sub>s of greater than 20 mmHg result in

PCO<sub>2</sub>s of less than 5 mmHg.

Since hydrogen ion concentration and  $HCO_3^-$  concentration vary as functions of  $PCO_2$ , forms with high PCO2s will have high bicarbonate concentration, and those with low PCO2s will have low concentrations.

As oxygen dependent metabolism developed an obligatory requirement for PO2 was established. Since the primary mode of gas transport is diffusion, PO2 in turn establishes the level of PCO<sub>2</sub> and bicarbonate. The values for PCO<sub>2</sub> and bicarbonate found in biological systems reflect diffusion as the gas transport mechanisms, but under certain circumstances these levels can be modified by specific transport mechanisms. This bimodal distribution of  $\text{CO}_2$  and bicarbonate results from the obligatory requirement for oxygen to subserve oxygen dependent metabolism.