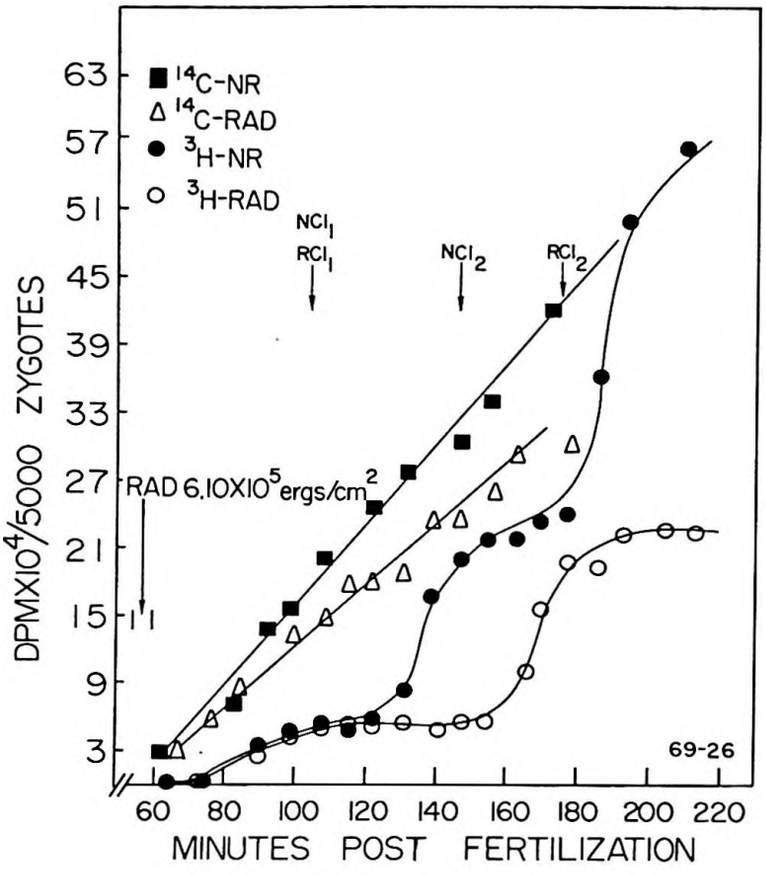

Figure 2.

ness as compared to the PR population. Thus, the PR has the capacity to reverse the UV lesion, which from all known evidence is a cyclobutane type pyrmidine dimer in DNA. This suggests that the "division related" protein synthesis is linked to DNA.

In separate experiments we have shown that the pyrmidine dimers are produced in E. parma zygotes at the same dose levels used in the above experiments and that these can be removed by PR. What the causal relationship between the studied synthetic events and the production of dimers actually is cannot be ascertained with the present information.

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OXYGEN CONSUMPTION DURING DIVING IN THE HARBER SEAL WITH SPECIAL REFERENCE TO A PARADOX OF INTERNAL GAS EXCHANGE

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During a prolonged dive, blood flow to all organs except brain, heart and central nervous system ceases. This permits all of the O_2 stores present in the circulating blood volume to be

available for oxidative metaolism in these organs while the peripheral tissues derive energy from araerobic glycolysis. The magnitude of residual O_2 consumption during the dive would be of obvious interest. In the present studies a model for the determination of diving O_2 consumption based on a volume approach rather than the classical flow approach has been developed. This model has been applied to the determination of diving O_2 consumption in 4 seals, Phoca vitulina. This model implies a rather striking paradox with respect to blood O_2 and CO_2 concentrations. Measurements of blood gas concentrations confirm the predictions of the model.

Consider that the blood volume (TBV) is separated into 2 compartments, an arterial compartment (Va) and a venous compartment (Vv). Total O_2 in the arterial compartment at any time (t) will be equal to $Va(Ca_{O_2})_t$ and in the venous compartment will be equal to $Vv(Cv_{O_2})_t$.

$$\text{Diving } O_2 \text{ consumption} = Va(Ca_{O_2})_{t1} - (Ca_{O_2})_{t2} \quad (1)$$

$$\text{This in turn will likewise be equal to } Vv(Cv_{O_2})_{t1} - (Cv_{O_2})_{t2} \quad (2)$$

Since TBV = Va + Vv,

$$\text{Then } Va = \frac{TBV\,[(Cv_{O_2})_{t1} - (Cv_{O_2})_{t2}]}{(Ca_{O_2})_{t1} - (Ca_{O_2})_{t2} + (Cv_{O_2})_{t1} - (Cv_{O_2})_{t2}}$$

$$\text{and } \dot{V}_{O_2} = Va(Ca_{O_2})_{t1} - (Ca_{O_2})_{t2}$$

Total blood volume was determined by the dilution of Evans Blue dye. Arterial blood was obtained sequentially during diving by means of an indwelling teflon catheter. "Mixed venous" blood was obtained sequentially during diving by means of a polyethylene catheter inserted in the extra-dural venous system.

Blood O_2 and CO_2 concentrations were determined manometrically using the method of Van Slyke and Neil. Diving O_2 consumptions measured by the above approach averaged approximately 15-20% of total cardiac output in the non-diving state, values comparable to those obtained in the same specie using the dye dilution method (Am. J. Physiol. 210:176, 1966). The calculated Vv constituted approximately 60% of the TBV, and calculated Va averaged approximately 40% of the TBV.

It may be noted that equation (1) and equation (2) would predict that if Va were smaller than Vv, the rate of change of Ca_{O_2} would be greater than the rate of change of Cv_{O_2}, and the rate of change of Ca_{CO_2} would be greater than that of Cv_{CO_2}. One would anticipate that (Ca_{O_2} - Cv_{O_2}) and (Cv_{CO_2} - Ca_{CO_2}) should progressively narrow despite a more or less constant cardiac output and metabolism. In each of the 4 seals studied there was progressive narrowing of the (A-V) O_2 difference and the (A-V) CO_2 difference actually showed a reversal so that arterial CO_2 concentrations became greater than venous CO_2 concentrations. Although the direct biological meaning of a reversed CO_2 concentration is obscure, this observation is in accord with recent studies showing higher alveolar than arterial CO_2 tensions during prolonged breath holding. The magnitude of the calculated diving O_2 consumption and the ability of the volume model to predict the direction of changes in blood gas concentration seems to validate the model. Further studies will be required to define the mechanisms by which a reversal of CO_2 concentration difference occurs between arterial and mixed venous blood.