Table 1. Reactions of various recognized parasites and disease entities with fluorescent antibody to *Minchinia nelsoni*. ++++, Maximum fluorescence, brilliant yellow green; +++, slightly less fluorescence; ++, definite fluorescence, yellow green, less brilliant; —, no fluorescence.

Test organism	Ref.	Type of reaction
Platyhelminths		
Trematoda		
Bucephalus sp.	(9)	—
Cestoda		
Tylocephalum sp.	(9)	—
Protozoa		
Sarcomastigophora		
Hexamita sp.	(8)	—
Sporozoa		
Nematopsis ostrearum	(6)	—
Minchinia costalis	(1,2)	—
Minchinia nelsoni		++++
Same, Delaware Bay	(1,2)	++++
Same, Chincoteague Bay	(3)	++++
Same, Chesapeake Bay	(1,3)	++++
(Also amoebula in specimens)		++++
Same from Wreck Shoal, James R., Va.*	(1)	+++
Haplosporidium sp. (Hyperparasite of *Bucephalus* sp.)	(7)	++
Ciliophora		
Ancistrocoma sp.	(6)	—
Thallophyta		
Fungi		
"Actinomycete" disease	(9)	—
"Mycelial" disease	(6)	—
Dermocystidium marinum	(8)	—
Diseases of unknown etiology		
"Rickettsial" disease	(9)	—
"Necrosis" of the gut	(9)	—
"Lymphocystosis"	(9)	—

* Not ideal fixation for this study.

tions. It was possible to follow a series of equally strong reacting stages of *M. nelsoni* through schizogony and sporogony (Fig. 1*C*). The intensity of reaction of the prespore stage was equal to the plasmodial stages (Fig. 1, *D* and *E*) and was not included in the illustration. Fluorescence occurred in the cytoplasm of sporoblasts and in the sporoplasm and capsule of the immature spore (Fig. 1*F*). The conjugate appeared unable to penetrate the mature spore wall and stained only the capsule (Fig. 1*G*). The only other organism in the oysters that fluoresced in the conjugate was an amoebula, which was either part of the schizogony cycle that has not been recognized or a rhizopoda. It was present in two of the infected oysters. Since whole oysters were used in these studies, the antigens included any organisms in the mantle cavity and the digestive system. When the distance separating the origins of these shellfish is considered, the degree of specificity is truly remarkable. Until further studies can demonstrate the relationships of this amoebula in the oysters, it is even possible that this reaction represents unabsorbed rhizopodal antigen. The amoebula was recognized in the gills, fluorescing as brightly as the plasmodia. The *Haplosporidium* sp. (*7*) hyperparasite of the trematode, *Bucephalus* (*6*), reacted in all stages of its life cycle with much less intensity than the *M. nelsoni*.

Since the intensity of reaction of the stages of sporogony tested in these studies of conjugated antiserum to *M. nelsoni* is unlike the reaction of any plasmodia stage of *M. costalis* with the same conjugate, since the reactions with the prespore stages are identical to those with the plasmodial stages in oysters from several geographic areas, and since Couch *et al.* (*3*) have demonstrated morphological bases for describing this as a distinct species, we believe that the foregoing evidence establishes the spore of Couch *et al.* (*3*) as the spore of *Minchinia nelsoni* and serves as further evidence that at least two species of haplosporidian exist in the oyster of the mid-Atlantic estuaries.

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10. We thank the staff of the U.S. Bureau of Commercial Fisheries Laboratory at Oxford, Maryland for assistance; Dr. A. Rosenfield and the staff of the Shellfish Mortality Program provided us with supplies and specimens. Supported by the U.S. Bureau of Commercial Fisheries contracts Nos. 14-17-003-87 and 14-17-003-113. Dr. J. Andrews of the Virginia Institute of Marine Science provided materials for comparative and distributional studies. We thank Mrs. Jamie Barrow, G. Kidder, D. Rintamaa, and D. Case for their assistance.

29 July 1966

Oxygen as a Primary Species in Radiolysis of Water

Abstract. *Recent results are summarized of γ-radiolysis of dilute nitrate solutions at neutral pH in the presence of oxygen and hydrogen as radical scavengers. Complete analysis of the system leads to values of the primary yields showing a net deficit of oxidized products of ~0.4. Analysis of results already available for the oxygen yield shows that oxygen originated from the radiolysis of water with a g value of ~0.1 (g values are derived values for yields of primary species for 100 electron volts). This finding gives material balance and provides evidence of oxygen being a product of water radiolysis.*

Radiolysis of water and aqueous solutions is commonly described in terms of primary species produced in yields independent of the solute (*1*). Well-established primary species are H_2, H_2O_2, OH, H, and e^-, the hydrated electron. However, recent quantitative determinations of these yields indicate a material-balance deficit of 0.6 ± 0.2 based on $\Sigma\, g(\mathrm{OH}) + 2g(H_2O_2)$ (*2*) (*g* signifies derived values for yields of primary species for 100 ev; *G* signifies the experimentally measured yield of product for 100 ev absorbed energy). To account for this deficit, Allen (*3*) suggested that there may be another, hitherto unnoticed, oxidizing species produced in water radiolysis, perhaps the oxygen atom. We now report experimental evidence that oxygen is a product of water radiolysis and indicating that it may originate as O atoms.

Recent work (*4*) on the γ-radiolysis of dilute nitrate solutions enables one to propose a mechanism that completely accounts for the experimental observations. The mechanism is based on the reduction of NO_3^- to NO_2 by e^- and H, NO_2 undergoing dismutation to yield NO_2^-; OH radicals reoxidize NO_2^- to NO_2. In the absence of added scavengers, $G(NO_2^-)$ is essentially given by $\frac{1}{2}\,(ge^- + g\mathrm{H} - g\mathrm{OH})$. Molecular hydrogen acts as an OH scavenger, the nitrite yield then becoming $\frac{1}{2}\,(ge^- + g\mathrm{H} + g\mathrm{OH})$. This aspect of the mechanism is confirmed by our evaluation of the rate-constant ratio: $k(\mathrm{OH} + \mathrm{H}_2):k(\mathrm{OH} + \mathrm{NO}_2^-) = 0.8 \times 10^{-2}$. Determination of $g(e^-)$ is based on the competition between oxygen and nitrate for the solvated electron, for which we obtain $k(e^- + \mathrm{O}_2):k(e^- + \mathrm{NO}_3^-) = 2.5$. The mechanism thus involves no unusual spe-

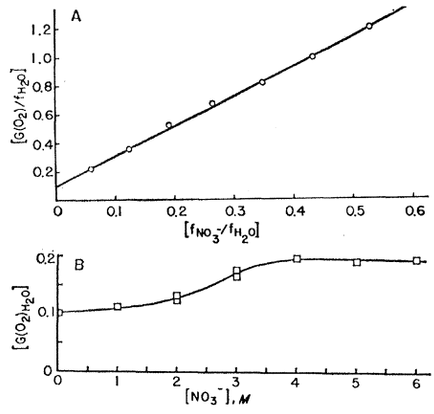

Fig. 1. *A*, G of total oxygen evolution as a function of energy fractionation between water and nitrate. *B*, G of O^{18}-labeled oxygen from the radiolysis of solutions enriched in H_2O^{18}, as a function of nitrate concentration.

cies or reactions. Detailed considerations (*4*) of the results enable us to determine the following values for primary product yields:

$$ge^- + gH = 3.41 \pm 0.23;\ gOH = 2.53 \pm 0.17;\ gH_2O_2 = 0.75$$

which, together with $g(H_2) = 0.44$ (*5*), give 4.3 for the sum of reducing species and 4.0 for the oxidizing species. Apparently a material-balance deficiency of ~0.3 exists.

However, another set of experimental data is available for this system. Mahlman (*5*) has reported G values for oxygen formation over the concentration range 1.0 to 7.0*M* NO_3^-. Although one may expect that most of this oxygen results from energy deposition in the nitrate ion, it is by no means obvious that all the oxygen originates in this way. Accordingly we have treated these data by the following relation:

$$G(O_2) = G(O_2)_{H_2O} f_{H_2O} + G(O_2)_{NO_3^-} f_{NO_3^-}$$

where f_{H_2O} and $f_{NO_3^-}$ are the fractions of energy deposited in the water and nitrate ion, respectively, and $G(O_2)_{H_2O}$ and $G(O_2)_{NO_3^-}$ represent the oxygen yields from water and nitrate. Figure 1*A* shows that the data fit this linear funtion well, and we obtain a value $G(O_2)_{H_2O} = 0.1$ from the intercept at infinitely dilute solution, which, within the limits of experimental error, is the amount needed to remove the material-balance deficit.

The unusual nature of this conclusion —that oxygen is produced from water in the radiolysis of dilute nitrate solutions—requires that other interpretations of these data be considered. Thus there may be a systematic error in the O_2 analysis that is not found in the (simultaneous) H_2 analysis; this error must also be linearly related to $f_{NO_3^-}$: f_{H_2O}, which we consider to be somewhat unlikely. Also, extrapolation of the linear relation to ~0.1*M* may not be valid; the relations may possibly change near the origin.

However, further evidence that the oxygen does in fact originate from the water is explicitly obtained by isotope methods. Mahlman, using 1.6 percent H_2O^{18}, has presented results (*6*) from which $G(O_2)_{H_2O}$ may be obtained as a function of NO_3^- concentration [Fig. 1*B*; the value of $G(O_2)_{H_2O}$ at $(NO_3^-) = 0$ is taken from the intercept of Fig. 1*A*]. Even in quite concentrated nitrate solutions, oxygen is produced from water, and the variation of production with nitrate concentration indicates that it is formed with a G of ~0.1 in dilute solution. We believe this to be clear evidence of oxygen being a product of water radiolysis.

The state of the oxygen on formation is not apparent, but we point out that the variation of $G(O_2)_{H_2O}$ with NO_3^- concentration can be nicely accounted for as a transition from the reaction $2O \rightarrow O_2$ in dilute solution to $O + NO_3^- \rightarrow O_2 + NO_2^-$ in concentrated solution; this suggestion implies, of course, that the oxygen originates in atomic form. Comparison with other (*7*) recently determined stoichiometries is interesting: Hochanadel, using the CO + O_2 system, finds $g(OH) = 2.59$ and $gH_2O_2 = 0.72$, in essential agreement with our results; he also finds stoichiometry with

$$\overset{\text{ox.}}{\Sigma} = 4.03 = \overset{\text{red.}}{\Sigma}$$

Dainton *et al.* (*8*), working with the identical system, also find stoichiometry, but their individual G values differ considerably from Hochanadel's. Seddon and Sutton (*9*) find stoichiometry in the NO system, but their evaluation is gOH = 2.9, as is Fielden's (*10*) in the MnO_4^-:HCO_2^- system; this value of gOH coincides with the sum of our gOH + g O_2 (in equivalents).

Thus the situation concerning the primary species in neutral solutions must still be regarded as unsettled. It may well be true that O atoms may be measured in most systems as the stoichiometric equivalent in OH radicals; or they may appear as O_2—which may not be expected. The scavenger systems used to determine primary species must be carefully evaluated for specificity.

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5 May 1966

Phosphorylase Kinase of the Liver: Deficiency in a Girl with Increased Hepatic Glycogen

Abstract. *Studies of a child with glycogenosis revealed an increased concentration of glycogen and low phosphorylase activity in her liver. Using mixtures of homogenates of the patient's liver and of normal liver, we found the low phosphorylase activity to be caused by a deficiency of phosphorylase kinase and not of hepatic phosphorylase. The fact that phosphorylase activity was restored to normal values by the addition of phosphorylase b kinase from rabbit muscle substantiates this conclusion.*

Since Hers (*1*) reported studies of two patients with low activities of hepatic phosphorylase and elevated concentrations of glycogen in the liver, a decrease in the activity of phosphorylase has come to be equated with a deficiency in hepatic phosphorylase (Type VI glycogenosis) (*2*). Such an interpretation does not take into account the complexity of the phosphorylase system (Fig. 1) which comprises at least three other enzymes (*3*).