Free Rad. Res. Comms., Vol. 5, No. 2, pp. 129–130
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ERRATUM

Page 234 of the paper by Czapski *et al.* (*Free Rad. Res. Comms* **4**, 231–236, 1987) should read as follows:

In several studies it has been demonstrated that some Cu(III) compounds, especially peptide compounds, the antibiotic drug bleomycin, and Fe(IV) aminopolycarboxylate complexes undergo intramolecular electron transfer reactions yielding degraded ligand.[43–49] It has already been demonstrated that O_2^- oxidizes Mn(II) compounds to either Mn(III) or MnO_2^+, depending on the conditions[40,50–52] and that these species can, in turn, oxidize other targets.[53] Fridovich and co-workers studied the effect of vanadate on the oxidation of NAD(P)H by O_2^-, and it appears that an oxidant is formed in the reaction between vanadate and O_2^-, which then oxidizes NAD(P)H.[54–56] In these systems SOD inhibited the vanadate-stimulated oxidation of NAD(P)H, while catalase had no effect.[55,56]

This mechanism explains the uniqueness of O_2^- as compared to other reducing entities. It assumes that O_2^- toxicity originates from O_2^- oxidative properties and therefore the other biological reductants cannot substitute O_2^- in the mechanism, and they are not toxic agents. This mechanism, of course, accounts for the protective role of SOD and the protective role of the chelators in some cases and their sensitizing effects in other cases. Ligands of iron, copper or manganese may change the redox potential of the central metal cation, and thus may change both the feasibility of reactions (6) or (6a) as compared to reactions (3) or (3a), as well as the relative rates of these reactions.

The mechanism involving Cu(III), Fe(IV) or Mn(III) may also explain the inability of OH· scavengers to exhibit protection in several cases, as Cu(III), Fe(IV) and Mn(III) are expected to react with OH· scavengers with different relative rates yielding different products as compared to OH· radicals. In some cases Cu(III) or Fe(IV) may dissociate to yield OH· radicals and one would expect that OH· scavengers would protect the systems, but relatively high concentrations of these scavengers would be needed as compared to the case where OH· is formed directly.

What is not clear enough is the role of H_2O_2 in these processes, as in many systems catalase as well as metal chelators have protective effects, although there are many cases where they do not inhibit the damage.[33,54–57] It is plausible that $M^{(n+1)+}$ forms with H_2O_2 complexes which are probably very potent oxidizing, and therefore toxic, agents. It is possible that in some systems the damage occurs partially through $M^{(n-1)+}$ (Haber-Weiss Mechanism) and partially through $M^{(n+1)+}$. In such a case the role of H_2O_2 is clear, but one should observe only partial inhibition of the damage by catalase.

CONCLUSIONS

We conclude that in systems where O_2^- is unique in causing damage as compared to other biological reductants, it may not reduce the metal ion but rather oxidizes it to form a highly oxidizing entity. This entity might decompose either via an intramole-

cular electron transfer reaction causing degradation of the ligand attached to the metal or it might react directly with the biological target. However, this proposed mechanism does not eliminate the formation of OH· radicals or other oxidizing species through the Haber-Weiss reaction catalyzed by metal ions. The latter case accounts also for damage initiated by some other reductants, while the first one does not. It is possible that in some systems the damage occurs through the Haber-Weiss mechanism and in others through our proposed mechanism or through both mechanisms.