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UNUSUAL MYOGLOBIN OF ELEPHANT

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ABSTRACT: Myoglobins are proteins found in muscle fibers and they store and carry oxygen. They also bind carbon monoxide (CO). Myoglobins of Loxodonta africana and Elephas maximus are different from myoglobins of most other mammals. Most significantly elephant myoglobins react with CO nearly eight times more strongly than other myoglobins. This means that elephants housed close to expressways (where emission of CO from motorvehicles is greatest) would be affected by the toxic gas more than other animals would. On the other hand, elephant myoglobin resists oxidation to a greater extent than normal myoglobins and, thus, it is more stable to actions of certain toxins.

Some time ago Dr. Alex Romero-Herrera of the Department of Anatomy, Wayne State University Medical School, told us about pink myoglobin (muscle hemoglobin) extracted from African elephant muscle. We shared Alex's bewilderment about pink myoglobin, since myoglobin extracted from other animals appears as dark as a turkey's dark meat on a dinner table. In order to solve the mystery, Alex began amino acid sequencing and we started the functional analysis of the pink myoglobin.

It is well known that in the amino acid sequence of a protein some amino acids are crucial and are evolutionary conserved. If any of these amino acids are mutated, the function of the protein could be impaired and the organism itself may not survive. The so called "distal histidine" of myoglobin and hemoglobin (oxygen-carrying pigment of the red blood cell) is one of those key amino acids. The name distal is given in contrast to proximal histidine, which is in close contact with the heme (iron containing part of the hemoglobin). The distal histidine, on the other hand, is placed directly opposite the heme of the hemoprotein at such distance where an oxygen molecule can be sandwiched between them. Under naturally occuring processes, in hemoglobin, if the histidine is substituted with other amino acids such as tyrosine or proline, the protein loses its capacity to bind oxygen. Although little is known as to the substitution at this site in myoglobin, a similar mutation could be catastropic for any organism. According to Alex, however, the histidine is indeed replaced with glutamine (another point mutation) in elephants and a few other animals. Despite this substitution, our study has shown that the interaction of elephant (African, Loxodanta africana; and Asian, Elephas maximus) myoglobin with oxygen is almost the same as that of other myoglobins which have distal histidine, such as whale and human myoglobins. Substitution of distal histidine with glutamine has never been reported for human hemoglobin, but has been reported for opposum hemoglobin. In addition, we have also discovered that the auto-

oxidation rate of elephant myoglobin is slower than that of other myoglobins. These observations explain why elephant muscle receives sufficient oxygen despite the substitution at the distal histidine site and why its myoglobin is maintained in a reduced pink form during the process of extraction while auto-oxidation might take place. Myoglobin becomes dark brown when it is oxidized.

An additional change became evident when we compared the reaction of carbon monoxide (CO) with the myoglobins. It is already known that the binding of carbon monoxide to myoglobin is much stronger than that of oxygen. Carbon monoxide poisoning arises from replacing bound oxygen with carbon monoxide. We have found that, in elephant myoglobin, its binding with carbon monoxide is about 8 times stronger than that of other myoglobins (Fig. 1). Carbon monoxide gets into elephant myoglobin faster than other myoglobins and once there it will come out at a much slower rate. In other word, elephants are much more sensitive to carbon monoxide than we are.

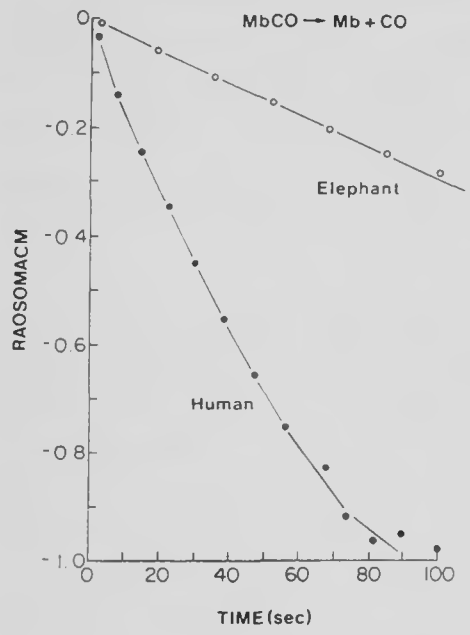

Figure 1. RAOSOMACM = Relative amount of separation of myoglobin (Mb) and carbon monoxide (CO) from the parent molecule MbCO. The amount of separation increases downwards on the vertical scale. This graph, therefore, provides evidence that elephant myoglobin release the CO part of the MbCO molecule more slowly than human myoglobin.

This uniquely altered protein has stimulated much curiosity among scientists who wish to probe the basic chemical and physical nature of ligand (non-covalent) binding to myoglobin and hemoglobin. These phenomena raise interesting questions; could elephants have successfully evolved on this earth had the atmosphere in the early days contained a higher level of carbon monoxide? Does the slower rate of auto-oxidation offer elephants any metabolic advantage? Does bringing elephants into a zoo next to a freeway or a carbon monoxide-producing factory significantly affect their health status? We wish to answer some of these questions in the near future.

Editors' Note: This article has been condensed from:

Bartnicki, D. E., H. Mizukami, and A. E. Romero-Herrera. 1983. Interaction of Ligands with the distal glutamine in elephant myoglobin. J. Biol. Chemistry, 258 (3):1599-1602.

Romero-Herrera, A. E., M. Goodman, H. Dene, D. E. Bartnicki, and H. Mizukami. 1981. An exceptional amino acid replacement on the distal side of the iron atom in proboscidean myoglobin. J. Mol. Evol., 17:140-147.