Red Cell 2,3-Diphosphoglycerate Contents and Oxygen Affinity in Heterozygous Beta-Thalassaemia

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Key Words
2,3-Diphosphoglycerate  
Oxygen affinity  
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Abstract
Red cell 2,3-diphosphoglycerate and whole blood P50 have been found to be significantly higher in β-thalassaemia heterozygotes than in normal controls. Such increases could be considered as central mechanisms which compensate the mild anaemia of β-thalassaemia carriers.

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Introduction
In different haemolytic anaemias, red cell contents of 2,3-diphosphoglycerate (2,3-DPG) increase [3]. The resulting rise of P50 values (PO2 at 50% saturation) induces a greater removal of oxygen by the tissues. Since heterozygous β-thalassaemia is in many cases associated to some degree of haemolytic anaemia [4], we have investigated the red cell 2,3-DPG contents and P50 levels in a group of β-thalassaemia carriers compared with normal controls.

Materials and Methods
We studied 30 symptom-free β-thalassaemia heterozygotes of both sexes (age: 18–45 years) and 20 normal controls of comparable age. The diagnosis of heterozygous β-thalassaemia was established on the basis of abnormal red cell morphology, decreased osmotic fragility, and increased Hb A2 level. Red cell indices were obtained from Hemalog 8 counter. The red cell 2,3-DPG levels were determined with enzymatic assay (Sigma). Whole blood P50 was measured using the mixing technique [2].

Results
The results obtained are summarized in table I. In β-thalassaemia heterozygotes the mean value of 2,3-DPG (21.1 μmol/g Hb) was significantly higher than in controls (15.4 μmol/g Hb); likewise, the mean level of the whole blood P50 was higher in the thalassaemia group (27.1 mm
Hg) than in the controls (25.8 mm Hg). In both groups no close correlation between 2,3-DPG contents and P50 values was observed.

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Table I. Mean values of blood haemoglobin, red cell 2,3-DPG and whole blood P50 in thalassaemia carriers and normal controls

Discussion

The significant increase of mean red cell 2,3-DPG and whole blood P50 levels in heterozygous 

α5-thalassaemia suggests that also in this condition an anaemia-compensating central mechanism takes place. This consists of a 2,3-DPG mediated increase of whole blood P50 levels; that is, a shift to the right of the oxygen dissociation curve. Pearson et al. [5] reported in α-thalassaemia carriers increased contents of 2,3-DPG, but normal P50 levels. However, their study lacked their own controls.

In our study the failure to have found a close correlation between 2,3-DPG and P50 values is not surprising, since changes in 2,3-DPG do not necessarily lead to changes of P50, because of the interference of various factors, such as ATP, pH, and temperature [1].

References


