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DEVELOPMENTAL HEMATOLOGY OF SS AND SC DISEASE IN ASSOCIATION WITH α-THALASSEMIA-2. A.E. Felice, E.M. Marino*, K.M. McKie*, and V.C. McKie*. Comprehensive Sickle Cell Center, Departments of Cell and Molecular Biology and Pediatrics, Medical College of Georgia, Augusta, GA.

The nuber and organization of the α, ζ and γ globin genes have been determined on the DNA of 400 patients attending the Pediatric Sickle Cell Clinics of our Center. The patients were participating in long-term prospective studies on possible effects of α-thal on the hematological changes accompanying postnatal development. CBC and Hb composition were obtained in the steady state on patients of different ages and correlated with the number of α globin genes. All patients included in this study had SS or SC, and normal γ and ζ genes but differed in the number of α genes due to normal α genotypes (αα/αα); α-thal-2 heterozygosity (-α/αα); and homozygozity (-α/-α). The type I or type II, -3.7 Kb α-thal-2 had the following gene frequencies; SS: 0.18; SC: 0.19. Neither the -4.2 Kb α-thal-2, nor the type III -3.7 Kb α-thal-2 have been observed in our patients. For both SS and SC patients, the gene frequency was invariant in the first two decades of life. Hematological analyses revealed complex interactions between α-thal, Hb F and development among SS but not SC patients. Although the MCV and RBC values of both SS and SC children gave the expected microcytosis and erythrocytosis due to α-thal, the Hb levels of the SS patients with αα/αα, -α/αα or -α/-α were the same up to the age of 5 to 7 yrs. Older children with SS -α/-α had higher Hb levels than the SS -α/αα which in turn were higher than those of the SS αα/αα. This difference emerged only after the proportion of Hb F declined to levels under 15%. The Hb F levels of the SS patients with 4, 3, or 2 α genes were similar after 7 yrs while for the SC patients, the values were similar after 3 yrs. The MCHC of the SS; αα/αα patients under 5 yrs of age could be as low as that of the SS -α/αα or SS; -α/-α. This might be due to asynchronous development of the MCH and MCV values in young children with SS and different numbers of α globin genes.

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