

**MICROPARTICLES IN SICKLE CELL DISEASE
AND THALASSEMIA MAJOR IN RELATION TO
HYPERCOAGULABLE STATE AND VASCULAR
COMPLICATIONS**

Thesis

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LIST OF ABBREVIATIONS

Abbrev.	Full term
ACS.....	Acute chest syndrome
AOD.....	Aortic diastolic diameter
AOS	Aortic systolic diameter
apl	Antiphospholipid antibodies
BM	Bone marrow
DBP.....	Diastolic blood pressure
ECs.....	Endothelial cells
ELISA	Enzyme linked immunosorbant assay
EMPs.....	Endothelial microparticles
ErMPs	Erythrocyte microparticles
G6PD	Glucose 6 phosphate dehydrogenase
Hb AS	Sickle Cell Trait
Hb F	Fetal Hemoglobin
Hb	Hemoglobin
HbA2.....	Hemoglobin A2
HbS	Hemoglobin S
HBV	Hepatitis B virus
HCV	Hepatitis E virus
HU.....	Hydroxyurea
LDH	Lactate Dehydrogenase
LV	Left Ventricle
MCH	Mean Corpuscular Hemoglobin
MCHC.....	Mean Corpuscular Hemoglobin Concentration
MPs.....	Microparticles
NO	Nitric oxide
PH	Pulmonary Hypertension
PMPs.....	Platelet microparticles

LIST OF ABBREVIATIONS (Cont...)

Abbrev.	Full term
PP	Pulse pressure
PRBC	Packed Red Blood Cells
PS	Pulmonary stenosis
SBP	Systolic blood pressure
SCCLD	Sickle cell chronic lung disease
SCD	Sickle cell disease
TF.....	Tissue factor
TM	Thalassemia major

INTRODUCTION

Thalassaemia and sickle cell disease (SCD) represent the most common forms of hereditary haemolytic anaemia and result from a partial or complete lack of synthesis of one of the major α - or β -globin chains of haemoglobin A or from a single amino acid mutation of the β -globin chain, respectively (*Weatherall, 2001; Fucharoen and Winichagoon, 2002; Ataga et al., 2007*). Unmatched globin chains are less stable and bind to the cytoplasmic surface of the red blood cell (RBC) membrane where they produce oxidative damage, which might be partly responsible for the membrane rigidity (*Schrrier, 2002*) with increased aggregability of RBCs (*Helley et al., 1996; Pattanapanyasat et al., 2004*).

SCD is characterized by chronic hemolysis and recurrent ischemia due to micro-vascular occlusion following the adhesion of erythrocytes and leukocytes to the vascular endothelium (*Stuart and Nagel, 2004*). Increased risk of cardiovascular disease has been reported in SCD and thalassemia (*Michaeli et al., 1992; Morris et al., 2003; Acar et al., 2003*). An abnormal response after transient arterial occlusion has been reported in homozygous sickle cell anemia (*Belhassen et al., 2001*). Structural and functional changes of the arteries are important features in cardiovascular disease (*O'Rourke, 1995*). These structural changes may translate functionally into alteration of arterial stiffness in vivo. Arterial stiffness is an important mechanical property, because it is related to vascular impedance