Redox proteomics in *Plasmodium falciparum:* Differential oxidatively modified proteins across intra-erythrocytic stages and chloroquine-treated parasites

Azar Radfar[1], Amalia Diez[1], Antonio Serna-Sanz[2] and José M. Bautista[1]

[1]Departamento de Bioquímica y Biología Molecular IV and [2]Unidad de Proteómica, Universidad Complutense de Madrid. Ciudad Universitaria. 28040 Madrid, Spain.

Plasmodium falciparum is the causative agent of the most virulent form of human malaria. Oxidative defence mechanism in *P.falciparum* seems to be a target of chloroquine (CQ) action and in this work, our objective has been to apply a redox proteomic approach to characterize and compare oxidatively modified proteins of the different intra-erythrocytic stages of chloroquine treated and non-treated Dd2 resistant parasites. Protein carbonyl detection as a measure of oxidative damage revealed that important groups of proteins such as molecular chaperons, enzymes of the glycolytic pathway and proteases became modified. Interestingly, some of these targets were already identified as oxidatively modified proteins in other eukaryotes. The vast majority of identified carbonylated proteins were of plasmodial origin (79%). Strikingly, 9 out of the 31 plasmodial proteins belong to chaperon families. Moreover, chloroquine increased protein oxidation mostly in mature stages. In CQ treated schizonts oxidative signal was stronger than in treated rings. The Hsp70 family remained the most oxidized proteins in all treated stages but particularly in the mature ones. Human annexin A7 was identified as one of the most oxidized proteins among identified spots in the ring stage treated cultures. Also, human carbonic anhydrase was also a major carbonylated spot in both trophozite and schizont treated cultures. Using this proteomic approach, we have identified those proteins that are specially susceptible to oxidative modification. The results presented will be discussed with regard to the importance of such proteins in the life of *P. falciparum* and in the disease development.