

(NADPH) of the enzymes. Platelets showed a reduction was 2.1 ± 0.2 nmol/min/mg of protein. In addition, surface NADH oxidation appeared (min/mg of protein). It is noteworthy that these results. Indeed, NADH oxidation was explained in terms of parallel pathways or might move inside to reduce intracellular NADH. To assess redox state of platelet membrane, we assessed reduction of NADH. Capsaicin has been recognized as a specific inhibitor of NADH reduction. Capsaicin was partially inhibited ($20 \pm 3\%$), whereas the other two were not. The first one member of the Ecto-NOX family is responsive to capsaicin. In the meanwhile, the other two are not responsive to NADH oxidase. To identify which Ecto-NOX is responsible for the reduction of NADH, we tested with the molecular weights corresponding to the three Ecto-NOX. We expressed both Ecto-NOX1 and Ecto-NOX2 in HEK293 cells. The results showed that Ecto-NOX1 is responsible for the reduction of NADH.

TRPV1 receptor 1 (TRPV1) and