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## Health improvement of HIV-1 infected patients treated by Immunorex TM: Lessons learned in 40 years on monoamine oxydase and DHEA metabolism research

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This lecture is to share our 40 years of experience on metabolism of monoamine oxydase and stress in rat model, followed by studies on DHEA and related peptides hormones ininflammatory conditionswhich leaded to Immunorex$_{TM}$. It can restore and prevent not only the $TH_1$ $TH_2$ deregulation focussing on IL 10 suppression known to enhance growth and dissemination of both HIV-1 and co-infected pathogens and to provoke cardiovascular and cerebrovascular diseases, but is also able to block overproduction of IL6 and IL10 suppressing B cells lymphomas. Consequently it restore, $TH_1$ and NK cells to treat and prevent cell malignancies. Immunorex$_{TM}$ demonstrated its capacity to block HIV-1 host cell entry through the restriction of the envelop proteins mediating cell-cell fusion (gp 120/gp 41) by inhibiting phosplolipase $A_2$ ($PLA_2$) activation suppressing new mutants resistance clinical isolate. Health improvement of HIV-1 infected patients was described by Maka et al, 2009; while in more recent datas, Immunorex$_{TM}$ induced antibody dependent cellular cytotoxicity mediated by specific anti-VIH-1 IgG known to be implicated in vaccine induced immunity. The capacity of Immunorex$_{TM}$ to control oxidative stress by inhibition of NADPH activation was also shown. Immunorex$_{TM}$ showed its capacity to improve health in cases of prostatic and of laryngeal cancer and inone case of chronic myeloid leukemia attributable not only to DHEA but also to the presence of salicin and threonine in Immunorex$_{TM}$ composition.

### Biography

Donatien Mavoungouis the Director of the Research Center on Hormonal Pathologies (CRPH) Gabon. He has authored more than 150 publications. He discovered IMMUNOREX$_{TM}$ DM28 inhibiting HIV-1 replication. He is Professor of Biochemistry and Endocrinology at the University of Health Science Libreville, Gabon. He received the Prize of the National Center for Scientific Research in 2009 and 2012. (Gabon).

## Ribosomal protein S6 kinase 1 regulates polyploidization of SP600125-induced megakaryocytic cell lines through phosphorylation at Thr421/Ser424 and dephosphorylation at Thr389

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Megakaryocytes (MKs) are one of the few cell types that become polyploidy**.** The mechanisms by which cells decide to become polyploidy are not fully understood. In this investigation, using Dami and CMK cells induced with SP600125, we successfully established relatively synchronized polyploid cell models with polylobulated nuclei, which was similar to the physiological polyploidization of primary MKs. We found that SP600125 induced polyploidization of Dami and CMK cells in concomitance with phosphorylation of ribosomal protein S6 kinase 1 (S6K1) at Thr421/Ser424 and dephosphorylation of S6K1 at Thr389. SP600125-induced polyploidization of Dami and CMK cells was partially blocked by H-89, a cAMP-dependent protein kinase (PKA) inhibitor, through binding directly to S6K1, leading to dephosphorylation at Thr421/Ser424 and phosphorylation at Thr389 of S6K1, independent of PKA. LY294002 partially blocked polyploidization of SP600125-induced Dami and CMK cells, although it markedly increased the phosphorylation of protein kinase B (Akt). This effect of LY294002 was enhanced by overexpression of a S6K1 mutant containing mutations of Ser411, Ser418, Thr421 and Ser424 to Asp, Asp, Glu and Asp respectively, but not S6K1 mutants with Thr389 to either Glu or Ala. In addition, PD184352 did not inhibit polyploidization of SP600125-induced Dami and CMK cells, although it abrogated the phosphorylation of p44/42MAPK. In contrast, U0126 inhibited the polyploidization of SP600125-induced Dami and CMK cells, although it did not abrogate the phosphorylation of p44/42MAPK. Our results suggest that SP600125 induced polyploidization of Dami and CMK cells through coordinated phosphorylation of S6K1 at Thr421/Ser424 and dephosphorylation at Thr389, in synergism with other signal pathways, independent of MAPK and Akt.