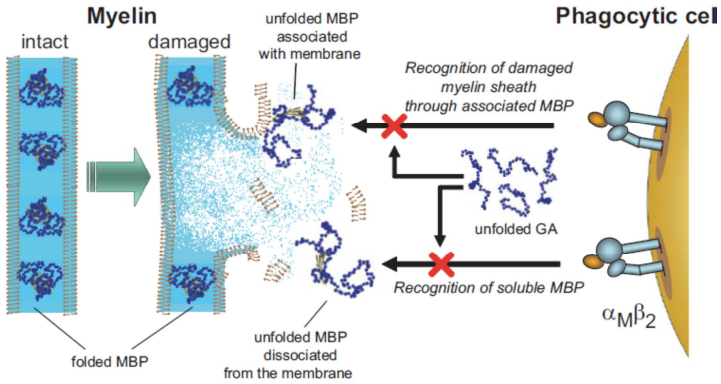
# On the biology of beta-2 (CD18) integrins and their role in neural inflammation

*Thomas Vorup-Jensen, Ph.D.*

*Department of Biomedicine, Aarhus University, Denmark*

*vorup-jensen@microbiology.au.dk*
*http://biomed.au.dk/forskning/forskere-og-forskningslaboratorier/u-aa/vorup-jensen-thomas/*




Beta-2 integrins are cell adhesion molecules expressed on most leukocyte subsets. The family of integrins with a common $\beta_2$ (CD18) chain includes $\alpha_L\beta_2$ (also known as LFA-1 or CD11a/CD18), $\alpha_M\beta_2$ (Mac-1, CD11b/CD18, or complement receptor 3), $\alpha_X\beta_2$ (p150,95, CD11c/CD18, or complement receptor 4), and $\alpha_D\beta_2$ (CD11d/CD18). CD18 integrins play a crucial role in the immune system, underscored by the clinical observation that failure to express the CD18 chain leads to leukocyte adhesion deficiency severely compromising immune protection. The classic representation of these molecules has focused on their cell-surface expression and role in both leukocyte migration and diapedesis through the blood vessel barrier into zones of inflammation. An emerging literature points to that soluble integrin complexes are also part of the inflammatory response in human disease. The $\beta_2$ integrins are shed from the cell membrane by the proteolytical activity of matrix-metallo proteinase-9. Combined, these processes contribute to the development of inflammation in number diseases, including inflammatory responses in the brain. The presentation will address some of mechanisms as well as the current therapies limiting the inflammatory response in neural inflammation through manipulation of $\beta_2$ integrin function.