Disclosure notice in SynAct Pharma AB

SynAct Pharma AB ("SynAct" or the "Company") hereby announces that the Company's shareholders TJ Biotech ApS (wholly-owned by the board member and CSO Thomas Jonassen) and Quantass ApS (owned by the CEO Jeppe Øvlesen and his wife Ghita Øvlesen) have carried out a corporate restructuring whereby the companies' holdings of shares in SynAct have been transferred through a merger to the newly formed company BioInvest ApS.

Prior to the restructuring, TJ Biotech ApS held 2,296,651 shares, corresponding to approximately 9.41 per cent of the number of shares and votes in the Company, and Quantass ApS held 1,456,263 shares, corresponding to approximately 5.97 per cent of the number of shares and votes in the Company. Following the restructuring, BioInvest ApS holds 3,752,914 shares, corresponding to approximately 15.38 per cent of the number of shares and votes in the Company, meaning that BioInvest ApS through the merger has taken over all shares and votes in the Company previously owned by TJ Biotech ApS and Quantass ApS. BioInvest ApS is a newly formed company, which is jointly owned by Thomas Jonassen 61 per cent) as well as Jeppe Øvlesen and his wife Ghita Øvlesen 39 per cent. BioInvest ApS did not own any shares in the Company prior to the restructuring. Existing lock-up agreements has been taken over by BioInvest (Thomas Jonassen, Jeppe Øvlesen, Ghita Øvlesen).

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About SynAct Pharma AB

SynAct Pharma AB conducts research and development in inflammatory diseases. The company has a platform technology based on a new class of drug candidates aimed at acute deterioration in chronic inflammatory diseases with the primary purpose of stimulating natural healing mechanisms. SynAct Pharma is listed on the Spotlight Stock Market (ticker: SYNACT). For more information, please visit https://synactpharma.com.

About AP1189

The mechanism of action of SynAct Pharma's lead compound AP1189 is to promote resolution of inflammation through melanocortin receptor activation directly on macrophages, thereby reducing the pro-inflammatory activity of macrophages and by stimulating so-called macrophage efferocytosis, a specific ability to clear inflammatory cells (J Immun 2015, 194:3381-3388). This effect has shown to be effective in disease models of inflammatory and autoimmune diseases and the clinical potential of the approach is currently tested in a clinical phase 2 study in patient with active rheumatoid Arthritis, in nephrotic syndrome and COVID-19 inflammation ARDS.
https://clinicaltrials.gov/ct2/show/NCT04004429?term=AP1189&draw=2&rank=1)
https://clinicaltrials.gov/ct2/show/NCT04456816?term=AP1189&draw=2&rank=2