We examined the trafficking and signalling of WT-MOPr in comparison with that of a recently identified naturally occurring variant (L83I) in HEK293 cells. Using ELISA, DAMGO induced a similar extent of internalization of both the WT-MOPr and the L83I variant. In contrast, morphine induced significant internalization of the L83I variant but had little effect on the WT-MOPr. Inhibition of dynamin function with dynasore inhibited the DAMGO-induced internalization of both the WT-MOPr and the L83I variant and the morphine-induced internalization of the L83I variant. Expression of DNM GRK2 attenuated the internalization of the L83I variant in response to both DAMGO and morphine, in addition to inhibiting the WT-MOPr response to DAMGO. Following immunoprecipitation of MOPr, DAMGO induced substantial phosphorylation of Ser$^{375}$ in both receptors, morphine induced far less phosphorylation, but morphine-induced phosphorylation was the same for WT-MOPr and L83I. We were able to detect co-immunoprecipitation of arrestins with the DAMGO- but not morphine-activated receptors. Investigations of cAMP signalling revealed no significant change in the EC$_{50}$ of either DAMGO or morphine when compared to values obtained for the WT-MOPr. These results show that the L83I variant rapidly internalizes in response to morphine in a GRK- and dynamin-dependent manner. The enhanced internalization of L83I in response to morphine is not due to increased phosphorylation of Serine$^{375}$, increased ability of MOPr to interact with arrestins, or an increased ability to signal via G-proteins.