The A<sub>2A</sub>-adenosine receptor is a G<sub>s</sub>-coupled receptor with an extended carboxyl terminus of more than 120 amino acids. Heterologous expression of affinity-tagged A<sub>2A</sub>-receptor in various cell types results in the intracellular retention of a large portion of the receptor indicating that folding intermediates of the native receptor are recognized by executors of cellular quality control. Using a proteomics approach based on tandem affinity purification (TAP) followed by nano-LC-MS/MS, we identified a number of candidate interaction partners, several of which belong to the class of heat-shock proteins (i.e., HSP90 and HSP70). These are thought to act as molecular chaperones. Accordingly, our working hypothesis posits that binding of the chaperones to the C-terminus assists in the folding of the receptors and control its fate. In the folded state, the chaperones are released, the receptor recruits the coat-protein complex-II machinery to the C-terminus and is exported to the cell surface. In contrast, tight interaction with folding intermediates of the A<sub>2A</sub>-receptor may direct these to degradation. We verified an interaction of HSP90 and HSP70 with the A<sub>2A</sub>-receptor by co-affinity-precipitation. Furthermore, treatment with HSP90-inhibitors led to a significant increase in surface expression of the receptor. Concomitantly, the fraction of HSP70-bound receptor increased. The fraction of HSP70-bound receptor also increased upon treatment of cells with kifunensine to inhibit of entry into the ER-associated degradation pathway. Taken together, these data are consistent with an HSP-relay system that prevents premature ER export of partially folded A<sub>2A</sub>-receptors.