Why are chemotaxis receptors clustered but other receptors aren't?

The chemotaxis network of bacteria such as E. coli is remarkable for its sensitivity to minute relative changes in chemical concentrations in the environment. Indeed, E. coli cells can detect concentration changes corresponding to only ~3 molecules in the volume of a cell. Much of this acute sensitivity can be traced to the collective behavior of teams of chemoreceptors on the cell surface. Instead of receptors switching individually between active and inactive configurations, teams of 6-20 receptors switch on and off, and bind or unbind ligand, collectively.Similar to the binding and unbinding of oxygen molecules by tetramers of hemoglobin, the result is a sigmoidal binding curve. Coupled with a system for adaptation that tunes the operating point to the steep region of this sigmoidal curve, the advantage for chemotaxis is gain i.e., small relative changes in chemical concentrations are transduced into large relative changes in signaling activity (specifically, the rate of phosphorylation of the response regulator CheY). However, something is troubling about this simple explanation: in addition to providing gain, the coupling of receptors into teams also increases noise, and the net result is a decrease in the signal-to-noise ratio of the network. Why then are chemoreceptors observed to form cooperative teams? We present a novel hypothesis that the run-and-tumble chemotactic strategy of bacteria leads to a "noise treshold", below which noise does not significantly decrease chemotactic velocity, but above which noise dramatically decreases this velocity.