An earlier study from our group used a 1000-cell biophysical model of a rodent lateral amygdala and provided a preliminary explanation of how and why certain neurons might be recruited into a fear memory trace after Pavlovian fear conditioning. In the present model we extended the work to investigate the role of specific mechanisms in this recruitment, including intrinsic excitability of cell, afferent tone and shock, neuromodulator receptors, and intrinsic excitatory and inhibitory connections. We first proposed an improved criterion to define ‘plastic’ cells after recognizing that the Repa criterion used to classify plastic cells favored several principal cells with very low firing rates. Using the improved criterion, we were able to replicate the formation of the two distinct Repa cell populations after conditioning. The model suggested that the most important factor was the intrinsic excitability of the cell, i.e., highly excitable cells had a much higher probability of being recruited into the fear memory trace. Although afferent tone and shock were required for a cell to be plastic, the presence of neuromodulator receptors, and the numbers of intrinsic excitatory and di-synaptic inhibitory connections a cell received also played important roles. Finally, we varied the size of the network and internal connectivity among principal cells in the model to study their impact on competition, and found that small networks and reduced connectivity also performed equally well. Plasticity in the inhibitory disynaptic pathway connecting principal cells via interneurons plasticity was found to play a key role in the formation of fear memories.