Developmental axon pruning is a general mechanism required for maturation of neural circuits. During Drosophila metamorphosis, the larval-specific dendrites and axons of early γ neurons of the mushroom bodies are pruned and replaced by adult-specific processes. We show here that the nuclear receptor ftz–f1 is required for this pruning and has two roles: (i) to activate expression of the steroid hormone receptor EcR–B1 whose activity is essential for γ remodeling and (ii) to repress expression of Hr39, a ftz–f1 homologous gene. If inappropriately expressed in the γ neurons, HR39 inhibits normal pruning likely by competing with endogenous FTZ–F1 that results in decreased EcR–B1 expression. EcR–B1 was previously identified as a target of the TGF–β signaling pathway. Here we show that the ftz–f1/Hr39 pathway acts apparently independent from TGF–β signaling, suggesting that EcR–B1 is the target of two parallel molecular pathways acting during γ neuron remodeling.