movement speed following the end of an arrest. These data indicate that NL1-89BLA neural activity is involved in initiating, but not maintaining, arrests.

In the emergence assay, mice began to exhibit arrests in the large, novel arena after their 26th exploratory trip and, generally, the number of arrests rose with the number of trips undertaken. Moreover, mice tended to arrest in previously visited places and in locations where they had previously paused. Interestingly, when mice visited familiar locations where they had previously arrested in the larger arena, the number of NL1-89BLA neurons showing activity and the level of their activity increased. In addition, optogenetic silencing of such neurons when the mice were in specific locations decreased the incidence of arrests in those locations but did not induce place avoidance or preference. These data suggest that NL1-89BLA neurons have a crucial role in the development of experience-dependent arrests in familiar places.

Previous studies implicate the central amygdala (CEA) in regulating movement. As the BLA sends projections to the CEA, the authors examined whether CEA-projecting NL1-89BLA neurons (NL1-89BLA-CEA neurons) could directly induce arrests. They found that most NL1-89BLA-CEA neurons are active during arrests in the emergence assay, and their activity transiently rises during these behavioural episodes. Optogenetic activation of these neurons reduced movement speed, whereas silencing these neurons led to an increase in movement speed. Moreover, optogenetic activation and inhibition of NL1-89BLA-CEA neurons promoted and impaired the development of experience-dependent exploratory arrests, respectively.

Together, these findings reveal a circuit in the amygdala that is important for inducing experience-dependent pauses during exploration in mice.