The neuronal basis of how sexual experience modulates male aggression

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Aggression is vital for animal survival and evolution, but excessive aggression should be suppressed for the sake of individual energy expenditure and social balance. Aggression level is influenced by environment, social experience and internal status. Drosophila melanogaster provides an ideal genetic system to understand the molecular and neuronal basis of different aspects of aggression. Fruitless (fru), a transcription factor that specifies neural circuits governing sexual behaviors, also specifies sex-specific differences of aggressive behavior in Drosophila [1]. A subset of octopamine neurons has been implicated in regulating aggression [2]. Prior social grouping with males reduces male aggression and involves distinct genetic and neural pathways [3,4]. However, it is still not clear whether social contacts with females play any roles in male aggression. Furthermore, the central neural circuits mediating the regulation of aggression by social experience remain elusive. Yuan et al. studied how prior female exposure influences male–male aggression by using powerful genetic tools in fruitfly [5].

Yuan et al. found that naive male flies show increased aggression in the presence of virgin females. However, this increase of aggression by virgin females was inhibited in social experienced males (those that had been housed with females for 24 hours prior to aggression assay). Neither visual nor olfactory mutants could reduce this inhibition effect on sex-related aggression, suggesting that vision or olfaction is not required for this process. Physical contact appears to be indispensable because the inhibition effect on aggression was diminished when the male and female were separated by a nylon mesh during grouping. Among the mutants they screened, the mutants for ppk29, a sodium channel for sensing non-volatile pheromones, impaired the inhibition of aggression. Suppressing the activity of either all the ppk29+ neurons or a subset of ppk29+/fru+ neurons resulted in reduced inhibition of aggression by female exposure. Thus, it is evident that ppk29+/fru+ neurons mediate the chronic non-volatile female pheromone signals to inhibit male–male aggression.

To figure out how chronic female contact exerts its effect on central neural circuits to suppress aggression, Yuan et al. screened for disinhibition phenotype when silencing distinct populations of neurons in the central nervous system. They found that a sexually dimorphic population of 5-hydroxytryptamine receptor 1B (SHT-1B)-expressing neurons mediates the prior female contact-induced inhibition of aggression. Using intersectional strategies, they were able to label and manipulate SHT-1B+/fru+ neurons. Silencing these neurons caused the disinhibition phenotype, while acutely activating these neurons strongly inhibited male–male aggression. Interestingly, female exposure may not act via 5-HT pathway to inhibit aggression, because either elevating 5-HT biosynthesis or altering the expression of SHT-1B receptor does not affect female contact-inhibited aggression. Yuan et al. found a few SHT-1B+/fru+ neurons co-expressed glutamic acid decarboxylase 1 (GAD), an enzyme for synthesizing an inhibitory neurotransmitter γ-aminobutyric acid (GABA).
Genetic intersectional strategies were employed to exclude GABAergic neurons from the 5HT-1B+/fru+ neurons. Either silencing or activating 5HT-1B+/fru+ neurons did not produce disinhibition or aggression phenotype. These results suggest that perhaps only the 5HT1B+/fru+ neurons remain sensitive to female pheromones. Taken together, these results suggest that GABAergic 5HT-1B+/fru+ neurons suppress male aggression by inhibiting downstream RDL+/fru+ neurons. It would be interesting to directly label and manipulate those GABAergic 5HT-1B+/fru+ neurons and examine the behavioral consequences.

This study opens a new avenue to explore how sexual experience modulates aggressive behavior and identifies a set of neural circuits involved in this process. It remains to be elucidated how pheromone signals were relayed from ppk29 neurons to those GABAergic 5HT-1B+/fru+ neurons. For example, are those GABAergic 5HT-1B+/fru+ neurons sensitive to female pheromones? The downstream RDL+ neurons remain to be clearly defined and the functional connectivity between the GABAergic 5HT-1B+/fru+ neurons and RDL+ neurons needs to be characterized. Most interestingly, what is the neural mechanism underlying the temporal transition from promoting aggression by acute female contact to inhibiting aggression by chronic female contact? An understanding of the neural circuits that mediate social modulation on aggression in flies may shed insights into the neural basis of human violence.

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MATERIALS SCIENCE

Science and nanotechnology of superhard materials

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Materials science has made spectacular progress recently in the fields of nanophysics and nanobiology. This is amply demonstrated by the research being undertaken in the area of nanodiamonds. The range and depth of research into nanodiamonds is immense, covering presolar environments such as ‘stardust’, through ultrahard wear-resistant components to an enormous range of applications in biomedical research. What was once a question of: ‘Is there any material harder than diamond’ [1] has now been reframed as, how hard can manufactured nanodiamonds-based materials be? This latter quest for producing such superhard materials is being amply researched by the outstanding research scientists at the State Key Laboratory of Metastable Materials Science and Technology, Yanshan University, Qinhuangdao, China, within the group led by Professor Tian [2–5].

The most recent findings of research into the properties of nanomaterials have shown that both nanotwinned nanodiamonds (nt-diamonds) and nanotwinned nano-cubic boron nitride (nt-cBN) can be manufactured at high pressure and high temperature (HPHT) with the highest hardness values ever reported for such materials [2,3]. Fig. 1 shows the development of the nanostructures, as revealed in high-resolution transmission electron microscopy that occurs when subjecting carbon onion nanoparticles to HPHT conditions of 10 GPa and 1850°C. In addition to transforming the carbon into a new monoclinic diamond phase—M—the nanodiamond grains produced under these conditions are heavily twinned at the nanoscale and have Vickers hardness up to 200 GPa. Further, the fracture toughness (KIC) of this nt-diamond material is extremely high, ranging from 10 to 15 MPa m1/2. For such hardness values, these nanomaterials reverse the usual trend in materials where the higher the hardness the lower the fracture toughness. While this inverse effect has been previously reported in nanocomposites consisting of diamond and nanoparticles of SiC phase, this is the first instance in single-phase nanodiamonds.

A similar story has been developed by Tian’s group in their study of cBN. Beginning with heavily faulted nanoparticles of onion-like BN, bulk samples of nanotwinned, nano grain-sized cBN were produced [3]. Hardness values greater than 100 GPa and exceptionally