Involvement of nicotinic acetylcholine receptor-signaling in the impairment of social behavior in the stressed mice

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[Purpose] We investigated the effect of (-)-nicotine (NIC) on social behaviors in the stressed model mice, and the expression of nicotinic acetylcholine receptor (nAChR) subunits or their intracellular signal-related molecules.

[Methods] The adult male C57BL/6J mice were exposed to forced swimming stress for 15 min. They were treated with NIC (0.3 mg/kg s.c.) for 7 days from the next day of exposure to stress.

[Results] The stressed mice showed the impairment of social behaviors in the social interaction test and the decreased expression of phosphorylated Akt protein in the hippocampus (HIP). These abnormalities were attenuated by repeated treatment with NIC. The expression of $\alpha 7$ and $\beta 2$ nAChR subunit proteins was decreased in the HIP of the stressed mice. $\alpha 7$, but not $\alpha 4\beta 2$ nAChR antagonist blocked the ameliorating effect of NIC on behavioral impairment in the stressed mice.

[Conclusions] These results suggest that repeated treatment with NIC is useful and effectively for stress-induced depressive-like behaviors. The remission of social behavior impairment by NIC may be mediated via regulating nAChR/Akt signaling in stressed mice.