



東北大学脳科学グローバル COE セミナー

(本セミナーは生命科学研究所単位認定セミナー2pt に指定されています)

演題: BRAIN CIRCUITS REGULATING MATERNAL MOTIVATION IN MAMMALS

講師: Prof. Michael Numan

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日時: 2009年6月12日 15:00-16:30

会場: 東北大学(星陵キャンパス) 医学部 5号館 201号室

Numan 博士はラット母性行動の神経機構研究の先達で、視床下部内側視索前野 (MPOA) や線条体、扁桃体などの破壊実験を通じて、母性行動発動に必要な神経回路の解明を 30 年以上にわたりリードしてこられました。本セミナーでは、母性行動研究について基礎からお話いただくとともに、母性行動に中脳ドーパミン系が果たす役割についての最新の成果についてもご紹介いただきます。

<概要>

How does the brain regulate maternal behavior in mammals? For the typical mammal, the hormonal changes which occur near the end of pregnancy act on the brain to facilitate maternal responsiveness toward infants. One important site where hormones act is a hypothalamic region called the medial preoptic area (MPOA). Research shows that the hormones estradiol and prolactin, and the neuropeptide oxytocin, act on the MPOA to stimulate maternal behavior. The neural outputs of the MPOA facilitate maternal behavior in two ways: by depressing circuits derived from the amygdala which give rise to avoidance, withdrawal, and defensive responses, and by exciting neural circuits which activate positive acceptance responses toward young infants. With respect to the latter, MPOA interactions with the mesolimbic dopamine system have been found critical for promoting positive maternal responses. Research on the neural basis of maternal behavior has been performed primarily with rodents or sheep as subjects. However, since maternal behavior is a defining characteristic of mammals, its core neural circuits should be evolutionarily conserved and therefore the rodent and sheep findings are expected to be relevant to the neural basis of parenting in humans. By understanding how the brain normally operates to control maternal behavior we gain insight into how dysregulated maternal circuits might cause faulty maternal behavior. Research on rodents, nonhuman primates, and humans, has shown that when infants are neglected or abused by their mothers they grow up to be poor mothers. Research on rodents indicates that lower levels of maternal care affect the offspring so that amygdala hyperactivity and MPOA hypoactivity occur and this may be the mechanism mediating the development of maternal behavior deficits in the offspring.

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