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► To cite this version:

Helena Janickova, Alice Abbondanza, Sylvie Dumas, Véronique Bernard. Control of behavior through nicotinic acetylcholine receptors expressed by different neuronal populations. 46th Annual Meeting of the Japan Neuroscience Society, Aug 2023, Sendai, Japan. hal-03972990v1

HAL Id: hal-03972990

<https://hal.science/hal-03972990v1>

Submitted on 3 Feb 2023 (v1), last revised 6 Feb 2023 (v2)

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Control of behavior through nicotinic acetylcholine receptors expressed by different neuronal populations

Helena Janickova¹, Alice Abbondanza^{1,2}, Sylvie Dumas³, Veronique Bernard²

¹*Institute of Physiology of the Czech Academy of Sciences, Laboratory of Neurochemistry, Prague, Czech Republic,* ²*Neuroscience Paris Seine - Institut de Biologie Paris Seine (NPS - IBPS), INSERM, CNRS, Sorbonne Universite, Paris, France,* ³*Oramacell, Oramacell, Paris, France*

Nicotinic acetylcholine receptors (nAChRs) are homomeric or heteromeric ion channels well recognized for their role in the control of neural signaling, behavior and learning. The widely expressed subtype of nAChRs, beta2 subunit-containing nAChRs, are located pre- or post-synaptically in different neuronal populations in almost every brain region. Because the connectivity and function of different neuronal types expressing beta2-containing nAChRs can be different or even antagonistic in local microcircuits, we hypothesize that the role of beta2-containing nAChRs expressed by these neuronal types would also differ. In addition, as the activity of neurons with different functions may tend to cancel out each other, less specific perturbation of nicotinic signaling may have less effect on behavior than targeting the receptors in specific neuronal population even if the absolute number of affected neurons is limited. To test the functional significance of nAChRs in different types of neurons, we decided to perform deletions of the widely expressed beta2 nicotinic subunit in specific neuronal populations in the mouse prefrontal cortex, using Cre-loxP and CRISPR-based approaches. First, we also used fluorescence in situ hybridization (FISH) to determine the pattern of beta2 expression. Then we tested the mice with the deletions for potential behavioral changes, focusing on the behavioral domains associated with the prefrontal cortex and nicotinic signaling. In particular, we tested social and anxiety-like behavior. The FISH analysis showed that the expression of beta2 subunit in the prefrontal cortex is present in approximately 50 % of both excitatory and several populations of inhibitory neurons. Our behavioral data suggested that the non-specific targeting of beta2 nicotinic subunit by the Cre-loxP approach may have little effect on behavior. In contrast, some of the more specific beta2 deletions in general excitatory or specific populations of inhibitory neurons led to changes in sociability, explorative and anxiety-like behavior. We conclude that the neuronal type-specific targeting of nAChRs seems more efficient. However, the final effect of the beta2 deletion on behavior may be determined not only by the identity of the affected neurons but also by the deletion method (Cre-loxP vs. CRISPR) and by the efficiency and spread of the used viral vector.