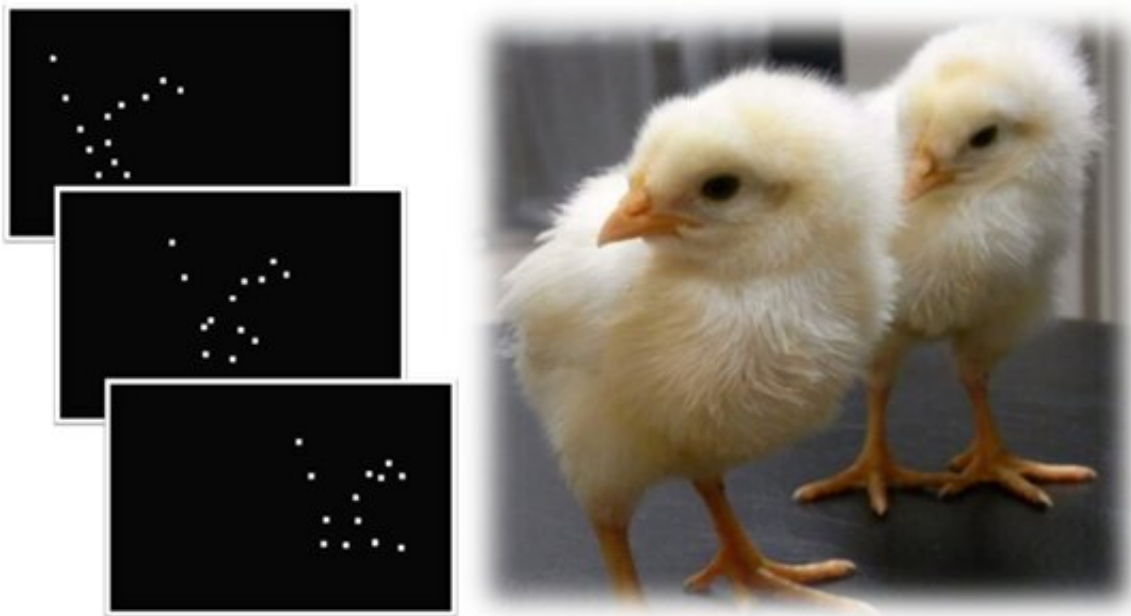


Neonicotinoid causes ASD-like symptoms in chicks, finds study

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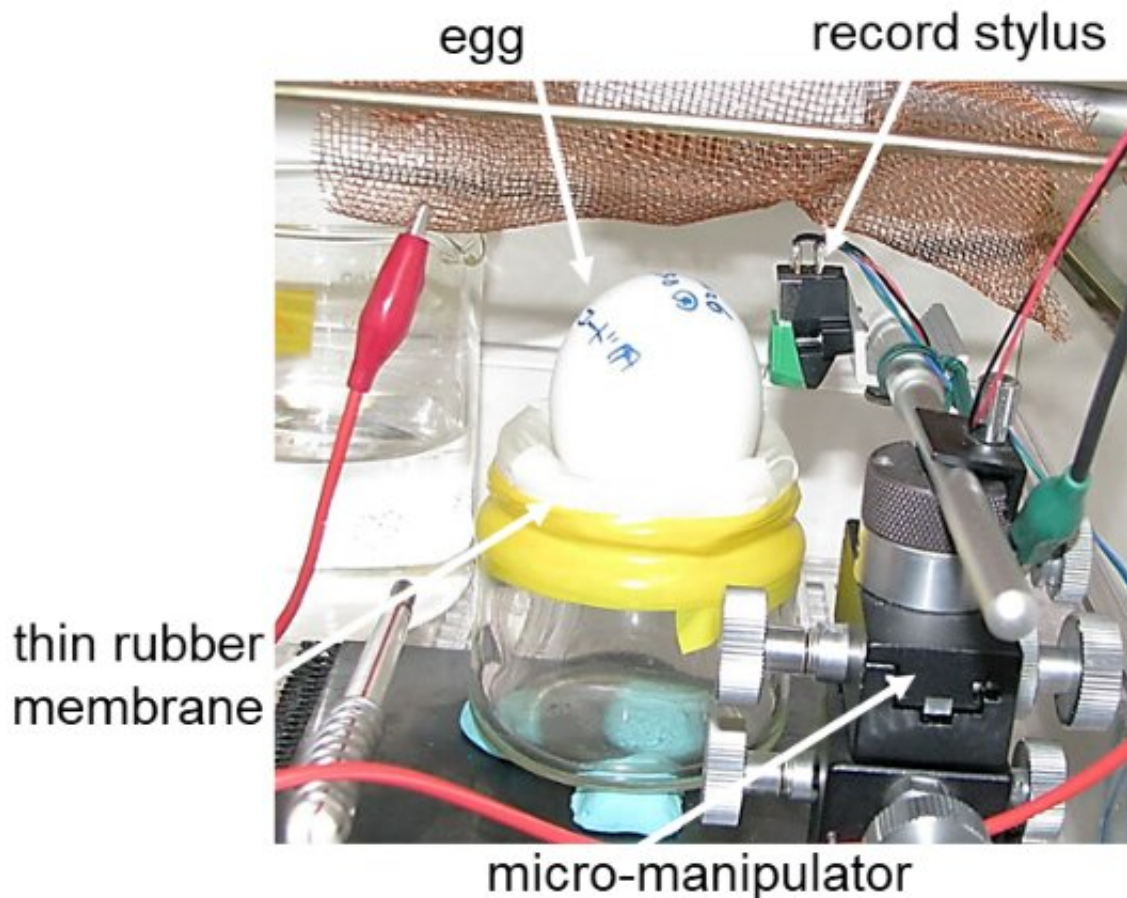


A simulation of biological motion (left) and leghorn chicks of the same breed as those used in the study (right). Credit: Toshiya Matsushima

Autism spectrum disorder (ASD) refers to a group of disabilities due to atypical brain development. Individuals with ASD have difficulties in social communication and interaction. Diverse causes of ASD are hypothesized, but most of them remain to be understood. A vast research effort has thus been invested to develop appropriate animal models to study the causes.

A team of researchers led by Professor Emeritus Toshiya Matsushima at Hokkaido University has found an ASD-like behavioral impairment in chicks, suggesting a molecular pathway of ASD pathogenesis. Their results were published in the online journal *Cerebral Cortex Communications*.

One of the earliest indications of ASD is a retarded preference for animate objects such as face configuration and biological motion (BM). This preference typically appears in neonates (newborns), but it is hampered in individuals with familial ASD risk. Common mammalian model animals such as rats and mice are not valid for studying this aspect of ASD as they do not spontaneously exhibit BM preference.

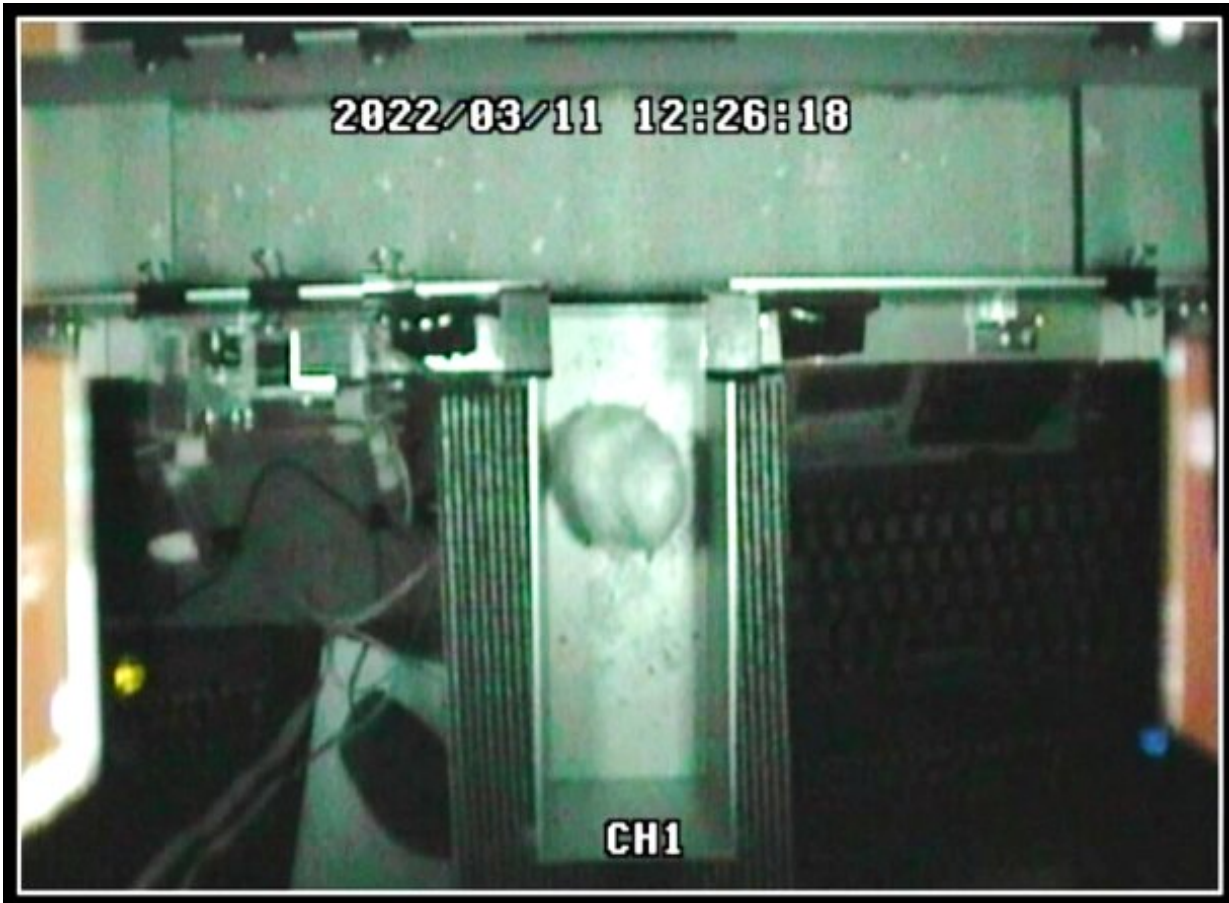


The device used to measure fetal movements of chick embryos in the study.
Credit: Toshiya Matsushima, et al. *Cerebral Cortex Communications*, November 18, 2022

The team used chicks as a model to study BM preference. Similar to humans, chicks exhibit a preference for BM very early in life, concomitant with social attachment formation by imprinting. Although birds and mammals diverged some 300 million years ago, some of their visual and motor control systems are conserved despite the evolutionary distance.

The authors initially hypothesized that spontaneous movement of fetuses is critical for the BM preference to develop. To test this, they injected fertilized eggs with ASD-risk chemicals while "listening" to the [fetal movements](#) through a record stylus gently placed on the shell surface. After hatching, preference of the chicks was tested in a T-shaped maze.

Chemicals that arrest fetal movements were systematically tested. Ketamine, tubocurarine, methyllycaconitine and imidacloprid (IMI) significantly reduced the BM preference without impairing the imprinting memory formation; these chemicals act on [nicotinic acetylcholine receptors](#) (nAChRs). Interestingly, when bumetanide—a drug reported to reduce severity in some ASD cases—was administered to chicks before imprinting, typical BM preference was resumed.



The T-shaped maze used to test the biological motion preference of the chicks in the study Credit: Toshiya Matsushima, et al. *Cerebral Cortex Communications*, November 18, 2022

Contrary to the initial hypothesis, however, the arrested fetal movement did not necessarily cause impairment in BM preference. Rather, the results show that transmission via nAChRs per se is critical. If disturbed, the BM preference is lost, and chicks fail to form selective social attachment to biological objects.

Particular concern resides in IMI, one of the most widely used [neonicotinoid](#) insecticides. As it impaired the BM at concentrations as

low as 1 ppm, environmental neonicotinoids could cause a similar retardation on human neonates if they are exposed during pregnancy, according to the study.

This study established advantages of using chicks as an ASD model. The primary advantage is the phenotype similarity between chicks and humans. Furthermore, the oviparous nature of chickens allows for precise and speedy screening of risk chemicals. But does the [chick](#) story really tell us about human ASD? The affected brain region must be clarified in terms of evolutionary neurobiology to reveal genuine similarities between birds and humans.

More information: Toshiya Matsushima et al, Fetal blockade of nicotinic acetylcholine transmission causes autism-like impairment of biological motion preference in the neonatal Chick, *Cerebral Cortex Communications* (2022). [DOI: 10.1093/texcom/tgac041](https://doi.org/10.1093/texcom/tgac041)

Provided by Hokkaido University

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