The long-range goal of our laboratory is twofold—to understand the fundamental nature of the computations that are carried out by the neocortex, including the computations that enable us to attend to sensory stimuli, and to understand how and why these computations fail in brain disease.

The brain never sits idle. Whether we are awake or asleep, watch TV or close our eyes, waves of spontaneous nerve signals wash through our brains. To be reliably processed, incoming sensory information has to stand out from this ongoing background activity. In his latest study, John Reynolds and his team wondered whether attention, which so efficiently tunes out external distractions, does the same for the internal racket.

Researchers had known for some time that paying attention to visual details increases the firing rate of neurons tuned for attended stimuli. The stronger the neural signal, the better we are able to perceive a stimulus. But neurons are very noisy computing devices, and even under the most controlled laboratory conditions, neuronal responses vary over repeated presentations of an identical sensory stimulus. If each neuron produced random noise that is independent from what its neighbor is doing, the brain cell on the receiving end could simply pool all incoming signals and average out the noise.

But most of the brain’s background noise originates in waves of spontaneous nerve signals and can’t be simply averaged out. However, an interesting thing happened when the researchers measured the activity of populations of brain cells in animals trained to play a video game that required rapt attention. When attention was directed to a visual stimulus on a computer monitor, the internal fluctuations or shared noise quieted down, increasing the salience of the incoming sensory information. This noise reduction substantially increased the fidelity of the neural signal, an improvement four times as large as the improvement caused by attention-dependent increases in firing rate alone.

A hallmark of brain disorders such as Alzheimer’s disease, autism, and schizophrenia is the loss of our capacity to attend to behaviorally relevant stimuli. This newly discovered neural mechanism has enormous implications for treating diseases in which attention fails, and the Reynolds laboratory is now embarking on a series of studies to understand how failures of this neural mechanism figure in brain disease.

For more information, please visit salk.edu/faculty/reynolds.html

Left to right:
John Reynolds, John Curtis, Emily Anderson, Tamara Berdyyeva, Jaclyn Reyes, Catherine Williams, and Jude Mitchell