controls vocal outputs through the RA (for robust nucleus of the arcopallium) neuron cluster, which indirectly stimulates vocal and respiratory muscles. When adult birds sing, RA neurons show a signature sequence of bursts during each syllable. Another pathway, called the anterior forebrain pathway (AFP), appears to be critical for song learning. AFP shares characteristics with the mammalian basal ganglia, which regulates movement and motor learning in mammals.

To explore the nature of the AFP's contributions to song learning, Fee and colleagues recorded brain activity from young zebra finches (54–79 days old) learning to sing. Then they injected young birds with drugs that temporarily blocked activity in a brain region that is part of the AFP called LMAN (lateral magnocellular nucleus of the nidopallium). Zebra finch songs typically contain three to seven syllables—the basic acoustic units of zebra finch songs—that follow a specific sequence. Thirty to 90 minutes after LMAN inactivation, the birds sang with less syllabic variation. This effect was especially dramatic in the youngest birds, which normally exhibit the greatest acoustic variation. LMAN inactivation, the authors note, “eliminated 75% of the difference in mean variability between juvenile song and adult directed song [wooing a mate, for example]—the most stereotyped form of song.” LMAN inactivation also reduced the birds’ variation in syllable sequence, which again hewed closer to the orthodoxy of adult song than to the exuberance of youthful experimentation.

The authors go on to show that changes in the firing patterns of LMAN neurons projecting into the motor pathway accompany changes in song. That LMAN inactivation reduces song variability quickly and reversibly, the authors argue, indicates that LMAN supports experimental behavior and controls song variability by providing rapid inputs to the motor pathway. This model requires that LMAN neurons show high variability across different song motifs—which is what Fee and colleagues found. As the bird sings, some as yet unknown brain areas must also evaluate the song against a template, modulating the actions of the motor pathway as a conductor might correct a performer’s mistakes in note and pitch until she masters the tune.

It’s thought that birdsong serves multiple purposes—staking a territorial claim, for example, and attracting a mate—though precisely how the song relates to fitness is still an open question. Whether inducing the type of exploratory motor behavior that’s so critical to motor learning is a fundamental feature of basal ganglia circuits also remains to be determined. But it does seem clear that these circuits play a significant role in generating the variability that songbirds need in order to acquire the communication skills of their parents—a finding that may shed light on how the brain produces the fluctuations required for learning other tasks. For more on song learning, see the primer by Fernando Nottebohm (DOI: 10.1371/journal.pbio.0030164, available online May 2005).

Ölveczky BP, Andalman AS, Fee MS (2005) Vocal experimentation in the juvenile songbird requires a basal ganglia circuit. DOI: 10.1371/journal.pbio.0030153

Infant Sleep: A Precursor to Adult Sleep?
DOI: 10.1371/journal.pbio.0030168

Sleep is absolutely essential for well-being. Just ask one of the 40 million Americans with sleep disorders who suffer crippling fatigue, impaired judgment, irritability, moodiness, and myriad health problems. Still, its precise function remains unclear. An intriguing role for REM sleep—the stage most closely associated with dreaming—was suggested almost 40 years ago when sleep researchers Howard Roffwarg and William Dement discovered that babies spend far more time in REM sleep than adults—prompting their hypothesis that infant REM sleep plays a role in central nervous system development.

A central element of their hypothesis revolves around the nature of infant sleep and whether the neural mechanisms of infant sleep differ significantly from those of adult sleep. Infant rats, like the offspring of other “altricial” species (born naked, helpless, and blind), spend most of their time in what’s now called active sleep, indicated by intermittent muscle twitching and low muscle tone (atonia)—behaviors characteristic of adult REM sleep. At issue is whether infant mechanisms are primitive, undifferentiated, and distinct from adult mechanisms or whether they contain elementary components that are integrated into the developing sleep system.

In a new study, Karl Karlsson, Mark Blumberg, and their colleagues tackle the technical difficulties involved in studying the tiny neonatal brain to investigate the neural activity associated with infant sleep states. The active sleep of week-old rats, they show, bears a striking resemblance to the conventional definitions of adult sleep. What’s more, the neural mechanisms underlying the infant sleep state contain the primary components of adult sleep.

In previous studies, Karlsson and Blumberg discovered a brainstem region in the ventromedial medulla, which they called the medullary inhibitory area (MIA), that appears functionally equivalent to the region that generates REM atonia in adults. They also found that the MIA doesn’t generate infant sleep on its own but depends on a network that spans both lower brainstem and midbrain regions. In this study, the authors set out to identify the neural structures that project to the MIA and better characterize the network.

Karlsson et al. first established that there are neurons that connect to the

DOI: 10.1371/journal.pbio.0030168.g001

Though naked, helpless, and blind, this week-old rat (pictured with a quarter) already has the fundamental neural components of adult sleep (Photo: Mark Blumberg)
MIA from areas in the medulla and pons. Then, by recording from neurons in these areas, they found neurons that are active only during sleep or wakefulness and that appear to control muscle tone and twitching. Neurons active mostly during atonia—indicating sleep—concentrated in the subcoeruleus (SubLC) region of the pons; those active mostly during wakefulness clustered in an area within the dorsolateral pontine tegmentum (DLPT) in the midbrain. The authors went on to link different sets of neurons with specific behaviors and brain regions. A group of neurons within the DLPT, for example, showed distinct bursts of activity just before muscle twitching. And a subset of SubLC neurons fired at much higher rates when atonia was accompanied by bouts of tail and neck muscle twitching.

Introducing lesions in the SubLC and another pontine nucleus, called the pontis oralis, caused significant changes in muscle tone and twitching. Lesions in the two pontine nuclei reduced periods of atonia but not the number of muscle twitches—in effect decoupling the key components of REM sleep, twitching and atonia. Lesions in the DLPT had the opposite effect: increased atonia and significantly less muscle twitching.

Altogether, the authors argue, these results show that sleep development elaborates on elementary components already in place soon after birth. If the neural mechanisms of infant and adult sleep were entirely different, then sleep might serve different purposes in infancy and adulthood. But the striking parallels outlined in this study suggest a developmental continuity between the two states. They also chart a course for future study that might even test Roffwarg’s view that the neonatal brainstem primes the central nervous system for the sensory challenges that lie ahead—and could even be the stuff that dreams are made of.