existing collection infrastructure, those costs were saved because of the foresight by USDA researchers and NMNH curators 80 years ago. One wonders about the full potential of museum collections as repositories of internationally useful databases if museums received even a fraction of the support for their long-term research efforts as short-term efforts in physics or in space exploration.

Failure to adequately fund museums and biodiversity surveys will come at a massive cost in terms of lost future opportunity, one that will be paid by future generations. The study of long-term ecological processes, such as those affected by the accelerating global traffic of invasive species, will require far greater resources than currently made available; decisions based on expectations of instantaneous research results will be short-sighted. Like investments into schools and education, investment into museum collections, global biodiversity surveys and taxonomic training is investment benefiting future generations, with incalculable economic and health benefits [7].

The Future of Ant Invasion Ecology

Ants, which are present in large numbers in many ecosystems, drive numerous ecological processes [8]. Consequently, introduced ant species, especially a few high-impact ant invaders such as the fire ant, cause fundamental changes in their new habitat, to the detriment of native organisms. For example, by competitively excluding native seed-dispersing ants, invasive Argentine ants have caused major shifts in plant compositions in the South African shrublands [9]. Likewise, by eliminating the native ants, which serve as food for horned lizards, Argentine ants have likely led to declines in lizard populations in California [10].

To date, most research on exotic invaders has largely focused on plants, arguably the most conspicuous invaders with the most obvious effects on the environment. Ants and plants share many life history traits, which make insights from one group potentially applicable to the other. For example, while invading plants have a tendency to reproduce clonally, so do invasive ants, whose colonies often bud, some species producing queens from 'asexual' fragments containing just workers and brood [11]. At the same time, the social structure and behavior of an ant colony is more easily dissected than the processes operating inside a plant, which may make ants a better model system to study organismal prerequisites enabling a successful invasion. Indeed, while the propensity for vegetative reproduction remains the only well-established ecological characteristic that unites invasive plants [12], successful ant invaders exhibit a suite of characteristics listed above [5,6,13]. With the aid of data compiled by Suarez *et al*. [4], future behavioral, ecological, and systematic research can directly test the relevance of these characteristics to the ants success as invaders, potentially answering questions relevant not just to ant invasions, but to biological invasions as a whole.

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Autism Spectrum Disorder: Seeing Is Not Understanding

Impairments in social and emotional skills are a defining feature of autism spectrum disorder. Recent research shows that structural and functional abnormalities within the neural system that matches observation and execution of actions — the mirror neuron system — may explain the social aspects of the pathophysiology of autism spectrum disorder.

Shirley Fecteau[1,2], Jean-François Lepage[1] and Hugo Théoret[1]

The discovery of 'mirror' neurons in the ventral premotor cortex of macaque monkeys — neurons which respond to both observation and performance of a particular action — and the growing evidence that neurons with similar properties are present in the human brain have led many to suggest a fundamental role for the mirror neuron system in social cognition. Specifically, the mirror

neuron system appears to be an essential component not only of action understanding [1,2] and imitation [3,4], but also of fundamental cognitive functions such as social interaction [5], empathy [6], language [5,7], mind reading [5,8], theory of mind [1,5] and emotional processing [9]. Specifically, mirror neurons are thought to mediate understanding of actions performed by others in the following way: each time an individual sees an action performed by a peer, neurons that represent that action are activated in the observer's premotor cortex. The same process seems to occur with higher cognitive functions: for instance, the neural networks normally activated during an experienced emotion allow us to understand the same emotion experienced by a peer. This process seems to underlie the basic aspects of social cognition by creating a link between others and ourselves [8].

The mirror neuron system is formed by a cortical network composed of the pars opercularis of the inferior frontal gyrus and the rostral part of the inferior parietal lobe (see [1] for more details). Importantly, the neural system that resonates in the observer's brain networks depends on the mirror function elicited — the observed action. In the case of emotional processing, for example, observation of a face expressing fear may engage the amygdala [10], whereas observation of a face expressing disgust recruits the insula [11], in conjunction with the classical mirror neuron system.

As several functions believed to be subserved by the mirror neuron system are impaired in autism spectrum disorder (ASD), ranging from imitation to empathy, abnormal mirror neuron function has been suggested as a possible neural substrate of the social impairments characteristic of this condition [5,6,8,12]. Deficits in imitation are well documented behaviorally in ASD, but it has only recently been suggested that brain activity related to these deficits is abnormal, as shown by converging data from magnetoencephalography [13], functional magnetic resonance imaging (fMRI) [14] and functional connectivity MRI [15].

In what may be the strongest demonstration of a mirror neuron system dysfunction in ASD so far, Dapretto *et al*. [16] recently reported fMRI activation patterns of children with ASD while they imitated facial expressions. Whereas imitation performance of children with ASD was equal to that of typically developing children, only the typically developing children showed enhanced bilateral activity in the pars opercularis of the inferior frontal gyrus, the main mirror neuron area. In fact, children with ASD showed no activation within the pars opercularis of either hemisphere during imitation. Moreover, similar differences in patterns of activation were found when participants passively observed facial expressions, suggesting that the deficit in imitation is the result of a deficit in observation.

These results strengthen the claim that individuals with ASD have difficulties in reading the emotional state of others because of a failure in adequately activating some of the brain areas that would normally be active if they were experiencing the emotion themselves. Of great importance is the fact that Dapretto *et al*. [16] also found that activity within the pars opercularis of the inferior frontal gyrus was inversely correlated with severity of social dysfunction, underlining the link between social behavior and mirror neuron system function.

In a strikingly complementary study, Hadjikani *et al*. [17] have recently reported that adults with ASD displayed significantly reduced cortical thickness in the main mirror neuron areas, namely the bilateral pars opercularis of the inferior frontal gyrus (also in the inferior parietal lobule and superior temporal sulcus). These areas are the same that failed to activate when children with ASD imitated facial expressions [16]. Again in agreement with functional data, cortical thinning in these areas was correlated with severity of communication and social symptoms.

It appears, then, that the neurophysiological dysfunction found in the mirror areas of individuals with ASD may be rooted in more general structural abnormalities giving rise to complex anatomical-functional interactions. These could potentially explain some of the variability observed in the social-behavioral symptoms of ASD. Taken together, these studies make a strong case for the mirror neuron hypothesis of ASD, particularly if one considers that even passive observation of meaningless hand movement elicits weaker mirror neuron system activity in individuals with ASD, as measured by transcranial magnetic stimulation [18] and electroencephalography [19], suggesting that mirror neuron system dysfunction in ASD is not restricted to emotional processing.

One could argue that abnormal mirror-neuron-system-related activations are explained by impaired visual recognition of biological motion (see [20] for a discussion of visual perception in ASD). However, a clear dissociation in neural activity related to visual recognition of biological movement and imitation has been reported, where abnormal neural activity in ASD was restricted to action observation [13]. Specifically, in early cognitive processing, individuals with ASD and healthy controls showed similar patterns of activation involving occipital cortex, superior temporal sulcus and inferior parietal lobule, suggesting normal visual analysis of biological motion. In later stages, however, differences were observed between ASD and normal participants, where weaker and delayed activity was found in the inferior frontal cortex and primary motor cortex of individuals with ASD.

If the existing body of evidence is to be believed, behavioral phenotypes that would emerge as a result of abnormal development of mirror neuron system function should lead to social and communicative deficits similar to those seen in ASD. Indeed, the correspondence between ASD phenotype and mirror neuron

system function is striking. Specifically, failures in mirror neuron system development should result in action understanding and imitation deficits which, in turn, would lead to impaired self-other representation, social and communicative deficits, and ultimately empathic, language, and emotional failures.

Despite this promising take on ASD etiology, the abnormal direct-matching mechanism described here is obviously one among many presumably abnormal processes in ASD. Indeed, the mirror neuron system hypothesis of ASD does not exclude the possibility that other cognitive processes also participate in the complex pathophysiology of ASD. Although the integrity of the mirror neuron system seems to be critical for action understanding, mirror neuron system failures probably do not account for all of the reported social impairments of ASD. Moreover, mirror neuron system function and its neural network are not entirely abnormal in ASD. As shown by Dapretto *et al*. [16], children with ASD are able to imitate facial expressions and display patterns of activity in the amygdala similar to those of healthy participants.

It is important to note that despite enormous efforts in the last decade to pinpoint the specific causes of ASD, the gold standard in diagnosing ASD still rests on behavioral observation; no biological or genetic marker exists as of yet. As such, abnormalities in mirror neuron system neural substrates — the inferior frontal and parietal areas — may be important cues in the diagnosis of ASD. From this knowledge, diagnostic markers, and ultimately therapeutic targets for treatment that would allow for early intervention, could be developed. The critical step that needs to follow these exciting results is to establish whether the reported abnormalities in mirror neuron system function have any clinical value.

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Plasmid Segregation: A New Class of Cytoskeletal Proteins Emerges

The discovery that a plasmid-partitioning ATPase forms astral cytoskeletal structures both unveils a new family of cytoskeletal proteins and suggests that cytoskeletal involvement is a universal feature of DNA segregation.

Zemer Gitai

The ability to propagate genetic information faithfully is a prerequisite for evolutionary success. To this end, many bacterial plasmids encode their own machinery to ensure their proper segregation and subcellular positioning. A recent study [1] finds that a member of the most common class of these plasmid-partitioning proteins assembles into cytoskeleton-like filaments, and that these filaments can focus into asters that strikingly resemble those found in eukaryotic mitotic spindles. These insights further our understanding of both the specifics of plasmid partitioning,