Introduction

Hope is the thing with feathers
That perches in the soul . . .
– Emily Dickinson

Ours is the age of biological knowledge. In the scope of its objectives and in its potential for transforming how we think about our place in the world, the Human Genome Project is the direct descendant of the Manhattan Project. This seemingly incongruous analogy has at its center the common theme of an assembly of scientists working together toward a common end and with great potential power. For the Human Genome Project, that end is deciphering the molecular composition of our genetic heritage.

Experiment lies at the heart of modern science (Dear, 1995; see also Bernard, 1865/1957). The idea of reconstructing who we are, of illuminating a moment in our evolutionary journey, and of knowing in full detail the underlying blueprint and structure of our biological material is the ultimate legacy of Charles Darwin and Gregor Mendel, arguably the progenitors of the modern biological sciences. The depiction of our entire genetic structure is as revolutionary as was Albert Einstein's reconstruction of the world of physics once inhabited by Isaac Newton.

We can only comprehend the radical nature of the Human Genome Project's import because we come prepared to understand

the world in terms of agents and action, direction. We come prepared to share and exploit experiences, to form meaningful connections – to be connected to others (Jaspers, 1913/1997). In this introduction, I lay the groundwork for (1) an orientation toward understanding and creating living objects and (2) the behavioral/neural underpinnings of our understanding of the embodied states of others. The cognitive achievement of distinguishing animate from inanimate objects and then recognizing the beliefs and desires of others and their personal histories – a related but distinct cognitive adaptation – in part underlies both our evolution and the devolution of this function. Those facts, along with a general ability for self-corrective inquiry, underlie important cognitive achievements.

CREATING AND UNDERSTANDING LIVING THINGS. Imagine injecting a gene into a virus that is taken up by aberrant cells, thereby correcting for a genetic pathology. This scenario is no longer the stuff of science fiction but is fact. As they are perfected, gene therapy and similar methodologies will be used in the treatment of cystic fibrosis, cancers, hemophilia, and other ailments (see Figure I.1). Although never eradicated, our vulnerability to a variety of diseases and biological markers will be reduced and is being reduced to a molecular level of analysis (see Kitcher, 1996; Rosenberg, 2006).

Biological scientists can take great pride in these events while maintaining a sense that great events in science carry potential dangers. This is because science, as Sir Francis Bacon noted, is power. It can be used for noble, mundane, or atrocious purposes. Along with the great advances against human disease and suffering, both this century and the last bear testimony to science as a potentially devastating force.

Recall Mary Wollstonecraft Shelley's (1818/1976) great novel about the origins of Dr. Frankenstein's creation and the power of science. As we expand the biological sciences, fears of mimicking a creation like that of Frankenstein become omnipresent. Dr. Frankenstein's

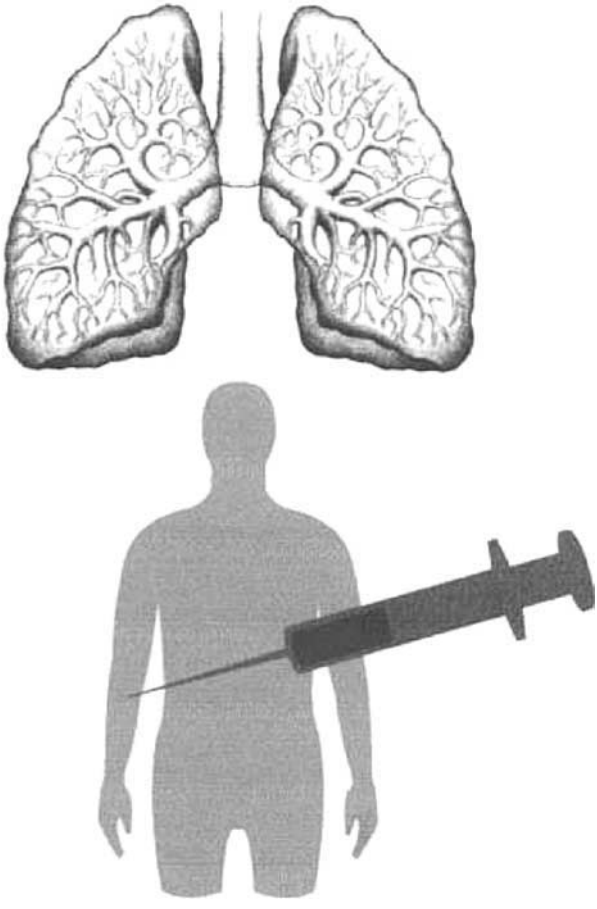

FIGURE I.1. A cartoon of injecting a gene that can correct for a disease of the lung like cystic fibrosis (Yansen & Schulkin, 2007).

nameless monster is a creature of our own making (see Figure I.2). We generated the power and knowledge to create this artificial man, yet the brute is a naive sort of blank slate on which the rest of humanity makes its imprint. He is not inherently evil, and he is without prejudice. Dr. Frankenstein yells out, "It's alive!" as he watches his creation move and gesture. But the consequences of the monster's agency, his aliveness, are dire.

FIGURE I.2. The monster of Dr. Frankenstein's creation (Yansen & Schulkin, 2007).

The world imposes, and the monster grows angry with disappointment and hurt. He begins to strike back and asserts to his creator:

> I am malicious because I am miserable. Am I not shunned and hated by all mankind? You, my creator, would tear me to pieces and triumph; remember that, and tell me why I should pity man more than he pities me? You would not call it murder if you could precipitate me into one of those ice-rifts and destroy my frame, the work of your own hands. Shall I respect man when he condemns me? Let him live with me in the interchange of kindness; and instead of injury I would bestow every benefit upon him with tears of gratitude at his acceptance. (Shelley 1817/1976, p. 130; see also McGinn, 1997/1999)

We have a cognitive capacity to distinguish the animate from the inanimate, but as our biological technologies surge forward, we begin

to see a blurring of the conventional distinctions between the natural world and the cultural one that we have constructed. This is the world in which we are trying to create something alive. If science is humanized and directed by sanguine judgments, then perhaps the object lesson of Mary Shelley's *Frankenstein* appears less formidable.

The emphasis of the book is the sense of animacy coupled with our sense of agency. We are far from truly simulating such links between biological tissue and inorganic devices, but our ultimate goal is the fusion of the two, the creation of animate objects. The combination of the biological with the inanimate of our creation is something of our generation, for which Mary Shelley's premonition has furnished us with an orientation. The cognitive preconditions reflect our own predilection to understand the world in terms of the living and the nonliving, whether or not someone is an agent (with beliefs, desires, and experiences to be considered).

Creating our own tissue is a modern form of the fusion of our concept of animacy (i.e., something alive) with the new tools of information processing and molecular biology. We are only at the beginning of this fusion. We are not nearly close to talking about agency (Sabini & Schulkin, 1994; Sabini & Silver, 1982), but its roots rest in our cognitive predilection to distinguish the animate from the inanimate (see Carey, 1985/1987; Keil, 1983; Meltzoff, 2004).

We may, for example, ameliorate diverse kinds of learning disabilities through the use of stem cells. Stem cells are undifferentiated cells that may be useful in correcting cells that do not function well. It is conceivable that stem cell tissue can be used for a wide variety of neurological disorders that are important for cognitive systems that learn, remember, and are stable (Gage, 1998); it may enable the most debilitated among us to become functional members of society. This birth and rebirth is now close to being a scientific reality (e.g., Altman, 1966; Gould & McEwen, 1993; Kempermann, 2006; Ming & Song, 2005).

Rebirth and rejuvenation are at the heart of animate objects. In fact, it is possible that the fusion of our creation (stem cells) has the

potential to ameliorate diverse diseases, including autism – a state in which one's sense of others' experiences is compromised (Baron-Cohen, 1995/2000).

AUTISM AND THE UNDERSTANDING OF OTHER HUMAN BEINGS. We are a species cognitively prepared to predict the behavior of others and to understand others by the kinds of beliefs and desires that figure into what they do, as well as what we predict they will do (e.g., Dennett, 1987; Premack, 1990). The devolution of this function is a feature of decreased social competence (Baron-Cohen, 1995/2000; Schulkin, 2000, 2007b).

Agency and animacy are two cardinal features in our cognitive lexicon that are replete with meaning, and that, in autistic individuals, are impaired. The concept of agency, in the sense in which I use the term, is tied to our beliefs, desires, preferences and goals, personal histories, and historical legacies. Animacy is tied to agency, but it is not the same; something can be alive without being an agent. The concept is rooted in our intellectual history and originally tied to something with a soul (or the Latin *anima*; Skrbina, 2005). Animacy is about something being alive. There are diverse meanings of this term as it applies to the concept of agency. The transition from something being alive to something being an agent is (1) the instantiation of beliefs, desires, and goals and (2) personal history (e.g., Dennett, 1987; Dewey, 1925/1989; Neville, 1974; Sterelny, 2000; Weissman, 2000).

These two categories of whether or not something is alive figure importantly in our understanding of the world (Carey, 1985/1987; Keil, 1979, 2007) and are tied to our ability to determine diverse properties of a thing. Our cognitive inclination is to explore when we have to, when settled views are disrupted (Dewey, 1925/1989). This inclination to explore and discover in the context of disrupted expectations is coupled with a keen sense for occasionally hitting on the right hypothesis (Peirce, 1899/1992). Cognitive systems, in the sense I suggest, are nothing like the old Cartesian, divorced, distant arbiter. Rather, they are

about adaptation and action (see also Dewey, 1916; Lakoff & Johnson, 1999). Cognitive systems evolved to make sense of our surroundings and to problem solve; they are linked to engaged self-corrective inquiry (e.g., Anderson, 1997; Dewey, 1925/1989; Meltzoff, 2007).

There is an interface between simulated cognitive systems in diverse forms of material (e.g., chips) and biological material. There is also a confluence of the neural, psychological, and biological sciences in simulated forms of expression. Simulating diverse forms of biological functions, fusing the artificial with the biological, is part of the exciting age in which we live (Clark, 2003; Kitcher, 1996). Of course, we are more than the narrow notion of machines – those absolute clockwork devices envisioned in the seventeenth century (Descartes, 1644/1967). We are neither Cartesian machines thinking in a vacuum nor empirical blank slates (e.g., Levinson & Jaisson, 2006; Pinker, 2007). We bring with us diverse forms of cognitive devices that underlie the embodied experiences, what Dewey used to call "lived experiences," or what others have called "embodied cognition" (see Gallagher, 2005; Gibbs, 2006; Johnson, 2007; Lakoff & Johnson, 1999; Prinz, 2004; Schulkin, 2004; Varela, Thompson, & Rosch, 1991). Our cognitive evolution is reflected in the diverse expansion of these two categories across domains of biological and social interactions. Our devolution, a decrease in function, is also reflected in human pathology (Jackson, 1884/1958).

Decreased cognitive expression, such as toward agency and animacy, is a feature of autistic individuals. Autism involves social withdrawal, lack of eye contact, and lack of responsiveness to surrounding social situations (e.g., Baron-Cohen, 1995/2000). Recognizing the beliefs and desires of others is part of recognizing them as people who have experiences, who are agents (e.g., Fromm, 1947; Schulkin, 1992; Frith & Wolpert, 2003; Leslie, 1987). This sort of knowledge requires communication, making eye contact, touching one another, and forming meaningful bonds – something diminished by the cognitive competence of autistic individuals. These individuals may be

good at solving mechanical problems, but they are severely limited in making social contact and at tasks that require focusing on another individual.

Two key cognitive features stand out in individuals with autism: they are more comfortable with less human contact and, in some instances (when controlling for IQ), are better able to solve problems that reflect mechanical (rather than personal) issues than are individuals without autism (Baron-Cohen, 1995/2000; Leslie, 1987). Although the ability to discriminate between animate and inanimate objects is present in autism, it is compromised (Baron-Cohen, 1995/2000). Autistic subjects often show a preference for inanimate objects and avoid human, animate contact. Autism is marked by a specific lack of interest in people and interpersonal interactions.

Shared human contact often entails looking into the eyes of another, watching what they are watching, and sharing experiences through vision (Baron-Cohen, 1995/2000; Tomasello, Carpenter, Call, Behne, & Moll, 2004). Experiments have consistently shown that autistic individuals have trouble sharing social contact through the visual system (Baron-Cohen, Tager-Flushberg, & Cohen, 1993/2000) – eye contact or gazing into the face of others is compromised (Baron-Cohen, 1995/2000; Specio et al., 2007). This is a fundamental impairment in gaining a foothold in the life world (Schutz, 1932/1967), the world of acknowledged human experiences, and in gaining fundamental human meaning through significant connections with others (Jaspers, 1913/1997).

Parsing out the social space of another human being, through the attribution of beliefs and desires, implies references to the experiences of others (e.g., Jaspers, 1913/1997; Mead, 1934/1972). After all, we believe that there is someone inside having an experience of one kind or another. Children fundamentally recognize animate objects very early on in ontogeny (Carey, 1985; Keil, 1987, 2007; Premack, 1990). Object contact, recognition of something as animate or inanimate (expressing intentional direction or not), is a fundamental cognitive

tracking event of objects (Premack & Premack, 1983), and is one way in which we are rooted in our understanding of one another. Facial expressions, eye contact, and shared attention (e.g., shared mutual awareness of common focus) on someone's face are obviously important sources of information (Darwin, 1872/1965; Ekman, 1972). Young children and adults use this information in forming attachments. An appreciation of these events is compromised in autism (Baron-Cohen, 1995/2000, 2008; Dalton et al., 2005) and is more pronounced in boys generally (Baron-Cohen, Knickmeyer, & Belmonte, 2005).

Key neural structures underlie our sense of other people's beliefs and desires (e.g., Frith & Frith, 1999; Frith, 2007). For example, in brain-imaging studies under diverse experimental conditions, regions of the frontal cortex are activated when the person recognizes the experiences of others (e.g., Baron-Cohen 1995/2000). An early study using computerized photon emission tomography to measure blood flow as an index of neural activation showed increased activation of the frontal orbital field when subjects were asked to think about mental as opposed to physical words (e.g., Baron-Cohen, 1995/2000, 2008). The results suggested increased activation (greater blood flow) in the orbitofrontal cortex when subjects were attending to terms about beliefs and desires, as opposed to terms about bodily considerations.

The frontal cortex and other cortical sites, including the amygdala (old cortex), have since been shown to be significantly involved in recognizing the beliefs and desires of others (Frith & Frith, 1999). Using functional magnetic resonance imaging (fMRI), the brain regions that were activated in controls were the orbitofrontal cortex, superior temporal gyrus, and the amygdala (Baron-Cohen et al., 1999; Wang et al., 2004). Autistic subjects, on the other hand, showed compromised activation in both cortical sites and in the amygdala when compared to subjects who did not have autism (see Critchley et al., 2000; see also Amaral, Bauman, & Schumann, 2003; Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007; Frith & Frith, 1999).

Perhaps chemical signals in the brain (e.g., oxytocin) that are tied to attachment behaviors and that are compromised in autistic individuals (Carter, Lederhendler, & Kirkpatrick, 1997/1999; Insel & Fernald, 2004; Insel, O'Brien, & Leckman, 1999) could be ameliorated in part by stem cells that could turn into oxytocin cells in the brain. We know that autistic individuals have lower levels of oxytocin than age-matched controls (Modahl et al., 1998; Green, Fein, Modahl, Feinstein, Waterhouse & Morris, 2001) and can benefit somewhat from infusions of oxytocin and from treatments that affect oxytocin expression (Hollander et al., 2003; 2006), which serve to ameliorate some of the symptoms associated with autism (e.g., repetitive movements) that compromise behavioral adaptation. Importantly, an oxytocin receptor gene has been linked to autism (Wu et al., 2005), and oxytocin regulation in a number of species is fundamentally linked to diverse forms of attachment behaviors (Carter, 2007; Carter et al., 1997/1999; Lim, Bielsky, & Young, 2005) that are essential for getting a foothold in a world through contact with others.

Perhaps oxytocin levels could be rejuvenated in chemical composition by stem cells and thereby restore some of the human contact essential for normal development and successful adaptation through the restoration of neural function in diverse brain regions (e.g., amygdala, regions of the neocortex) (see Figure I.3).

Treatments for autism are an important scientific goal because the condition makes it difficult for people to function successfully, though they may have no physical problems and, in some cases, their intelligence is unimpaired. The pathology demonstrates the fundamental importance of agency and animacy to our interaction with the world.

COGNITIVE AND NEURAL PREDILECTION TO DETECT SELF-PROPELLED MOVEMENT. We are a species with elaborate taxonomic and thematic resources (e.g., Carey, 1985, 1987; Murphy, 2002). Understanding how the mind works entails understanding something about the