

# Finding Our Roots

MELISSA INGRAM

## The Impending Genetic Revolution

**A** couple arrives at their local geneticist's office. They sit down at his desk, anxiously awaiting the results of their in vitro fertilization.

"Have you given any thought to whether you would like a boy or a girl?" the doctor asks with an endearing smile. A television screen sitting on his desk shows four pink spots, magnified greatly; each is a successfully formed zygote. The parents are capable of producing children without the help of a geneticist; they have already had one son together. They have come to their geneticist's office to make a more perfect child the second time around.

The wife glances nervously at her husband. "We would like a boy for our older son to play with," she says.

"Good! I've already screened out the more undesirable traits—all the common diseases, obesity, aggressive behavior—and selected for intelligence, strength—"

"Isn't it better if, you know, some things are left up to chance?" the husband interjects, his apprehension apparent in the way he leans forward, scrunching his brow.

The geneticist shakes his head dismissively. "You and your wife could have conceived naturally a thousand times and still not gotten the results my office did. Your son will still be you—just, the *best* of you."

These parents, like most other middle- and upper-class parents in the futuristic world of the movie *Gattaca*, will "choose" their child. A number of their eggs and sperm have been collected, screened, and assembled to produce the most desirable version of their genes. The resulting progeny will have everything—genetically, at least—working in favor of its success from birth. This routine is all in a day's work for the local geneticist.

Some people fear that this fictive world will come to fruition sometime soon; others cannot wait for when parents-to-be will have nearly absolute

control over their children's characteristics. Reality will never parallel the simplicity of *Gattaca*, however, because genetics is not that clear-cut.

## Cloning Revisited

In 1997, Ian Wilmut and his research team announced in *Nature* the birth of Dolly the sheep. She was the first mammal to be successfully cloned from an adult cell, and the process of making her was tedious and extensive. In total, 277 couplets of cells were originally fused, of which 29 tiny zygotes survived. Only 7.7% of the implantations of those 29 zygotes into carrier sheep resulted in pregnancies, and only one live lamb, Dolly, was eventually born ("Viable" 811). And her life as a clone was by no means perfect. At age five Dolly developed a severe arthritic condition; at age six, half the life expectancy of sheep, she was euthanized. Dolly's arthritis and lung disease were conditions usually found only in much older sheep (Briggs).

The 1997 birth of Dolly, a symbol of the future of genetic engineering, drew significant public attention. People thought that humans would be next (Holliman; Huxford). But the media neglected to mention the time and resources needed to produce this wonder of modern biology, or the complications she developed as a direct result of the cloning procedure. In 2003, Wilmut admitted that "the most striking thing . . . that emerged during Dolly's life is that mammalian cloning remains a repeatable, but inefficient procedure. It is still true that only 1–5% of reconstructed embryos develop to become viable offspring, regardless of variations in species, cell type, or . . . protocol" ("Editorial" 99).

This perspective contrasts sharply with the optimistic media discourse surrounding Dolly, and the unsettling ease with which the two parents in *Gattaca* select their offspring's traits. Soon after the parents arrive at their toughest decision—which genes to give their son—the geneticist's team must assemble all those bits of genetic information into something the mother-to-be's reproductive tract will recognize as one of its own. That is a complicated, important topic *Gattaca* didn't show. And these kinds of simplifications are not limited to science fiction films and the media's coverage of socially relevant discoveries, where emotional appeal can matter almost as much as scientific accuracy. Nearly all exchanges between scientists and the public result in the media taking liberties with the details.

## Simplifying Complexity

In 2007, Olivia Judson, an evolutionary biologist and popular science writer, wrote “The Selfless Gene” for *The Atlantic*. The title is a clever spin on Richard Dawkins’s groundbreaking 1976 book *The Selfish Gene*. Dawkins described the behavior of individual genes as inherently self-interested: a gene will try to ensure that it is passed on to offspring, no matter what the cost. In “The Selfless Gene” Judson expands Dawkins’s discussion by exposing an ironic complication in his thesis—that animals, at the organismal level, can further their “selfish genes” by acting *selflessly*. When animals cooperate, they are more likely to reproduce than if each lives independently (145).

Judson introduces us to Hamilton’s rule, which predicts why and to what extent organisms will behave altruistically to propagate their self-interested genes. According to Hamilton, “genes that promote the altruistic act will spread if the benefit . . . is high enough and if the genetic relationship . . . between the altruist and the beneficiary is close enough to outweigh the act’s cost . . . to the altruist” (Judson 145). In essence, the closer the kinship, the more likely an act of self-sacrifice is—as long as the altruistic act benefits the children or descendants. For instance, lionesses “live with their sisters, cousins, and aunts,” hunting together and sharing child-care. Although a lioness without a mate may not have cubs herself, her action in helping her relatives protect and raise the family cubs makes their chances of survival greater, thus ensuring that the genes she shares with them also survive and spread (Judson 145). Humans helping close relatives are really then, on the genetic level, helping themselves. In *Gattaca*, the older brother, Vincent, and Anton, his genetically perfected younger brother, share a similar set of genes inherited from their parents. In one scene, Anton nearly drowns in a lake, but Vincent pulls his younger brother back to shore, risking his own safety in the process. Even if Vincent had died saving his brother, his genes would still have been passed on to the next generation—through Anton. “‘Expensive’ behaviors,” like the drive to rescue someone, “evolve only in the context of close kin,” Judson explains (145). Evolutionary success, measured in the number and health of progeny, is negatively affected by such “‘expensive behaviors,’” but the impact is mitigated when one is helping a family member who shares many of the same genes.

Not all scientists agree that the evolution of altruism is as simple as Judson claims. Two researchers at the University of Connecticut have found that emotional closeness, not genetic likeness, influences individuals’ willingness to behave altruistically (Korchmaros & Kenny 262). They theorize that

an individual is unlikely to act altruistically toward a genetically similar relative if the two are not also emotionally attached. In *Gattaca*, Vincent leaves home as a teenager, severing all ties to his parents and his little brother Anton. In a chance meeting more than a decade later, Anton gathers the evidence necessary to indict Vincent for a serious crime and deliberates exposing his brother to the authorities—something most siblings would never consider doing. But this kind of behavior is possible for Vincent and Anton because they are not emotionally close, despite their genetic similarities. Kin selection alone does not seem to be the root of altruism, as Judson implies in “The Selfless Gene.” It also depends on emotional closeness, the presence of specific genes, and development. While Judson—using vivid, powerful examples—conveys the evolutionary thrust for altruism as a particular behavioral trait, we must consider both the wide-angle explanation for altruism and the deeper, specific processes that occur at each successive transmission of “selfless genes” to appreciate fully our phenomenal propensity for giving. Molecular factors, acting above the level of DNA but still within a single cell, can silence a gene’s expression. Without accounting for these “epi-genetic” processes, the geneticist’s work in the world of *Gattaca* and the evolutionary biologist’s theories about behavior will have limited accuracy.

## **Fat, Yellow, and Selfish**

Suppose we clone Sally, a lab mouse at a healthy weight with a brown coat. We would expect Sally’s clone to be identical to her in every regard, and indeed, her first cloned offspring is a slender, brown mouse. Being curious scientists, we repeat the laborious cloning procedure, this time changing Sally’s nutrient intake while she is pregnant. This second clone has a yellow coat and grows to more than double Sally’s size! After a sedentary, ailment-ridden life, this morbidly obese mouse dies of a heart condition linked to its tremendous weight and generally poor health. We are left with an intriguing puzzle: how could Sally’s diet affect the health and appearance of her identical clone so starkly?

Many theories and many mice later, scientists conducting experiments like this arrived at the answer. When a pregnant mouse consumes sufficient methyl groups (-CH<sub>3</sub>), which are found in rich supply in folic acid and grains, a gene called *agouti* is turned off at a key point in her clone’s development, and the clone matures normally (*Epigenetics*). If she is deprived of that essential chemical compound, however, her clone will never stop producing the protein that the *agouti* gene codes for, and the clone will suffer a terrible fate.

The methyl groups the pregnant mouse consumes find their way to the DNA of the embryo inside her and attach at specific points in that embryo's *agouti* gene. The gene thus becomes unreadable to the processes that would turn it into a physical trait, and the young clone is saved from a lifetime of obesity and disease (Waterland 401).

This process, DNA methylation, is only one of the epigenetic processes that regulate whether genes are readable or not. Scientists like to say that readable genes are “turned on” and unreadable genes are “turned off,” with epigenetic processes like methylation acting as a switch of sorts. In humans, genes that are “on” at full power in our youth are “turned off” one-by-one as we age, while other genes that were dormant when we were young are “turned on.” Both processes are affected by our diet and lifestyle.

The implications of this switching model are huge. Adverse conditions that have a genetic basis could, in theory, simply be turned off. The messy business of replacing the “obese” gene with a “skinny” gene could be avoided through nutrition, saving the individual from the potentially harmful effects of gene cloning that Dolly suffered and saving the geneticists, like those in *Gattaca*, valuable time and resources. More important, if diet can control the genes of mice, the traditional divides between “nature” and “nurture,” “genetic” and “environmental,” dissolve. Kinship and emotional closeness may be only two of a plethora of factors that help explain the complex ways humans interact with one another.

## **Blame Mom, not Dad**

On the long arm of the human chromosome 15, a region of genes—called 15q11-13 by the scientists who study it—is involved in a wide array of essential processes. Unfortunately, early in development this region might accidentally get chopped off the chromosome. When the paternal copy of this set of genes is deleted, the individual develops Prader-Willi syndrome (PWS). If the maternal copy of this set of genes is deleted, the individual develops Angelman syndrome (AS) (Xin 1389). PWS is characterized by low muscle tone, short stature, “temper tantrums and compulsive traits,” and hyperphagia—an insatiable appetite—that leads to obesity unless carefully controlled from infancy (Cassidy 3). People with PWS desire food so much that “hoarding, . . . eating of unappealing food items, and stealing of food or money to buy food” is common among them (Cassidy 4). If their appetites are not kept in check by an external source, their mortality rate is increased six-fold compared to other developmentally disabled individuals because of obe-

sity-related illnesses (Cassidy 5). In contrast, Angelman syndrome is distinguished by severe mental retardation, epileptic seizures, decreased need to sleep, lack of speech, a happy demeanor punctuated with bouts of inappropriate laughter, and no hyperphagia (Van Buggenhout 1367).

How can two such different disorders result from changes to the same region of DNA? Our understanding of genetics rests on the assumption that each gene codes for a certain function. If the geneticists in *Gattaca* insert a strength gene into an embryo that develops into Anton, he ought to be strong regardless of whether the scientists took the gene from his mother or his father's cells. A gene is a gene. But recent studies—such as that of PWS/AS—have shown that developing cells can often recognize whether the mother or father donated a particular gene and will replicate the gene accordingly. The “parental recognition” phenomenon in PWS/AS is achieved by the same type of methylation that causes the *agouti* gene to be silenced in mice. The only difference is that it is targeted, specific to only one copy of a gene, which makes accounting for how it works much more complicated.

Scientists are only beginning to understand how and when cells recognize the origin of a gene, but we can see again that the *Gattaca* vision needs considerable revision. Instead of simplifying our understanding, the “genetic revolution” has opened our eyes to the amazing complexity of our inheritance.

### **Ad infinitum . . .**

We cannot “find” the roots of altruism, or any other trait. We cannot pin it down to a single gene, a single evolutionary purpose, or even a particular set of causative agents. Just when we think all the aspects of a particular behavior have been accounted for, more layers of complexity and interaction come to light. Simplified thinking—and even scientific theories—that credit one factor exclusively out of an array of inputs can lead to distorted versions of reality.

And yet, sometimes genetics really is that simple—such is its unpredictability. *Gattaca* got something right. Although Vincent and Anton have not spoken in over a decade when Anton is presented with the opportunity to send his brother to prison, their bond—an indescribable bond—remains. After careful deliberation, Anton helps cover Vincent's missteps instead of abandoning his kin. Selfish or selfless, reasonable or unreasonable, our senses find a way to prevail.

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