Spence, J. T., & Helmreich, R. L. (1978). *Masculinity and femininity: Correlates and antecedents*. Austin: University of Texas Press.
Swaab, D. F., & Fliers, E. (1985). A sexually dimorphic nucleus in the human brain. *Science, 228*, 1112–1115.
Turkheimer, E. (1991). Individual and group differences in adoption studies of IQ. *Psychological Bulletin, 110*, 392–405.
Udry, J. (1988). Biological predispositions and social control in adolescent sexual behavior. *American Sociological Review, 53*, 709–722.
Wilson, J. Q., & Herrnstein, R. J. (1985). *Crime and human nature*. New York: Simon and Schuster.

CHAPTER 7

WHY FAMILIES HAVE LITTLE INFLUENCE

Imo spat out the sand clinging to her sweet potato, put it into the sea, and rubbed it vigorously with her free hand. She ate the cleaned potato, enjoying its salty taste. Nearby, Nimby watched—and thrust her potato into the sea. She didn't get all the sand off, but it still tasted better than ever before. The two young playmates' example taught others; soon their age-mates, both male and female, had caught on to the potato-washing routine. Imo's mother also learned, and soon was teaching potato washing to Imo's younger siblings. Imo's father, though he enjoyed a reputation for toughness and leadership, was too stubborn to try the new trick.

The potato-washing clan members were not humans, of course. They were rhesus monkeys inhabiting the unpopulated Japanese island of Koshima, where curious researchers had provisioned the band with fresh sweet potatoes by leaving them on a beach. Although my rendition has taken some literary license, it holds true to the basic events (Kummer, 1971). Imo was the name assigned to the brilliant monkey who first came up with the idea of washing the potatoes, and later discovered that sand and grain could be separated by throwing them onto water. Her potato-washing innovation was copied first by other juvenile males and females, and then by older females (about 18% initially), who passed it on to their offspring. Adult males failed to pick it up, partly because they had less contact with feeding juveniles, but perhaps also because they resisted novelty in general. After a few years, potato washing was an established tradition among the Koshima monkeys, and the episode had moved into the lore of the social sciences.

But one lesson of the Koshima monkeys has been all but ignored in socialization science: Cultural transmission occurs *outside* the family

and *inside* the family with equal facility. Indeed, the direction of influence is "wrong" for these monkeys. The innovation was originated by a preadolescent female, who taught other juveniles, who then taught their mothers, who then raised their offspring in this new tradition. In the Koshima case history, parent-to-child transmission did occur; however, it was only one of several transmission pathways, and (at least initially) far from the most important.

This chapter discusses the following question: "Why does variation in family environments have so little influence on children's personality development?" To answer this question, the chapter works backward from this conclusion to the conditions that must be responsible for it—and moves a large intellectual distance, from individuals to the sweep of evolutionary and cultural history. The chapter takes several tacks. First, it suggests that human learning mechanisms are general with respect to informational source, and that a disposition to tie learning exclusively to a family source is unlikely to have evolved in humans; second, it explores methods of describing the cultural transmission of traits; third, it considers some of the forces maintaining genetic variability; and fourth, it considers the dual role of culture and genetics in the maintenance of human traits. The last topic leads necessarily to a high level of abstraction—probably one too high to be immediately applicable to solving such social problems as street crime, school dropouts, or poverty. But this level of abstraction is needed as an intellectual inspiration for middle-level developmental theories that may supply the practical and theoretical means to solve pressing social problems.

The Generality of Learning

A Thought Experiment

In physics, central insights sometimes come from "thought experiments." With all due respect to Albert Einstein, who practiced thought experiments more brilliantly than anyone else, let us carry family effects to a logical extreme. In our thought experiment, what is learned in a family context is weighted more heavily by the learner than what is learned from any other person. Concepts acquired by direct parental teaching, behaviors modeled by imitating parents, and emotional states induced by family life stay with children throughout their adult lives. Such effects are so robust that children never change the ideas, habits, or feeling

states acquired during early socialization. A situation like that depicted in George Bernard Shaw's *Pygmalion* is impossible: A young woman cannot give up her Cockney accent, her poor table manners, or her habits of thought, because what her working-class parents have taught her is fixed in her forever. Our thought experiment assumes that a tendency to learn from parents is a "hard-wired" instinct—in other words, that a "learn from parents only" gene has gone to fixation (i.e., 100%) in the human population, whereas its defeated "rival," the "learn from any source" gene, has gone extinct.[1]

Our primate ancestors probably did not possess a hard-wired disposition to learn just from their parents. My evidence, though indirect, is persuasive: No current monkey or ape species seems to be so tightly restricted in its learning capacities. Thus, for humans to possess such a powerful and inflexible disposition, the rare "learn from parents only" gene must have arisen by mutation in our evolutionary line, and then spread through the human population because it increased the inclusive fitness of its bearers. (The term "inclusive fitness" refers to whether the gene's bearers and their immediate relatives leave more offspring than others. Immediate relatives count [hence: "inclusive"], because they are more likely than unrelated persons to carry the same gene as a known bearer.)

Some features of parent–child transmission are certainly attractive. Family traditions are well preserved. And if parents make some innovation in a cultural tradition, then their children quickly and reliably reproduce it. If it is reproductively beneficial, the innovation may slowly spread through the population much as a gene would, because its bearers should enjoy larger family sizes than others, contributing more members to future populations.

A problem with a "learn from parents only" gene, though, is that its bearers will ignore innovations introduced by anyone other than their own parents. Thus if children learn from their parents to make arrows *without* dipping the heads in poison, they will continue to do so, even after others in the population discover poisoned arrowheads and teach this technique to their own children. In contrast, the bearer of the "learn from any source" gene will try the innovation and rapidly adopt it. Those individuals who carry the rare mutation for "learn from parents only" will fail to take advantage of many successful innovations. Furthermore, their innovations will stay in lines of parent–child descent, with children in each line able to adopt only the few innovations their own parents have managed to make. They will also miss innovations made by unit-

ing ideas from several unrelated adults. In general, their cultural learning will have properties of genetic change: It will be slow, depending on how many children innovative parents leave behind.

If a "learn from any source" gene is to thrive in human evolution, our thought experiment must assume some ability on the part of people to select those innovations that contribute most to reproductive success; otherwise, sticking with a family tradition may be better than trying an innovation of dubious value. Certainly, evaluating some innovations does not require great genius. A blow gun dart that flies straight is immediately better than one that doesn't. Small and large improvements in game-stalking practices, farming practices, and other areas important to survival and reproduction can be recognized for what they are—improvements. I do not claim that people consciously know the value of all cultural practices—(e.g., the benefits of cod liver oil for obtaining Vitamin D were probably not consciously understood by Eskimos); nevertheless, the value of many innovations is recognizable.

Benefits were probably seen even before people became as knowledgeable as they are today. In the "great leap forward" period, 40,000 years ago in western Europe, an explosive wave of cultural innovation occurred: Cro-Magnon people discovered art, musical instruments, tools with different and specific functions (e.g., needles, awls, spear points set in shafts, mortars, fishhooks, and rope), and trade, all in one historical moment (Diamond, 1992). I believe that the value of many of these cultural innovations must have been immediately perceived. Indeed, if rhesus monkeys, who are intellectually no match for our evolutionary ancestors, can acquire a simple but useful innovation such as potato washing, it is no great leap to infer that the bearers of a gene for "learn only from parents" should be outreproduced by the bearers of one for "learn from any source," whenever the latter have any sense at all about which innovations are best kept and which are best dropped.

Models of Cultural Transmission

Formal models can give some indication that *cultural* transmission from parent to child alone is unlikely. Carey (1991) tried to create a mathematical model of parent–child cultural transmission that would be similar to standard behavior genetic models, in which parental phenotypes directly affect children's environments. In his model, maternal and paternal cultural inheritance would be blended; that is, a child's environment would lie midway between the mother's and father's traits. For instance, a child exposed to a painfully shy father and a moderately shy mother would be regarded as having an environment midway between the parents' shyness levels. But Carey's cultural model produced some anomalous results. When parent-to-child cultural transmission was the only kind allowed, the regression coefficient linking parental traits to children's environments had to be exactly .50 for meaningful results to be obtained; no other values would do. Such an odd and unexpected restriction implies a logical flaw in his model. As Carey observed, this flaw may have been the failure to recognize multiple avenues of cultural transmission:

> It is indisputable from observation of human behavior that members of *Homo sapiens* are not constrained to imitate the behavior of only mother and father. Other conspecifics are also imitated. Perhaps a generalized mechanism for imitation evolved instead of a specific one for imitating one's parents. (1991, p. 442)

Figure 7.1 presents quick schematic diagrams illustrating various models of cultural transmission (Cavalli-Sforza & Feldman, 1981). In the top diagram (A), "vertical" transmission from parent to child is presented; here, cultural inheritance mimics genetic inheritance, and vice versa. The next diagram (B) shows "horizontal" transmission among age-mates. Childhood games like hopscotch pass along purely in this way, from one generation of children to another, without intervening assistance from adults. The third diagram (C) illustrates one form of "oblique" transmission, in which an unrelated person in the adult generation is a source of knowledge for a child. Rock stars and movie idols are clearly oblique transmitters to the next (not much younger) generation. The fourth diagram (D) represents oblique teaching influence, in which a single teacher transmits cultural knowledge to many individuals. This pattern applies formally to the media of television, radio, books, and newspapers, as well as to the typical educational model employed in most schools. Finally, the fifth diagram (E) illustrates a reversal in the vertical flow of culture—from child back to mother, father, and an unrelated adult. As in the case of the Koshima monkeys, the young may be the innovators and the old may be the imitators.

Sex role learning provides a direct illustration of the potential of multiple models to influence behavior. Of course, for most such behaviors, human instincts also guide children into the right channels. But what if a behavior appears that is so novel that children are unsure whether

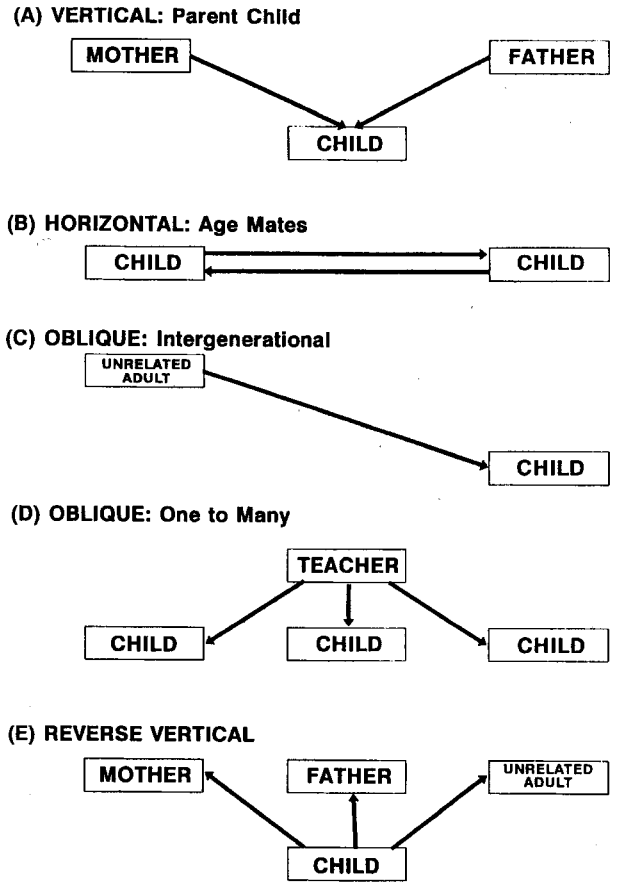


FIGURE 7.1. Trait transmission models.

it is male- or female-typed? How will they know what to do? As Chapter 6 has shown, some theories of sex role development emphasize socialization influences in the family, but children's acquisition of sex-appropriate behavior is robust. When parents avoid stereotypic behavior in their daily lives, children often display sex-typed behaviors anyway.

A fair rule—one unlikely to lead youngsters badly astray in most circumstances—is to imitate what *most* same-sex individuals do. This majority rule implies attention to what is typical for one's sex, and inattention to the potentially misleading influence of a few odd, sex-reversed

parents or teachers, whose guidance may well lead away from cultural norms and away from average biological proclivities.

David Perry and Kay Bussey (1979), working in the tradition of the famous social learning theorist Alfred Bandura, created a choice experiment to test the influence of multiple models. They employed eight adult assistants—four females and four males—to make arbitrary choices between pairs of small items that were not obviously sex-typed. The adult might face a choice between an apple and a banana (as far as I know, fruits remain psychologically sex-neutral, although adult imaginations and botanical theories may not be so restrained). But for the young children in the control condition at least, no preference was shown for one choice over the other. The other experimental conditions were designed to create preferences. In one, the same-sex majority ruled (the four men made one choice, whereas the four women made the other), giving the children a clear direction as to a sex-typed social standard. In another condition, the adults were divided: Six adults made a sex-typed choice, but two, one male and one female, broke ranks with their sex and chose oppositely.

Adult examples were earnestly copied when they were consistent with the children's own sex. Table 7.1 shows the number of male-typed choices made by the children (out of 16 opportunities). When same-sex majorities ruled, the young boys made an average of 14 male-typed choices; the young girls made just 3 (i.e., they made 13 female-typed choices). As within-sex consensus began to break down (in the condition where three same-sex adults made the "right" choice and one the "wrong" choice), less imitation was shown (boys 12, girls 6). Finally, in the control condition, in which the children lacked any models, the preferences were about evenly split: The boys made the male-typed choice 9 of 16 times, the girls 8 of 16 times (a nonsignificant difference). Ironi-

TABLE 7.1. Children's Following of the Majority Rule in Making Choices

Child	Consistent same-sex adult models	Inconsistent adult models	No adult models
Boy	14	12	9
Girl	3	6	8

Note. Average number of male-typed choices out of 16 choice opportunities. Adapted from Perry & Bussey (1979). Copyright 1979 by the American Psychological Association. Adapted by permission.

cally, Perry and Bussey included in their article's title that "imitation is alive and well" in the origin of sex differences. And so it is. But without much fanfare, the two social learning theorists placed parent-centered conceptions of sex role learning in an early grave, as they recognized: "We would expect that children who initially adopt responses by . . . imitating a same-sex parent would ultimately drop the responses . . . if they eventually realize that no other same-sex individuals perform the responses but that many opposite-sex persons do" (1979, p. 1709).

From the current perspective, as children have more potential adult or child models, the weight on parental example becomes progressively diminished. Parents may seem pretty important to a 3-year-old child, who has seen few other adults or children. But to a 16-year-old, the weight on the parental example should be no more than $1/n$, where n is the total number of adult or child models relevant to a particular trait. Like any other source of information, parents make a contribution—though one of no greater *a priori* strength than any other (except that the parent–child emotional bond may pull a child's attention initially), and on many occasions one of even less strength, because acceptance will be biased by "majority rules" and by the functional value of particular knowledge and behaviors. As many newly arrived immigrants have discovered, children may learn the folkways of a new country better than those of an old: For finding mates and employment, the folkways of the new country will work, whereas those of the old country will often fail miserably.

The Transmission of Emotions?

Thus far, the examples of cultural transmission have dealt with the transmission of knowledge or the imitation of specific behaviors. Yet theories of family influence may focus more on the emotional aftereffects of familial socialization—on scars believed to have been inflicted by parents' emotional neglect or cruelty, whether conscious or unwitting. "Ah," say the proponents of familial influences, "you can't deny the powerful legacy of a family's love or denial."

If only the world would follow our emotional intuitions! One problem—perhaps not self-evident to northern Europeans, for whom nuclear families are typical ones—is that worldwide family patterns are as varied as pre-European-contact societies. In much of Africa, as described

by anthropologist Patricia Draper (1989), the family "unit" consists of a mother and female relatives. Men play a less direct emotional role in the lives of their children than fathers do in the lives of northern European children. Indeed, biological fathers and mothers are often both physically absent, as children are cared for by kin (primarily older women who are no longer in their reproductive years). Nor are attachments to female relatives simple: As a child is shuttled from the mother to the other female kin and back, multiple attachments and sources of emotional bonding form. Furthermore, even when children are living with their biological mothers, older siblings often adopt the role of child minders. Polygyny, a common form of marriage in Africa, places a father at some psychological and structural distance from a mother and her children; he often sleeps in a separate hut, either alone or with his adolescent sons. A notion of a dense family crucible in a child's emotional life—with a child's attachments confined to a biological mother and father—is no more true, in general, of humans than the idea that the Neanderthals got their news on television.

I may have drawn contrasts between the northern European pattern and the African pattern too sharply. Although the nuclear family is held up as an "ideal" of family organization, more diverse forms are usually the reality in Western industrialized societies. Given recent high divorce rates, children often possess nonbiological relatives from new marriages—stepsiblings and stepparents. And northern European children possess uncles and aunts and grandparents, a larger family circle in which emotional attachments can form. Finally, even when no biological relatives are available, the many ways in which children can cope with unloving parents should not be underestimated. To argue a moment from anecdote, an acquaintance of mine had emotionally cold and distant parents, but found succor in the nearby Italian family of a friend. As a child, he became so close to the members of this other family that they habitually set a place for him at the table. And now, as an adult, his visits home mean visits both to his biological parents and to his "adoptive" ones.

But emphasizing the emotional complexity of "families" skirts the issue of "Why does variation in families have so little influence on emotional development?" An evolutionary reason is that genes that did not permit recovery from early emotional trauma—whether inflicted by parents or by others—would have been excluded from the population by the sieve of natural selection, whereas genes that permitted recovery

from early emotional trauma would have been favored. No organism can afford to be as brittle as an egg, cracked and unrepairable after life's first hardship. As said in the vernacular, "Life's a bitch, and then we die"; trauma, pain, and difficult trials can be avoided only by the extremely lucky, or by those who die very young. In reviewing her behavior genetic studies, Sandra Scarr (1992) has expressed this evolutionary perspective:

> Fortunately, evolution has not left development of the human species, nor any other, at the easy mercy of variations in their environments. We are robust and able to adapt to wide-ranging circumstances—a lesson that seems lost on some ethnocentric developmentalists. If we were so vulnerable as to be led off the normal developmental track by slight variations in our parenting, we should not long have survived. (pp. 15–16)

The converse message of this statement, though, is less encouraging. For when children seem to be following less desirable developmental paths (e.g., children who pick on classmates aggressively or who are extremely anxious), the traits may be heritable developmental outcomes that stubbornly resist familial actions attempting to change them. As Scarr (1992) has bluntly reminded social scientists,

> . . . for children whose development is on a predictable but undesirable trajectory and whose parents are providing a supportive environment, interventions have only temporary and limited effects. . . . Should we be surprised? Feeding a well-nourished but short child more and more will not give him the stature of a basketball player. Feeding a [child with a] below-average intellect more and more information will not make her brilliant. Exposing a shy child to socially demanding events will not make him feel less shy. (pp. 16–17)

Fundamentally, the lesson is that desirable and undesirable traits alike are maintained in human populations neither by parental intention nor by parental blunders; etiology is more complex and multifaceted than such a simplistic and overly optimistic picture would suggest. As Chapter 5 has indicated, the typical environmental explanations of social pathology—social class, child-rearing styles, and others—take their explanatory power from genetic variation underlying behavioral variation in modern industrialized societies. If we are to understand the maintenance of traits, we must learn more about the sources of this genetic variation. Before turning to this task, let us consider an example of how socialization science can examine various models of cultural transmission.

The Diffusion of Cigarette Smoking: Examining Models of Cultural Transmission

Cigarette smoking is an interesting behavior for contrasting different models of cultural transmission. Cigarettes are an old cultural innovation, discovered first by Native Americans and then spread around the rest of the world (Ferrence, 1989). Manufactured, rolled cigarettes were used in the United States in the 1800s, but smoking did not become popular among adult males until after World War I and among adult females until after World War II. Smoking was first popular among the better-educated segments of the population, and then diffused to less well-educated groups. However, in a complete reversal of smoking patterns for people born early in this century (before 1910), smoking in younger groups is much more common today among the poorly educated than among the well educated.

Cigarettes (by virtue of containing a physiologically addicting substance, nicotine) manage to promote themselves, but smoking also has cultural meaning to young adults, who are certainly unaddicted during the early stages of experimentation with cigarettes. These cultural meanings, as well as physiological pleasure, give cigarettes their "functional value" that maintains them in the population. But each new generation is naive to cigarettes. From age 10 to about age 20 years, individuals either become regular smokers, experiment with cigarettes and then quit, or avoid them altogether. Almost no one—not even a middle-aged man in a full-blown midlife crisis—begins smoking later in life. Although he may buy a red Porsche, he doesn't try Camel cigarettes for the first time.

As we have seen for personality and intellectual traits, socialization scientists most often refer to social influences in the family as the cause of the intergenerational transmission of smoking behavior. And as usual, they use only weak and ambiguous evidence: the well-replicated association of smoking in a biological parent with smoking in a biological child. Rates of smoking in the offspring of smokers can be two to four times those in the offspring of nonsmokers.

But this interpretation foolishly neglects to consider the genetic component of parent–child similarity. Table 7.2 summarizes reports of two twin studies, an adoptive study, and a family study. In all these studies, the offspring of smokers were adults at the time they were surveyed. Smoking's heritability averaged 43%, whereas smoking's rearing environmental variation was close to zero. In other words, effects of rearing variation (e.g., parents' lighting up or not, or having cigarettes in the

TABLE 7.2. Rearing Effects for Cigarette Smoking?

Type	Heritability (h^2)	Shared rearing variation (c^2)	Citation
Family	42%	N/A	Eysenck (1980)
Twin	36%	<0%	Carmelli, Swan, Robinette, & Fabsitz (1990)
Twin	50%	<0%	Swan, Carmelli, Rosenman, Fabsitz, & Christian (1990)
Adoptive	N/A	<0%	Eysenck (1980)
Mean	43%	0%	

Note. In Eysenck (1980, p. 242), the biological parent–child correlation was .21 (n = 533 pairs); the adoptive parent–adoptee correlation was –.02. In Carmelli et al. (1990, p. 70, Table 3): adjusted cigarettes, MZ twin r = .32, DZ twin = .14 (n = 2,390 MZ twin pairs and n = 2,570 DZ twin pairs). In Swan et al. (1990, p. 45, Table 3): adjusted smoking, MZ twin r = .42 and DZ twin r = .17 (n = 176 MZ twin pairs and n = 184 DZ twin pairs). N/A, not applicable.

home or not) were nil by the time the children had reached adulthood. In Eysenck's (1980) report on adoptees, the smoking correlation of *biologically unrelated* parent–child pairs was essentially zero (r = –.02). Parental smoking may influence a child's risk through genetic inheritance: The role of parents is a passive one—providing a set of genes at loci relevant to smoking risk, but not socially influencing their offspring.

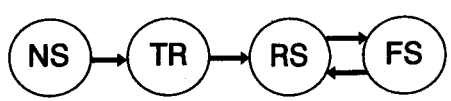
Socially, learning to smoke is primarily a peer group process: Age-mates provide cigarettes, smoking opportunities, and words of encouragement. As established in many studies, nearly all adolescents first acquire smoking by experimenting with cigarettes with their friends and acquaintances—usually other adolescents close in age to themselves. After the smoking habit has been established, however, the presence of others is no longer as crucial for maintaining it; some evidence suggests that adults smoke with a goal of keeping a consistent blood level of the psychoactive substance, nicotine. In the cultural transmission models of Figure 7.1, note that smoking habits correspond most closely to horizontal transmission among age-mates (model B), not to vertical parent–child transmission (model A).

With my colleague Joseph Rodgers, I recently modeled mathematically the horizontal "contagion" of various adolescent-onset behaviors, including alcohol use, smoking, and sexual intercourse (Rowe & Rodgers, 1991a, 1991b). Our models share the basic structure of mathematical models of the spread of cultural innovations or that of infectious dis-

ease organisms (see Cavalli-Sforza & Feldman, 1981). For this reason, we sometimes call them "epidemic" models, in an analogy with epidemic disease. People who have not adopted a new behavior are the "susceptibles" in a population; those who have adopted it are the "carriers." But such analogies are not intended to be taken literally; we certainly do not mean to make a moral judgment about smoking or any other behavior by means of this analogy. The analogy is an aid to understanding, because concepts such as the (population) mixing of "carriers" and "susceptibles" puts the emphasis on horizontal transmission, and away from the vertical transmission models so dominant in socialization science.

Consider the horizontal spread of smoking in adolescent groups (Rowe, Chassin, Presson, Edwards, & Sherman, 1992). Our smoking model assumes that four smoking stages exist: (1) "nonsmoker," (2) "trier" (defined as smoking no more than once per week), (3) "regular smoker," and (4) "former smoker." As shown in Figure 7.2, transitions exist between these stages: A child may move from nonsmoker to trier, from trier to regular smoker, and back and forth between former smoker and smoker, as smokers can both quit and (unfortunately) relapse. In a transition from nonsmoker to trier, we assume that social contacts are involved. If John is a nonsmoker, for example, he must meet a cigarette-using friend or acquaintance before starting to smoke himself.

This assumption is fairly noncontroversial: According to my research, nearly 90% of American adolescents initiate smoking in a small group of one to three friends and acquaintances. Of course, not everyone obeys a mathematical model—but because the exceptions may be relatively rare, they are omitted here. We may also assume that the transition from trier to regular smoker does not involve any kind of social contact. According to this model, kids get "hooked" as a result of suffi-



NON SMOKERS
TRIERS
REGULAR SMOKERS
FORMER SMOKERS

FIGURE 7.2. Smoking stages.

cient smoking experience, not because of repeated social pressures to do so. This assumption may be more controversial, but it works well in our mathematical model (Rowe et al., 1992).

To give readers a feel for the model, the proportion of new triers is assumed to depend on social contacts with smokers. Mathematically, what the model says is this:

$$(1) \quad \text{New } P_{\text{TR}_{a+1}} = TP_{\text{S}_a} \cdot P_{\text{NS}_a}$$

where T is a rate constant, $P_{\text{TR}_{a+1}}$ is the proportion of new triers at age a plus 1 year, P_{S_a} is the proportion of smokers (including both triers and regular smokers) at age a years, and P_{NS_a} is the proportion of nonsmokers at age a years. If few smokers exist in a population at a given time, clearly smoking rates can increase only slowly. As new smokers are added to this population, contacts between smokers and nonsmokers become more common, and the rate of spread increases still further.

The rate of spread also depends on the constant, T. When T takes on a larger value, spread is more rapid. In equation (1), T represents a population average; of course, some individuals have greater T values than others, either because they are more susceptible or because they have more contact opportunities.

What determines the rate constant? One influence is the probability that a person will try cigarettes, when she is given the chance. Given the prevalence of smoking, T is probably greater for poor adolescents than rich ones, reflecting different expectations and values surrounding smoking. At the individual level, T may vary with heritable personality traits; for instance, individuals who are genetically more impulsive probably have greater T values than ones who are not. T can also depend on the degree of social mixing: The more encounters between smokers and nonsmokers, under circumstances where experimentation with cigarettes is possible, the greater the value of T for a particular population. In a sense, a dissection of T uncovers the many causes of smoking, and hence the conceptual complexity of this single number.

The transition from trier to regular smoker requires a different mathematical representation:

$$(2) \quad \text{New } P_{\text{RS}_{a+1}} = jP_{\text{TR}_a}$$

where j is a rate constant, $P_{\text{RS}_{a+1}}$ is the proportion of regular smokers at the next age, and P_{TR_a} is the proportion of triers at the prior age. Here,

a constant proportion of triers become regular smokers at any age. For example, if $j = .2$ and $P_{\text{TR}} = .6$, then the proportion of those becoming regular smokers would be .12.

Difference equations can be used to represent our model for the children of smoking and nonsmoking parents.[2] There are four equations for the children of smoking parents, and four for those of nonsmoking parents. We used these equations to fit data on smoking collected by Laurie Chassin and Clark Presson, and their colleagues, in the college town of Bloomington, Indiana (Rowe et al., 1992). The sample size was about 5,000 students in grades 6–12. The model was fitted to proportions—that is, the proportion of nonsmokers, triers, regular smokers, and former smokers in each grade (this was done separately for the children of smoking and nonsmoking parents). Sixth-grade proportions were fed into the model equations, which then returned *predicted* ones for grades 7–11. On the assumption that social contacts predominate within sex, males and females were fitted separately.

For simplicity, predictions of former smokers have been omitted from Table 7.3, which shows the predicted proportions of nonsmokers, triers, and regular smokers in grades 7–12 separately for the daughters of smoking and nonsmoking parents. About half our sample had at least one smoking parent, so the two sets of population growth curves represent about equal numbers of children. As shown, the children of smoking parents were about twice as likely to smoke as those of nonsmoking parents. Although the increase rates appear fairly similar for the two kinds of children, a mathematical analysis revealed that the *rate constants* were greater for the children of smoking parents than for those of nonsmoking parents. In the nonsmoker-to-trier transition, they were .52 and .32, respectively; in the trier-to-regular smoker transition, they were .14 and .10, respectively.

Thus, vertical "influence" (parent to child) is described in our model as greater susceptibility to peer influence. Furthermore, the familial role is interpreted here as a genetic one, attributable to children's inheritance of different personality traits. The values of both rate constants may be changed by familial background traits—one reflected in the social influence of age-mates, the other in the development of long-term psychological dependence on smoking. The literature on smoking allows one to postulate what these heritable traits may be. For example, sensation seeking and extraversion have been statistically linked with smoking behavior; they may influence the nonsmoker-to-trier transition. The other transition, from trier to regular smoker, may depend in part on other

TABLE 7.3. Predicted Smoking for Female Children of Smokers and Nonsmokers

School grade	Nonsmokers	Triers	Regular smokers
	Smoking parents		
7	.62	.31	.04
8	.55	.34	.07
9	.46	.38	.11
10	.36	.41	.15
11	.28	.45	.18
12	.20	.46	.22
	Nonsmoking parents		
7	.74	.12	.00
8	.68	.17	.01
9	.61	.23	.03
10	.54	.30	.05
11	.45	.36	.07
12	.37	.41	.10

Note. Epidemic model fit, $\chi^2 = 16.6$, $df = 25$, $p > .05$. A small chi-square value indicates a good fit. Parameter values: smoking parents, $T_1 = .52$, $j_1 = .14$; nonsmoking parents, $T_2 = .32$, $j_2 = .10$. Relapse rate $v = .05$; quitting rate $u = .18$.

heritable traits, including the body's own physiological adaptation to nicotine.

Like the monkey Imo's potato-washing innovation, smoking behavior cascades through society via horizontal transmission. Heritable trait variation is relevant to the flow of these social innovations through society, but variation in how children are reared may have little relevance. Rather, the time scale for social influences on smoking is shorter than a biological generation. Changes in price, in availability, and in social knowledge of cigarette's harmfulness to health diffuse quickly through society via the pathways of Figure 7.1, altering cigarette usage patterns. The genes underlying susceptibility to cigarettes change much more slowly, at the pace of biological rather than cultural evolution. And finally, cultural and biological evolutionary pathways may interact—a "coevolution" of changing cultural innovations and changing gene frequencies. That is, a cultural innovation may reduce its adopters' average number of surviving children (and hence lower their biological fitness), or it may increase reproductive rates and survivorship.

At first glance, one might think that the health risks of smoking make it biologically maladaptive. But smoking's harmfulness to fitness is probably weaker than is commonly supposed, because its ill-health effects may be delayed beyond the reproductive years. In addition, one social meaning of smoking is the early initiation of adult roles and behaviors, including sexuality. Teenage smokers may adopt a general lifestyle that leads to earlier and more frequent childbearing. Thus, contrary to common belief, smoking may be biologically adaptive (though undesirable) for young adults, at least over the short term, in the current cultural climate of the United States.

In summary, smoking must be understood in terms of a "diffusion–exposure" model of the spread of cigarette use and its attendant beliefs and attitudes. An "initial use" theory must explain the beginning of experimentation with cigarettes; an "amount–persistence" theory must explain why some adolescents who experiment with cigarettes eventually cease smoking, whereas others become addicted (see Carey, 1992, for another mathematical approach to these ideas). Most broadly, effects of cultural innovations will be played out against long-term population changes in gene frequencies—a biological concomitant to cultural change.

Forces Maintaining Genetic Variability

Why Are Some People "Bad"?

This broader evolutionary view reveals a limiting myopia in socialization scientists' understanding of how "bad" behavioral dispositions persist. No modern theory of human evolution can possibly postulate a simple human nature, lacking self-interest as well as social interest in others, lacking antisocial tendencies as well as prosocial ones, or lacking motives that conflict as much as ones that complement.

A complete analysis of human nature, of course, falls outside this book's scope. But some discussion of the evolutionary forces maintaining genetic variability is necessary if socialization science is to move from family-based theories of trait maintenance and transmission to more powerful and general coevolutionary ones.

Socialization scientists' error has been to ignore completely the role of differential reproductive rates in maintaining behavioral variation. If genetic variation determines trait variability, then the crucial question is that of which genes are put into the next generation. The answer in

turn depends on the relative reproductive rates of the genes' bearers, and on the survivorship of the bearers' offspring. Given the moral neutrality of the guiding hand of evolution, "bad" traits can evolve as easily as "good" ones. Indeed, typical animal behavior patterns contain many examples of biologically evolved traits considered morally reprehensible in human societies—from "forced extrapartner copulation" in mallard ducks (something loosely akin to human rape) to the killing of unrelated infants by male troop leaders among rhesus monkeys.

To take a less extreme trait as an example, Marten deVries (1984) investigated infant temperament among the Masai, a nomadic, Nilo-Hamitic people living in central Kenya; he classified babies as either "easy" or "difficult" in temperament. The 10 easiest and 10 most difficult babies were chosen for further study. deVries then left Kenya for about a year before returning to continue his field work. During the interim, a devastating drought caused a heavy loss of cattle (the main food resource for the Masai), and child mortality rates increased sharply. On his return, deVries managed to relocate 13 of the 20 originally studied families; he discovered that five of seven babies classified as "easy" in temperament had died, whereas only one of six difficult ones had met the same fate ($p < .07$). deVries speculated that the "difficult" babies might have outsurvived the "easy" ones because, under a condition of resource scarcity, their noisy demands brought them the additional amount of parental care and food that their quieter age-mates never received. In the entire Masai sample, the correlation of difficult temperament with larger body size endorsed deVries's "squeaky wheel" hypothesis.[3] Thus, traits that parents of young infants may regard with some dismay and apprehension may, under certain conditions, be evolutionarily favored.

The capacity of "bad" traits to evolve is even more apparent when one considers how reproductive success can be balanced evolutionarily against the dark force of mortality itself. Young males of many species are more violent and more willing to take high-stakes risks than older, established males or females. Of course, males deciding not to fight for social status and mating opportunities could do so, eating jungle fruits and surviving into admirable dotage; however, their behavioral tendencies would lack any genetic representation in the next generation. The same conditions may apply to adolescents and young adults in human societies. If the more aggressive, risk-taking adolescents had an 80% survival chance, but fathered (on average) 3.0 children, their reproductive success would surely exceed that of more cautious males with a 95%

survivorship and, on average, 2.3 biological children. Evolutionary processes can easily maintain trait prevalences, though they may upset moral sensibilities. In both U.S. cities and suburbs, adolescents may unconsciously use high-risk behaviors to increase their reproductive chances, as violent behavior and teenage fatherhood do occur together in adolescent males.

Hawks and Doves

Game-theoretic ideas give us powerful metaphors for real evolutionary processes, ones probably operating daily in our societies to produce behavioral variability. The game metaphor of "Hawks and Doves" describes the ability of evolution to maintain mixed traits or mixed motives in a population (Dawkins, 1989; Maynard-Smith, 1982). For a human analogy, imagine two adolescent boys confronting each other over a girl they both like. The boy taking the "Hawk" strategy will fight. In contrast, a boy adopting a "Dove" strategy will make a few threats, but if a real fight then ensues, he will flee quickly from the scene. For simplicity, we may assume that boys act either as Hawks or as Doves for their entire lives. But the model works just as well if every adolescent boy has both motivational systems, but spends part of his day as a Hawk and part as a Dove. Thus a population may be composed of two-thirds Hawks and one-third Doves, or of people who behave as Hawks two-thirds of the time and as Doves one-third of the time.

For both Hawks and Doves, payoffs must be in some currency that ultimately counts as greater or lesser reproductive success. Fighting may cause injuries that reduce average success in mating and fathering children. When Hawks meet, both may get hurt. When Doves meet, they may posture and threaten, but ultimately both may leave with some social prestige intact. Suppose that winning a fight enhances social prestige. When a Hawk meets a Dove, the outcome is foreordained: The Hawk wins the fight and enhances his social status, while the Dove leaves defeated. These ideas can be translated into a set of numerical "payoffs" for both the Hawk and the Dove lifestyles:

−.3 to the Hawk meeting another Hawk
.6 to the Hawk meeting a Dove
0 to the Dove meeting a Hawk
.2 to the Dove meeting another Dove

In this interpretation, these payoff numbers represent real gains or losses of social prestige that ultimately increase or decrease fitness. For convenience, the units are treated as though they were scaled to reproductive rates—so that, for instance, the –.3 means that fighting Hawks suffer, *on average*, the loss of one-third of a child. (Clearly, no Hawk loses exactly one-third of a child—some have one child fewer than they would if they had never fought, some none fewer, some two fewer, etc.) The units can be rescaled relative to average reproductive rates in the full population: If the average reproductive success is 2.0 among all adolescent boys, then a Hawk's encounter with another Hawk reduces it to 1.7. His encounter with a Dove increases it to 2.6.

Overall social prestige and reproductive success thus depend on a whole history of social contests. Under such conditions, a Hawk's payoff will depend on how often his contests are with another Hawk (in which he may be injured), and how often with another Dove (in which he surely wins). If the population were divided into "p" Hawks (a proportion between 0 and 1) and "q" Doves, and if encounters with a Hawk or Dove partner take place essentially by chance (unlikely in the real world, but a useful simplification for demonstrating general principles), then p proportion of the time a Hawk encounters another Hawk, and q proportion of the time a Hawk encounters another Dove. Weighting the *payoff per encounter* by the relative frequencies of encounters yields a payoff for each life history. Living as a Hawk leads to this equation:

$$(3) \quad \text{Hawk payoff} = -.3p + .6q$$

Because a Dove will also encounter other Hawks and Doves at the same relative rates, the equation is the same except for the payoffs:

$$(4) \quad \text{Dove payoff} = 0p + .2q$$

Using these two equations, and substituting different values for p and q (where $q = 1 - p$), one can derive the payoffs for the two life histories according to different population compositions. Table 7.4 presents results for equations (3) and (4) for proportions from one-seventh Hawks to six-sevenths Hawks. When Hawks are rare relative to Doves, they do better reproductively; that is, they average 2.47 children, whereas the Doves average just 2.17 children. In contrast, when the Doves are relatively rare, they do better reproductively than Hawks, averaging 2.03 children versus Hawks' 1.83. When the population composition is four-

TABLE 7.4. Reproductive Payoffs in Hawk and Dove Contests

Population composition		Average lifetime births	
Hawks	Doves	Hawks	Doves
1/7	6/7	2.47	2.17
2/7	5/7	2.34	2.14
3/7	4/7	2.21	2.11
4/7	3/7	2.09	2.09
5/7	2/7	1.96	2.06
6/7	1/7	1.83	2.03

sevenths Hawks and three-sevenths Doves, the two life histories do equally well reproductively (payoff = 2.09 in each case; see Table 7.4).

Therefore, over generations, differential reproductive rates always take population compositions to this one equilibrium. The dynamic nature of this process can be modeled with a computer program a few lines long.[4] As shown in Figure 7.3, a population starting with 95% Hawks and 5% Doves reaches equilibrium in about 30 generations. One starting with the opposite composition (95% Doves and 5% Hawks) takes a little longer, reaching equilibrium after about 60 generations. After about 80 generations the population composition would be completely stable, except if perturbed.

This process is called "frequency-dependent selection." Rare behavior patterns enjoy increased reproductive success while they are rare, but reduced success as they become more common. With several such behavior patterns, an equilibrium point may exist at which all behaviors possess equal reproductive success. Unless research investigators can long outlive their subjects, verifying frequency-dependent selection in human populations will be extremely difficult, if not impossible. True, the assessment of *current* selection is possible in modern societies, and this evidence could be used to strengthen the case for a selective process. But long-term selection would be extremely difficult to study, given the weaknesses of historical data, as well as the many ambiguities that must accompany reconstruction of the Pleistosine period in which humans evolved.

Nevertheless, the idea of such a selective process maintaining genetic variability in a context as rich and as diverse as human societies is intuitively appealing. Consider that street criminals offer a rough parallel to our game-theoretic Hawks, and law-abiding people to our Doves.

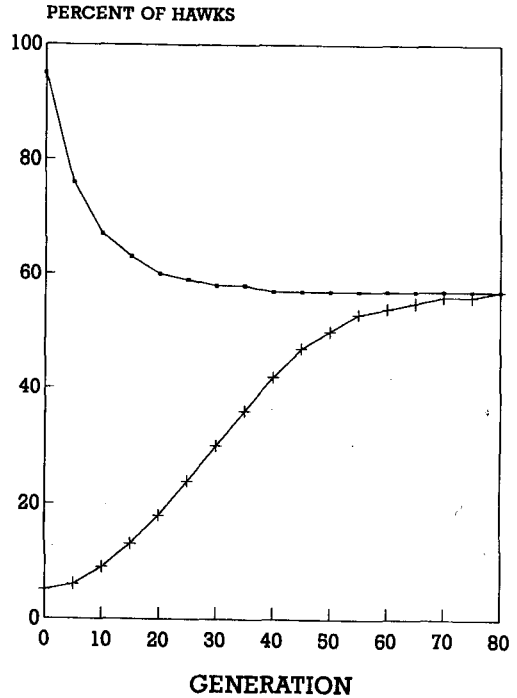


FIGURE 7.3. Approach to equilibrium in the Hawks and Doves game.

Like Hawks and Doves, people at extremes of criminality or law obedience fail to switch to the other life history, or at least switches are quite uncommon (Loeber, 1991). The tendency toward crime is also heritable (Rowe & Osgood, 1984). And in sharp distinction from the case for schizophrenia or severe mental retardation, criminals have children at reasonable rates, often having them earlier in life than noncriminals (Robins, 1966). These empirical observations—a heritable and relatively stable life history, and a lack of reproductive loss—meet the requirements for real-life contests between Hawks and Doves. For the full process to hold, one must further imagine that criminals do better reproductively when they are rare (perhaps when society is less vigilant), and do more poorly when they are common (when society may be more vigilant, and when their mutual encounters would extract their own reproductive cost).

Evolutionary biology is now receiving some recognition in the environmentalist strongholds of the social sciences. Two sociologists, Lawrence Cohen and Richard Machalek (1988), also applied the concept of

frequency-dependent selection to crime. They argued that a population of cooperating and productive individuals, in which exploitative individuals were rare, would invite "invasion by opportunistic alternative strategists" (p. 481). Hence, populations composed of both productive noncriminals and exploitative criminals should be the rule rather than the exception, and in this way, "crime can be said to be 'normal' in populations" (p. 481). Cohen and Machalek saw that criminal strategies can be understood in the same dynamic way as Hawk-versus-Dove contests: as a competition of behavioral alternatives, with success at least partly dependent on the rarity or commonness of the different strategies themselves.

Nonetheless, the form of these ideas offered by Cohen and Machalek (1988) de-emphasized heritable traits. The relevance of birth rates to passing criminal dispositions from one generation to the next was not mentioned. Indeed, Cohen and Machalek described most criminal acts not as results of "genetic selection," but rather as the results of "strategies employed by motivated persons, often cognitively aware that their behavior violates normative rules" (p. 477). They further imagined a situation in which two populations would have nearly identical traits of "age, gender, IQ, arousal levels, ethnicity, body type, values, and so forth" (p. 496). In such perfectly matched populations, different behavioral strategies could still spread and find their own equilibriums, but through cultural rather than genetic transmission.

The theory of frequency-dependent selection recognizes that genetic and cultural systems are *dual* modes of transmission, each operating with some independence of the other. One may find nongenetic reasons why criminal and noncriminal strategies may have different frequencies in two populations. For example, a novel form of criminal behavior (e.g., "computer viruses") may be introduced in one population and not but not another. This partial independence, though, fails to excuse ignoring biology in criminal behavior, as there is ample evidence of its importance. In reality, individuals do differ in heritable traits that make criminal strategies more or less attractive. Understanding criminality demands that the ideas of cultural and genetic transmission be considered simultaneously. In summary, although frequency-dependent selection is difficult to prove, the Hawks–Doves metaphor is appealing because it can account for the maintenance of antisocial behavioral tendencies without a return to false theories about variation in child rearing.

Evolution operates in many ways, not all of which are frequency-dependent. The existence of environmental "niches" that can be better

occupied by one genotype than by another is another general process that maintains variability. As discussed in Chapter 5, in human societies people of very different levels of measured intelligence occupy different social niches. They can earn enough income to support families, despite their disparate economic activities. Thus, the existence of different occupational roles tends to encourage continuing genetic variation in IQ; conversely, genetic variation in IQ tends to create occupational niches, which are filled by people of different levels of ability.

Further Sources of Variation

Social competition is not the only source of genetic variation. First, new DNA mutations in each generation may create variability. Second, biological pathogens exert selective pressure on all complex organisms. Tooby (1982), in particular, argues that the bulk of genetic variability in human populations may arise from pathogen-driven selection; this position may be extreme, but it is not without merit.

Biological pathogens—those awful germs that make us sick—are a major selective force because they weed out genotypes that succumb to them, while surviving genotypes manage to reproduce. One of the few documented cases of heterozygote advantage, sickle cell anemia, arises from the selective pressure of malarial disease on human populations (Durham, 1991). In the case of sickle cell anemia, individuals with one abnormal gene (s) and one normal gene (S) are less susceptible to malaria than those with two normal genes (SS). In the former individuals, red blood cells infected with the malarial organism become misshapen, and they are then destroyed by natural processes in the body. In normal individuals, infected cells are not removed, so that the risk of severe malarial disease (which may cause death) is greater. But the protection conferred by the sickling gene carries a heavy price: Individuals born with it in a double dose, genotype *ss*, will die (without medical intervention) in infancy or in early childhood. Other cases of genetic variation may also hold evidence of natural selection wrought by disease, although these instances are less well documented. In the well-known ABO blood group, the O gene may confer protection against smallpox; hence its greater frequency in European populations, in which the disease was once rampant (Diamond, 1990). Other blood group genes may tell similar stories for other illnesses.

Tooby and Cosmides (1990) speculate that this pathogen-driven genetic variation may also influence psychological traits—not by design, of course. Protective gene products work inside the cell to ward off disease organisms. Cumulatively, though, this genetic variation may affect the nervous system, and hence behavior. Thus viruses and bacteria may unwittingly create the genetic variation leading to psychological differences.

The Effects of Genetic Variation

According to Tooby and Cosmides (1990), psychological adaptations are more likely to be maintained by genes that are fixed (i.e., the same in everyone) than by those that vary. The reasoning behind their assertion is subtle: Genotypes get broken apart and reassembled in the process of sexual reproduction. In other words, the genotype of a parent is not the same as that of any child. But complex adaptations must require the cooperation and coordination of many genes scattered about the chromosomes to create, through interdependent steps, a finely tuned neurological system. If such a finely honed system existed in one parent, it would be taken apart when one random half of genes were passed to a child—because the exact combination of genes that had existed in a parent would not exist in a child, unless the genes were *already identical* in both parents.

The implication of this analysis is that most genetic variation fails to create new, complex adaptations, or new kinds of human psychology. Rather, it may modify universal psychological tendencies set by those genes humans share as a species. True, in the case of human gender differences (as discussed in Chapter 6), a genetic "switch" does exist on the Y chromosome that determines two different psychologies—male and female—but we have not found any corresponding bimodalities in psychological traits within the sexes.[5] Therefore, I agree with Tooby and Cosmides that genetic variation in the personality realm may change just response and perceptual thresholds under particular conditions, and that genetic variation in the intellectual realm may change just the *quantitative* capacity to assimilate and manipulate information mentally. In neither case does genetic variation change the underlying adaptive plan. That is, most humans react emotionally in broadly the same way to similar circumstances, even though the range of differences in emotional attachment between criminals and noncriminals may give the appearance of qualitatively different human psychologies. Similarly, the men-

tal processes used in reasoning are similar in most humans, even though the capacities of the very bright and very dull are so disparate as to give an appearance of qualitatively different human psychologies. Overall, much remains to be learned about the sources and functions of genetic variability. But certain points are undisputable: It exists; it is thrown up at the social system in each generation; and we ignore these facts at our peril.

The Need for Theories of Coevolution

The conclusion of this chapter is that a broader socialization science must be based on theories of gene–environment coevolution (Durham, 1991; Boyd & Richerson, 1985; Wilson, 1975; Lumsden & Wilson, 1983). These theories are currently undergoing the processes of discovery, analysis, and refinement, and their final form remains to be seen. In a seminal effort to put forth a theory of coevolution, Durham (1991) has reviewed existing coevolutionary theories and proposed a general model of his own. Like other theories of coevolution, Durham's distinguishes two independent but interacting systems of inheritance: genetic and cultural inheritance. They are independent systems because, though they both carry information from one generation to the next, they operate under different rules and by different mechanisms. The genetic system relies on the biological process of sexual reproduction and on genes—physical stretches of DNA. The cultural system relies on the transfer of information between human minds. The unit here is more difficult to define than a gene, and it certainly cannot be cut out of a molecular biologist's gel. Following Dawkins (1989), Durham proposes the concept of a "meme"—a unit of information resembling a "gene" in that it is transmissible between generations, is potentially variable in human populations, and is able to influence eventual phenotypes. In a cultural system, social innovation has a role corresponding to mutation in a biological system: It introduces new variability in memes. But the cultural system of inheritance also violates restrictions placed by biology on genetic inheritance. As shown in Figure 7.1, memes may move between minds in the same generations, from parent to child, from child to parent, and from teachers to students.

Any theory of coevolution must address two fundamental questions. First, what filters the replication of memes from one generation to the next? Second, what is the relation of biological to cultural fitness?

Durham (1991) has identified a number of mechanisms that influence the likelihood of cultural transmission. One is merely the rate of biological reproduction of subgroups that use a particular meme: Ideas may flourish as their bearers grow more numerous, much as genes may flourish. But ideas also replicate independently of growth in numbers of people; indeed, new innovations may sweep through a population without a change in its genetic composition. In the United States, an example would be the shift in the late 20th century toward regarding working women more favorably (Firebaugh, 1992).

Memes' succcess (replication) depends on their "cultural fitness"—that is, on their attractiveness to people in a particular culture, enabling them to spread there. Although many processes may influence a meme's cultural fitness, Durham has emphasized human choice and decision making. Durham calls this process "selection according *to* consequences," as opposed to the unconscious Darwinian "selection *by* consequences." It is the ability of people to compare different cultural variants, and to decide which ones possess the greatest utility for them.

Of course, in understanding the spread of particular memes, a key must be the criteria by which they are selected. Here, Durham (1991) has distinguished "primary values" from "secondary values." The former have a more direct and stronger biological component than the latter, but both are ultimately tied to biological evolution. Primary values include the instinctive love of parent for child, sexual attraction, and other emotions with a strong biological basis. The secondary values are elaborations of these primary ones through cultural evolution. In U.S. society today, for example, competing belief systems exist about the importance of marriage and the necessity of childbearing within the context of marriage; these are secondary values, each with a particular history of cultural evolution. In summary, theories of selective mechanisms are not well advanced, but uncovering them is essential for understanding cultural transmission.

The second question concerns the relation of cultural and genetic evolution. Genes may hold culture on a leash, but at issue is how short that leash is. At times, the leash may be quite long, because examples can be found in which cultural memes have damaged their bearers' biological fitness. Joined in a communal group, the 19th-century Shakers believed in community dancing and in sharing all material things, and they left us a legacy of elegant furniture (Halsey & Johnston, 1990). The Shakers, however, also eschewed sexual relations—a cultural practice that totally opposed their biological fitness, as they left behind no children.

Because the Shakers also converted few adherents, their opposition to reproduction ultimately led to their own demise.

More recently, some Western companies made the meme of bottle feeding and its associated technology available to Third World countries, and thus inadvertently caused an increase in child mortality over that of breast-fed children. The cultural fitness of the bottle-feeding meme conflicted in societies that adopted it with its lack of biological fitness. Durham (1991) has made the insightful observation that many instances of coevolutionary "opposition"—cases in which a meme hurts biological fitness—may result from memes imposed on one social group by another. This reasoning applies to conquests, which are often followed by the destruction of the defeated society's cultural traditions.

Although other examples of conflicts between culture and genetic evolution can be found, I agree wholeheartedly with Durham that in most instances the two forces for change are mutually supportive. That is, cultural memes chosen within a particular social group more often than not increase their bearers' reproductive success. Durham has called this kind of gene–meme relationship "enhancement," because bearers of memes with greater cultural fitness also possess greater biological fitness. Examples of such memes are cultural values opposing the marriage of close relatives—a practice that is biologically damaging because of the way inbreeding may cause genetic abnormalities. Cultural practices may also favor particular genotypes, as when dairying cultures made a genetic ability to digest milk an advantage (Durham, 1991).

In "neutrality," choice among memes lacks a relation to biological fitness. An example is the arbitrary relationship of a word to an object so signified: Whether one says "boat" or "bateau," for instance, makes little difference for biological fitness. Durham says more about the relation of cultural and genetic evolution, but we need not consider all his ideas here. What we need to remember is that a multiplicity of relationships can exist between genetic inheritance and culture. The leash metaphor may be extended from one leash to many leashes—some tight, others so loose as to be unnoticeable.

The purpose of this book is not to present a full-fledged theory of coevolution. Rather, it is to shake socialization science out of its complacent emphasis on the family as the bearer of culture, and on familial variation as the environmental cause of observed phenotypic diversity. In the light of data reviewed in the previous chapters, both assumptions appear to be false to the core, leading to a theory of social and personality development that is weak and has little ultimate intellectual power.

In this chapter, I have presented reasons for the weakness of the family-based model. I have found it unlikely that any genes could have become fixed in human populations that would restrict learning to parental example; the generality of learning diminishes family influence. Other transmission pathways should be routinely modeled in socialization science, and the "epidemic" model of smoking provides one such example (an extensive developmental literature on "peer influence" does this, although without explicitly using cultural transmission models). Both genetic and cultural avenues of trait maintenance must be considered. A number of biologically selective mechanisms—most notably, frequency-dependent selection—may account for the maintenance of difficult personality dispositions, ones commonly attributed to variation in family environments. Finally, a theory of phenotype development must identify both genetic and cultural components in a theory of gene–environment coevolution. Although the two interact, cultural memes have a transmission history separable from that of genes, and an understanding of human social behavior must adopt this dual perspective.

The diverse paths of cultural transmission also permit different genotypes to find their own environmental "niches." Each individual is potentially a recipient of information from parents, peers, teachers, unrelated adults, and so on, so that the range of information sampled broadens rapidly after early childhood. True, family effects can be stronger when no other opportunity exists for a particular kind of exposure. As mentioned in an earlier chapter, musical performance at the high school level shows some rearing influence. But violin lessons are usually first a parental idea, and schools lack programs to expose all children to classical instruments.

In some closed cultures, opportunities may be limited because all adults agree to impose fairly consistent social norms. For instance, among the Amish, a fairly rigid social structure can be maintained because all families agree to limit the exposure of young people to other alternatives. Nonetheless, even Amish teenagers "sow their wild oats"; in some cases allowance is made for their behavior, but in others the violator must leave. A process of expelling nonconformists may lead to genotypic as well as to cultural selection. For most forms of social behavior, though, the industrialized societies afford so many opportunities for sampling different memes that nongenetic parent–child resemblance is ultimately weak or nonexistent. In a few unfortunate cases, middle-class adoptive parents have been shocked when their adoptees discovered violent subcultures, which were previously unknown to them. The process that I

have variously called in this book "gene–environment correlation," "niche picking," or "an extended phenotype" needs much further exploration.

Current socialization science reminds me of a famous *New Yorker* cover showing a map of the United States dominated by Manhattan, with the remainder of the country barely represented. In much the same way that Manhattan crowds everything else out of the consciousness of New Yorkers, families dominate socialization science. Although New Yorkers may be incorrigible in their thought habits, it is time for socialization scientists to adopt a broader basis for their empirical and theoretical work.

Social and Policy Implications

Best-selling novels rarely have unhappy endings; similarly, books about genetics and social science usually close with some kind of sugarcoating about how biological traits are not really determined, or about how a heritable trait is malleable. These endings are not false ones, but in the context of this book's central discoveries they may be misleading, at least in emphasis.

The malleability of heritable traits cannot be doubted. Physical height is a commonly cited example. In the United States, heights have increased over the past century, despite the heritability of height (80% or better). Height gains are real and noticeable to designers of doors and airline seats (although further improvements can be made in these areas). But these historic gains do not mean that well-nourished middle-class children are still growing taller; indeed, groups of children born more recently are no longer putting on extra inches. Had twin or adoption studies recruited families with well-nourished and malnourished children, they would have shown that rearing variation affects height—a result that they do not find today.[6] With adequate nutrition now widespread, a major impediment to attaining one's genetic potential for height has been removed. Genetic variation in height remains, nonetheless, persistently operative.

The nostrums for many social problems involve recommendations that we rear our children differently. Ironically, for such policy recommendations to be at all effective, heritability must be much less important for the traits in question than is the estimate of the shared rearing component of variation. Consider two traits—one with a heritability of 45% and a family rearing influence of 25%; another with a heritability

of 10% but no family rearing variation. With the former, we would have a good idea of what social policy choices to adopt. We would advise parents to rear their children as do families with the "best" outcomes, whatever those methods may be (e.g., putting the children into cribs with fancy mobiles, or taking them to museums, or making sure that both parents stay home with them), because in this case 25% of trait variation is open to programmatic manipulation. The latter trait shows only a small amount of genetic variation and much unshared environmental variation—but how do we identify what unshared influences to change with our policy options? They may be anything from embryological development to a bad teacher. Shared rearing variation, not heritability, is a standard for the upper-bound influence of some social policies—those policies that change the environments of the most disadvantaged to be like those of the most advantaged.

This book concludes, however, that variation in shared rearing experiences is a weak source of trait variation. As with malnutrition, everything possible should be done to combat child abuse, child neglect, and other parenting wrongs. And as with height, doing away with the greatest harms may make some improvement in trait distributions. Many problem youths, though, come from the range of normal parenting variation, from families that are working- or middle-class and that are not extremely poor—in other words, from that range of diminished or nonexistent family influence. Changes in parenting styles may make only a small dent in the sum total of our social problems. Too, if social scientists come to accept these conclusions, the idea that the way academics raise children would really be best for everyone must be abandoned as well. If environmental interventions are to succeed, they must be truly novel ones, representing kinds of treatments that will be new to most populations.

These remarks will certainly call out Lewontin, Rose, and Kamin's (1984) "fire brigade":

> Critics of biological determinism are like members of a fire brigade, constantly being called out in the middle of the night to put out the latest conflagration, always responding to immediate emergencies, but never with the leisure to draw up plans for a truly fireproof building. Now it is IQ and race, now criminal genes, now the biological inferiority of women, now the genetic fixity of human nature. All of these deterministic fires need to be doused with the cold water of reason before the entire intellectual neighborhood is in flames. (p. 265)

But in this case, I hope that the fire brigade arrives late. It is time to rethink socialization science critically. The best policy recommendation is not for us to throw out all deterministic notions, or to throw up our hands; it is to try to understand how things really work and what levers for change may exist in them. Theories that do not seek components, that do not simplify and seek abstract principles, and that do not look for determinants and causes are unlikely to leave socialization science more advanced than it is today. When Lewontin et al. (1984) start to propose scientific directions rather than criticize, their prose loses its power and immediacy, and confuses individual differences with universal developmental processes:

> . . . we would insist on the unitary ontological nature of a material world in which it is impossible to partition out the "causes" of the twitching muscle of the frog into x percent social (or holistic) and y percent biological (or reductionist). The biological and the social are neither separable, nor antithetical, nor alternatives, but complementary. All causes of the behavior of organisms, in the temporal sense to which we should restrict the term *cause*, are simultaneously both social and biological, as they are all amenable to analysis at many levels. (p. 282)

But there is much to discover in our genes, as there is much to discover in better models of cultural transmission. Let us hope that in the next generation social scientists keep after the causes of behavior, whatever they may be, and are ready to let discovery guide policy, whatever it may be. The lessons of the monkey Imo and her cousins should not be lost on socialization science.

Notes

[1]Evolutionary models typically work first with selection at a single locus, where gene A is a rival to gene a. This approach is convenient because it simplifies both mathematical and conceptual treatments of evolution. It is assumed that the selective pressure at the hypothesized loci would apply to other loci; hence many genes would finally produce either the "learn from parents only" or "learn from any source" trait, as distributed in a real population.

[2]A difference equation represents the state of a system at later age in terms of its state at the prior age. For more quantitatively minded readers, the equations used in this analysis are given below.

Children of smoking parents:

(1) $P'_{NS_{a+1}} = P'_{NS_a} - (T_1 \times M \times P'_{NS_a})$

(2) $P'_{TR_{a+1}} = P'_{TR_a} + (T_1 \times M \times P'_{NS_a}) - j_1 P'_{TR_a}$

(3) $P'_{RS_{a+1}} = P'_{RS_a} + j_1 P'_{TR_a} - uP'_{RS_a} + vP'_{FS_a}$

(4) $P'_{FS_{a+1}} = .54 - P'_{NS_{a+1}} - P'_{TR_{a+1}} - P'_{RS_{a+1}}$

Children of nonsmoking parents

(5) $P_{NS_{a+1}} = P_{NS_a} - (T_2 \times M \times P_{NS_a})$

(6) $P_{TR_{a+1}} = P_{TR_a} + (T_2 \times M \times P_{NS_a}) - j_2 P_{TR_a}$

(7) $P_{RS_{a+1}} = P_{RS_a} + j_2 P_{TR_a} - uP_{RS_a} + vP_{FS_a}$

(8) $P_{FS_{a+1}} = .46 - P_{NS_{a+1}} - P_{TR_{a+1}} - P_{RS_{a+1}}$

where $M = P'_{TR_a} + P_{TR_a} + P'_{RS_a} + P_{RS_a}$

u = the quitting rate

v = the relapse rate.

[3]A *within-family* correlation of body size and temperament in the Masai would be stronger evidence for the hypothesis. A within-family correlation would show that a child with the more difficult temperament was also heavier than his or her sibling.

[4]The basic computer program has these lines of code:

```
 10 dim p(1000)
 20 'created May 14, 1992'
 30 'hawk.bas'
 40 'basic program for evolution of Hawks and Doves'
 50 input "starting proportion of hawks";p(i)
 60 for i = 1 to 1000
 70 t = i + 1
 80 b = -.3*p(i) + .6*(1 - p(i)) + 2.0
 90 c =.2*(1 - p(i)) + 2.0
100 rb = b/(b + c)
110 rc = c/(b + c)
120 'recursive equation based on differential birth rates'
130 p(t) = p(i) + rb*p(i) - rc*p(i)
140 ct = ct + 1
145 if t > 20 goto 210
150 if t <= 5 then goto 160 else goto 165
160 if ct = 4 then goto 170 else goto 200
165 if ct = 5 goto 170 else goto 200
170 print "generation = ", t, "Hawks = ", p(t)
180 ct = 0
200 next
210 print "stop"
```

[5]Genes predisposing toward homosexual orientation may be an exception (Bailey & Pillard, 1991).

[6]In a sample of black South Africans, where over 20% of the children (68 of 300) had been hospitalized for kwashiorkor, strong correlations existed between parental education and family crowding on the one hand, and both physical and cognitive outcomes in 5- and 6-year-old children on the other. For instance, parental education correlated .48 with height, .41 with head circumference, and .51 with vocabulary (Goduka, Poole, & Aotaki-Phenice, 1992).

References

Bailey, J. M., & Pillard, R. C. (1991). A genetic study of male sexual orientation. *Archives of General Psychiatry, 48,* 1089–1096.

Boyd, R., & Richerson, P. J. (1985). *Culture and the evolutionary process.* Chicago: University of Chicago Press.

Carey, G. (1991). Evolution and path models in human behavioral genetics. *Behavior Genetics, 21,* 433–444.

Carey, G. (1992). Simulated twin data on substance use. *Behavior Genetics, 22,* 193–196.

Carmelli, D., Swan, G. E., Robinette, D., & Fabsitz, R. R. (1990). Heritability of substance use in the NAS-NRC twin registry. *Acta Geneticae Medicae et Gemellologiae, 39,* 91–98.

Cavalli-Sforza, L. L., & Feldman, M. W. (1981). *Cultural transmission and evolution: A quantitative approach.* Princeton, NJ: Princeton University Press.

Cohen, L. E., & Machalek, R. (1988). A general theory of expropriative crime: An evolutionary ecological approach. *American Journal of Sociology, 94,* 465–501.

Dawkins, R. (1989). *The selfish gene* (2nd ed.). Oxford: Oxford University Press.

deVries, M. W. (1984). Temperament and infant mortality among the Masai of East Africa. *American Journal of Psychiatry, 141,* 1189–1194.

Diamond, J. (1990). A pox upon our genes. *Natural History, 2,* 26–30.

Diamond, J. (1992). *The third chimpanzee: The evolution and future of the human animal.* New York: HarperCollins.

Draper, P. (1989). African marriage systems: Perspectives from evolutionary ecology. *Ethology and Sociobiology, 10,* 145–169.

Durham, W. H. (1991). *Coevolution: Genes, culture, and human diversity.* Stanford, CA: Stanford University Press.

Eysenck, H. J. (1980). *The causes and effects of smoking.* London: M. T. Smith.

Ferrence, R. G. (1989). *Deadly fashion: The rise and fall of cigarette smoking in North America.* New York: Garland.

Firebaugh, G. (1992). Where does social change come from? Estimating the relative contributions of individual change and population turnover. *Population Research and Policy Review, 11,* 1–20.

Goduka, I. N., Poole, D. A., & Aotaki-Phenice, L. (1992). A comparative study of black South African children from three different contexts. *Child Development, 63,* 509–525.

Halseyk, W. D., & Johnson, B. (1990). *Collier's encyclopedia* (vol. 20). New York: Macmillan.

Kummer, H. (1971). *Primate societies: Group techniques of ecological adaptation.* Chicago: Aldine.

Lewis, A. M. (1992). APA, mental health community react to Goodwin's resignation. *Psychological Science Agenda, 5,* 1.

Lewontin, R. C., Rose, S., & Kamin, L. J. (1984). *Not in our genes: Biology, ideology, and human nature.* New York: Pantheon.

Loeber, R. (1991). Antisocial behavior: More enduring than changeable? *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 393–397.

Lumsden, C. J., & Wilson, E. O. (1983). *Promethean fire: Reflections on the origin of mind.* Cambridge, MA: Harvard University Press.

Maynard-Smith, J. (1982). *Evolution and the theory of games.* Cambridge, England: Cambridge University Press.

Perry, D. G., & Bussey, K. (1979). The social learning theory of sex differences: Imitation is alive and well. *Journal of Personality and Social Psychology, 37,* 1699–1712.

Robins, L. N. (1966). *Deviant children grown up: A sociological and psychiatric study of sociopathic personality.* Baltimore: Williams & Wilkins.

Rowe, D. C., Chassin, L., Presson, C. C., Edwards, D., & Sherman, S. J. (1992). An "epidemic" model of adolescent cigarette smoking. *Journal of Applied Social Psychology, 22,* 261–285.

Rowe, D. C., & Osgood, D. W. (1984). Sociological theories of delinquency and heredity: A reconsideration. *American Sociological Review, 49,* 526–540.

Rowe, D. C., & Rodgers, J. L. (1991a). An "epidemic" model of adolescent sexual intercourse: Applications to national survey data. *Journal of Biosocial Science, 23,* 211–219.

Rowe, D. C., & Rodgers, J. L. (1991b). Adolescent smoking and drinking: Are they "epidemics"? *Journal of Studies on Alcohol, 52,* 110–117.

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Developmental Psychology, 63,* 1–19.

Swan, G. E., Carmelli, D., Rosenman, R. H., Fabsitz, R. H., & Christian, J. C. (1990). Smoking and alcohol consumption in adult male twins: Genetic heritability and shared environmental influences. *Journal of Substance Abuse, 2,* 39–50.

Tooby, J. (1982). Pathogens, polymorphism, and the evolution of sex. *Journal of Theoretical Biology, 97,* 557–576.

Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality, 58,* 17–67.

Wilson, E. O. (1975). *Sociobiology: The new synthesis.* Cambridge, MA: Belknap Press.

INDEX