15

Rough-and-Tumble Play

The Brain Sources of Joy

When children play, they exercise their senses, their intellect, their emotions, their imagination—keenly and energetically. . . . To play is to explore, to discover and to experiment. Playing helps children develop ideas and gain experience. It gives them a wealth of knowledge and information about the world in which they live—and about themselves. So to play is also to learn. Play is fun for children. But it's much more than that—it's good for them, and it's necessary. . . . Play gives children the opportunity to develop and use the many talents they were born with.

Instruction sheet in Lego® toys (1985)

CENTRAL THEME

When children are asked what they like to do more than anything else, the most common answer is "to play!" It brings them great joy. And roughhousing play is the most fun of all, even though most investigators recognize other types such as "object play" and "fantasy play." Although thousands of papers have been written on the topic, play is still considered a frivolous area of inquiry among most neuroscientists. Only recently have some become interested in the underlying brain issues. Now increasing numbers of investigators are beginning to realize that an understanding of play may reveal some major secrets of the brain and yield important insights into certain childhood psychiatric problems such as autism and attention deficit disorders (or hyperkinesis, as it used to be known). It is now certain that the brain does contain distinct neural systems devoted to the generation of roughhousing or rough-and-tumble (RAT) play. Indeed, one of the best species for systematic study of this behavior is the laboratory rat, and practically all the work summarized here is based on such play in rats. Although our knowledge about the underlying PLAY systems remains rudimentary, RAT play appears to be intimately linked to somatosensory information processing within the midbrain, thalamus, and cortex. Certain synaptic chemistries are especially effective in arousing play (e.g., acetylcholine, glutamate, and opioids), while others reduce playful impulses (e.g., serotonin, norepinephrine, and GABA), but neuropharmacological studies tell us little about the adaptive function(s) of play. There is an abundant theoretical literature regarding these functions, comparable to that found in dream research, but relevant data are decidedly scarce. The description from a leaflet in a box of Lego® toys says it all. Now it is necessary to judge the various possibilities with rigorously conducted experiments. Fortunately, roughhousing PLAY systems appear to be conserved in the brains of many mammalian species, and we should be able to obtain a credible answer to the functional questions, even for humans, by carefully analyzing animal models. We anticipate that play will be found to have many beneficial effects for both brain and body, including the facilitation of certain kinds of learning and various physical skills. Most important, play may allow young animals to be effectively assimilated into the structures of their society. This requires knowing who they can bully, and who can bully them. One must also identify individuals with whom one can develop cooperative relationships, and those whom one should avoid. Play probably allows animals to develop effective courting skills and parenting skills, as well as increasing their effectiveness in various aspects of aggression, including knowledge about how to accept defeat gracefully. It seems that most of the basic emotional systems may be recruited at one time or another during the course of play, and in higher organisms, play may encourage organisms to test the perimeters of their knowledge. In short, the brain's PLAY networks may help stitch individuals into the social fabric that is the staging ground for their lives. Is it any wonder, then, that play is such fun—perhaps one of the major brain

sources of joy? It is sad that play research has not been of greater interest for neuroscientists, but perhaps that is because they are having great fun working on the minutest details of the most trifling problems (or so it may seem to outsiders). However, it is often there, among the fine details of nature, that scientists find startling things that can move heaven and earth. This is what Einstein did when he imagined what it would be like to ride a beam of light, and he remained mentally young and playful throughout his life. Perhaps the modern search for the mythological "fountain of youth" should focus as much on the neurobiological nature of mental youthfulness and play as on ways to extend longevity.

Conceptual Background for the Neural Sources of Ludic Urges

A great deal of joy arises from the arousal of play circuits within the brain. Although this is a reasonable assertion, it can only be a supposition until the identity of play circuits has been more completely revealed by brain research. That play is a primary emotional function of the mammalian brain was not recognized until recently, but now the existence of such brain systems is a certainty. For instance, juvenile rats will exhibit roughhousing or RAT ludic behaviors (from *ludare*, meaning "to play") even if they have been prevented from having any prior play experiences during earlier phases of development. Just as most young birds fly when the time is ripe, so do young mammals play when they have come of age. Young rats start to play around 17 days of age, and if denied social interaction throughout the early phases of psychosocial development (e.g., from 15 to 25 days of age), they play vigorously as soon as they are given their very first opportunity.[1]

Thus, the impulse for RAT play is created not from past experiences but from the spontaneous neural urges within the brain. Of course, a great deal of learning probably occurs during the course of roughhousing play, but this is ultimately the result of spontaneously active PLAY impulses within specific circuits of the brain, some of them in ancient parts of the thalamus, which coax young organisms to interact in ludic ways on the field of competition. It may well be that various neuronal growth factors are recruited during play (see Chapter 6), but evidence at such molecular levels of analysis remains nonexistent.

Although we presently have little detailed knowledge about the underlying brain mechanisms of play, rigorous psychobiological experiments are finally being conducted. We now have the empirical and conceptual tools to identify the primal circuits that lead animals to play. This work may eventually yield a neural understanding of what it means for humans to experience joy, or at least one of the most intense forms of joy. This work will also eventually reveal the true adaptive nature of play, but for the time being our ignorance remains vast, especially since it is hidden by an abundance of compelling theories propounded liberally by psychologists and others, without sufficient evidence.

Although play reflects genetically ingrained ludic impulses of the nervous system, it requires the right environment for full expression. For instance, fear and hunger can temporarily eliminate play.[2] In most mammals, play emerges initially within the warm and supportive secure base of the home environment, where parental involvement is abundant. Jane Goodall described the sequence of events as play first unfolds in chimpanzees: "A chimpanzee infant has his first experience of social play from his mother as, very gently, she tickles him with her fingers or with little nibbling, nuzzling movements of her jaws. Initially these bouts are brief, but by the time the infant is six months old and begins to respond to her with play face and laughing, the bouts become longer. Mother-offspring play is common throughout infancy, and some females frequently play with juveniles, adolescents, or even adult offspring."[3] The role of the mother in guiding the play and initial social interactions of young children is evident in humans, and such trends are evident even in rats.[4] In many species, fathers seem less playful and less socially tolerant than mothers, but humans *may* be an exception, perhaps partially because of cognitive mediation. In any event, it is now clear that the most vigorous play occurs in the context of preexisting social bonds. As discussed in the previous chapter, it is not unusual in nature for social bonds to be stronger between infants and their mothers than their fathers, who all too commonly exhibit little enthusiasm for nurturance.

Thus, contrary to conventional wisdom, it may be that females of most species remain more playful than males (at least in friendly, nonharmful ways) as they approach adulthood. As we will see, the prevailing notion that males intrinsically have stronger play tendencies[5] is certainly not justified for rats, and we should doubt it for other species until *well-controlled* studies have been conducted. The larger size and stronger competitive/aggressive urges of males may make their play rougher, so that social reinforcement of victory makes them appear more playful during the later stages of juvenile life. However, this difference may reflect the drive to attain dominance (which may, of course, become integrally associated with PLAY circuits), rather than elevated neural impulses for vigorous and joyful social interaction.

The stronger urge for social dominance in males (which is only one component of RAT play) may have incorrectly led to the widespread supposition that roughhousing play impulses are more intense in males than in females. For instance, in humans, the apparent heightened male enthusiasm for rough sports may be due as much to their biologically and socially based "power needs" as to any intrinsic differences in the arousability

of their basic PLAY circuits. This is affirmed by the fact that the recent liberalization of sports policies in America has led to a stupendous growth in female participation in competitive sports. In any event, the extent to which human enjoyment of sports emerges from activities of primal PLAY circuits will be an important (but yet unresolved) question for us to consider.

Overview of the Experimental Analysis and Sources of Play

In most primates, prior social isolation has a devastating effect on the urge to play. After several days of isolation, young monkeys and chimps become despondent and are likely to exhibit relatively little play when reunited.[6] Apparently, their basic needs for social warmth, support, and affiliation must be fulfilled first; only when confidence has been restored does carefree playfulness return.[7] Laboratory rats, on the other hand, deviate markedly from this general pattern and thereby provide a useful model for the systematic analysis of play mechanisms within the brain. Laboratory rats show a greater emotional equanimity in coping with social isolation as compared to many other mammals (see Figure 2.1). Also, as emphasized in the previous chapter, the social-bonding mechanisms in laboratory rodents are comparatively weak. Perhaps for this reason, isolation housing does not readily produce obvious depressive responses in laboratory rats and mice.[8] Thus we can take advantage of social-deprivation variables to control levels of playfulness. Prior social isolation systematically increases roughhousing play in juvenile rats, while social satiation systematically reduces it (Figure 15.1).[9]

The facilitation of play in rats by prior isolation is due not simply to social deprivation itself but to the specific effects of play deprivation. If one houses animals together in the tight confinement of a "jungle gym" type of living environment where they cannot readily roughhouse, they show abundant play in an open play arena. Likewise, if one houses juvenile rats with adult animals that are not very playful, they will play with other juveniles as intensely as if they had just emerged from total isolation.[10] In any event, with the use of prior social deprivation, RAT play can be analyzed efficiently in the laboratory. The systematic nature of the results again affirms that the urge to play is an intrinsic function of the mammalian nervous system.[11]

Although there is substantial diversity in the specific play patterns exhibited by different mammalian species, the evolutionary roots probably go back to an ancient PLAY circuitry shared by all mammals in essentially homologous fashion. It is also possible that creatures other than mammals (especially birds) exhibit social play, but avian play is less predictable and hence harder to study.[12] Accordingly, the present discussion will be restricted to mammals, although the evolutionary roots may well go back to an era predating the divergence of mammalian and avian lines more than a hundred million years ago (see Appendix A). Once we understand the

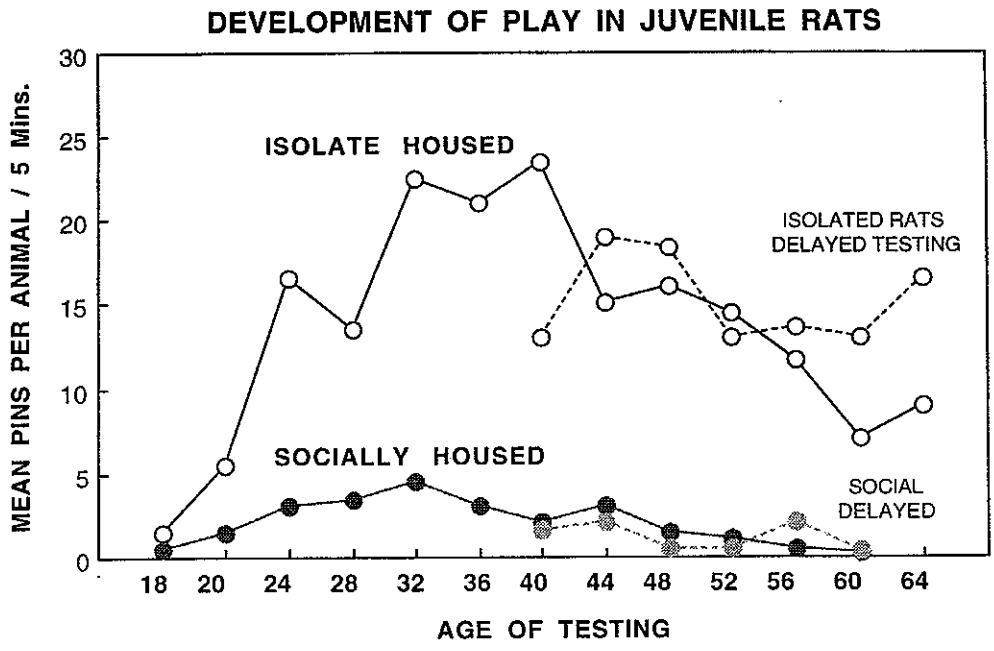

Figure 15.1. Ontogeny of play in socially isolated and socially housed laboratory rats. (Adapted from Panksepp, 1981; see n. 18). The pinning measure is depicted in Figure 15.2.

neural circuitry of mammalian play, it should be easier to determine whether birds have homologous brain mechanisms, or whether their seemingly playful behaviors emerge from different types of neural systems.

Before discussing the most basic form of play—namely, roughhousing play—it should again be emphasized that several distinct forms are widely recognized in human research. Human play has been divided by social and developmental psychologists into exploratory/sensorimotor play, relational/functional play, constructive play, dramatic/symbolic play, and games-with-rules play, as well as RAT play, of course.[13] This last form, rough-and-tumble play, is presently easiest to study in animal models, but except for a few outstanding pieces of work, it has received the least attention in human research.[14] This is understandable, for roughhousing is boisterous and often viewed as disruptive and potentially dangerous by adults. Of course, kids love it (it brings them joy), and animals readily learn instrumental responses to indulge in it.[15] This is the main form of play that other mammals exhibit, and it remains possible that the relatively solitary motor play of many herbivorous animals, such as running, jumping, prancing, and rolling, emerges from the same basic PLAY urges that control roughhousing play between young animals. Unfortunately, there is no neurological evidence yet that allows definitive conclusions.

Although human play has been extensively taxonomized, it is still worth contemplating to what extent the various forms are merely higher elaborations (culturally derived, as well as higher neuroevolutionary variants) on a single primal theme: Are there multiple executive circuits for play in the human brain, or do they all reflect manifestations of a single underlying PLAY system of the mammalian brain? Until demonstrated otherwise, we should be parsimonious and subscribe to the single command-circuit alternative.

Just as each basic mammalian emotion can be expressed in many ways in human cultures—including dance, drama, music, and other arts—arousal of a single basic ludic circuit could add "fun" to the diversity of playful activities. In other words, PLAY impulses that are processed through the higher cognitive networks of the human cortex (i.e., via social constructions) may result in many seemingly distinct forms of human play. The common denominator for all, however, may arise from basic neuronal systems that were originally designed to generate RAT ludicity. Indeed, it is a testable proposition: Once we unravel the details of RAT PLAY circuits, their role in other forms of play can be evaluated. Accordingly, let us briefly entertain ways in which play diversity in humans may emerge from the "simplicity" of a single system.

Perhaps, in humans, the source energy for roughhousing play can be channeled voluntarily into a large variety of distinct activities. At times humans simulate playfulness and thereby attempt to evoke ludic feelings indirectly through pretenses. For instance, children like to stage various skits and shows, but as they attempt to perform seriously, all too often they simply end up giggling in glee. Perhaps the culturally sanctioned playful expressions, such as dancing, remain emotionally hollow until the ancient circuits of playfulness—affectively characterized by "lightness," "joy," and "flow"—are recruited?

Through their attempts to voluntarily activate the natural ludic mechanisms of the brain, humans may achieve totally new forms of playfulness (including various games, toys, and dramatic and linguistic devices). In this context, it should be remembered that each basic emotional system can energize a number of distinct behavioral options, and perhaps PLAY systems help generate a diversity of emotional behaviors upon which learning can operate. It must also be noted that playfulness in humans can eventually be expressed in symbolic ways, which may be largely linguistic, such as puns, joking, and verbal jibes, that lead to a great deal of mirth and laughter.

A discussion of the functions of play will be reserved for a later section, but here I will anticipate the main conclusion. PLAY circuitry allows other emotional operating systems, especially social ones, to be exercised in the relative safety of one's home environment. Play may help animals project their behavioral potentials joyously to the very perimeter of their knowledge and social realities, to a point where true emotional states begin to intervene. Thus, in the midst of play, an animal may gradually reach a point where true anger, fear, separation distress, or sexuality is aroused. When the animal encounters one of these emotional states, the playful mood may subside, as the organism begins to process its predicaments and options in more realistic and unidimensional emotional terms. In human children this may often consist of running to mother in tears, with complaints about the injustices they have encountered to see what type of social support and understanding (i.e., kin investment) they might be able to muster.

Finally, as will be discussed more fully later in this chapter, play and exploratory systems (i.e., of the type discussed in Chapter 8) appear to be distinct in the brain. Although these concepts are often combined in human research,[16] as if they reflected synergistic processes, they appear to be independent and at times mutually exclusive. For instance, psychostimulants such as amphetamines, which invigorate exploratory activities, markedly reduce play behaviors.[17] Indeed, when placed in new environments, animals typically exhibit strong exploratory activity with little tendency to play until they have familiarized themselves with the new surroundings.

In sum, we now have highly effective laboratory procedures to analyze the neural substrates of RAT play. A straightforward experimental approach will surely yield more important insights into the nature of this phenomenon than any armchair theorizing of the type

highlighted in the description of children's play at the beginning of this chapter.

A Description of Rough-and-Tumble Play

It is difficult to capture the dynamic image of real-life play in words. But the overall impression given by practically all mammals is a flurry of dynamic, carefree rambunctiousness. In rats, one sees rapid spurts of activity, toward and away from a play partner. Sometimes one animal "bowls" the other animal over, which leads to a flurry of playful chasing. In turns, the animals pursue each other, with rapid pivoting and role reversals. Animals often pounce on each other's backs as if they are soliciting vigorous interaction; these "dorsal contacts" can be easily quantified and have been commonly used as an explicit measure of play solicitations (Figure 15.2). Sometimes the dorsal contacts do not yield reciprocation, instead ending up as prolonged bouts of dorsal grooming. At other times, the recipient of play solicitations responds by either running away or twisting laterally; an apparent bout of wrestling ensues, in which one animal winds up on its back with the other animal on top. This "pinning" posture can also be easily quantified (Figure 15.2) and is the clearest measure of the consummatory aspects of play. If animals are allowed to play on an activity platform, one can also obtain an overall measure of RAT activity. There are surely many other ways to monitor play, and each of these measures can be subcategorized. For instance, most pins are of short duration, occurring in the midst of ongoing "wrestling" matches, while others are more prolonged, often signaling the end of a play bout. Dorsal contacts can be strong or sustained, or made passingly as one animal bounds leapfrog-style off another The precise details of play episodes vary widely among different mammalian species, but the general flavor remains the same—one of joyful social exchange with a strong competitive edge. It may come as a surprise to some, but young rats given no other ludic outlets love to be tickled by and play with a frisky human hand.

RAT play in most species exhibits a characteristic developmental time course, with the amount of play increasing during the early juvenile period, remaining stable through youth, and diminishing as animals go through puberty (see Figure 2.1).[18] We presently know essentially nothing about the neurobiological factors that control this inverted U-shaped developmental function. Presumably it is related to aspects of brain maturation, as well as neurochemical shifts that occur during development.[19]

Play dominance clearly emerges if two rats are allowed to play together repeatedly.[20] After several play episodes, one rat typically tends to become the "winner," in that it ends up on top more often during pins. On the average, the split is that the winner ends up on top about 70% of the time, while the "loser" achieves less success, but the continuation of play appears to require reciprocity and the stronger partner's willingness to handicap itself. If one animal becomes a "bully" and aspires to end up on top all the time, playful activity gradually diminishes and the less successful animal begins to ignore the winner. There are reasons to believe that similar dynamics are present in human verbal play, which is a common way for folks to get to know each other and to best each other.

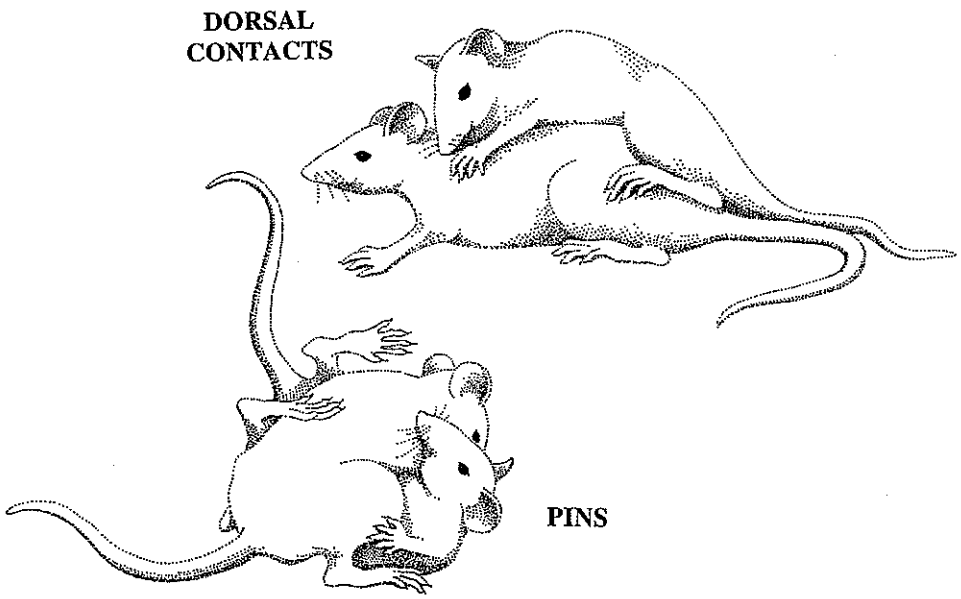

Figure 15.2. Two major play postures that are used to quantify rough-and-tumble play.

As might be expected, body weight is an important factor in dictating which animal of a pair becomes the winner, as is neurochemical activity.[21] With regard to weight, approximately a 10% weight advantage, just like in human boxing and wrestling, is sufficient to give a statistical edge to the heavier combatant (we are here ignoring the more complex issue of physical strength, not to mention psychological "strength," which as we will see is partly opioid-mediated). Neurochemically, if one animal of a play pair is given a small dose of an opiate agonist such as morphine and the other is given a small dose of an opiate antagonist such as naloxone, all other things being equal, the animal receiving morphine always becomes the winner (Figure 15.3). A similar effect is seen in vehicle-treated rats pitted against naloxone-treated ones, but a morphine-treated animal does not invariably win against controls.[22] These effects suggest that brain opioids control social emotionality, so that without brain opioids an animal tends to feel psychologically weaker, causing it to lose because it is more prone to experience negative feelings such as separation distress, as discussed in the previous chapter. To the contrary, control animals as well as morphine-treated ones may prevail because they experience heightened social confidence, a feeling of psychological strength that presumably emerges from the neurochemical correlates of social bonding.

Of course, there are alternative explanations for these effects, as there are for all the findings of science (see Figure 2.3). For instance, opiate receptor antagonists may reduce or eliminate the opioid-mediated reinforcing pleasure of social interaction, while opiate agonists enhance such forms of reinforcement. Also, it is possible that the opiate antagonists make some playful blows more painful, while morphine dulls those play-reducing sensations. Surely, if the scrapes of life become less painful, animals should play more. In any event, the play-dominance effects of opioid manipulations are remarkably clear-cut in animals that begin receiving these agents at the outset of their mutual play experiences (Figure 15.3). The fact that it takes some time for the full manifestation of the effects suggests that social learning promotes the emergence of the dominance asymmetry. However, if social dominance has already transpired prior to the neurochemical manipulations, play-dominance patterns do not shift readily. Indeed, past social learning is a powerful force in all social encounters. On the basis of this simple fact, one must again wonder whether some of the effects that have been widely disseminated in the literature, such as the oft-reported sex differences in play whereby males supposedly exhibit more RAT play than females, merely reflect assertiveness biases that have emerged from prior social learning based on body weight and strength asymmetries between the sexes. Persuasive data are presently not available on this issue.

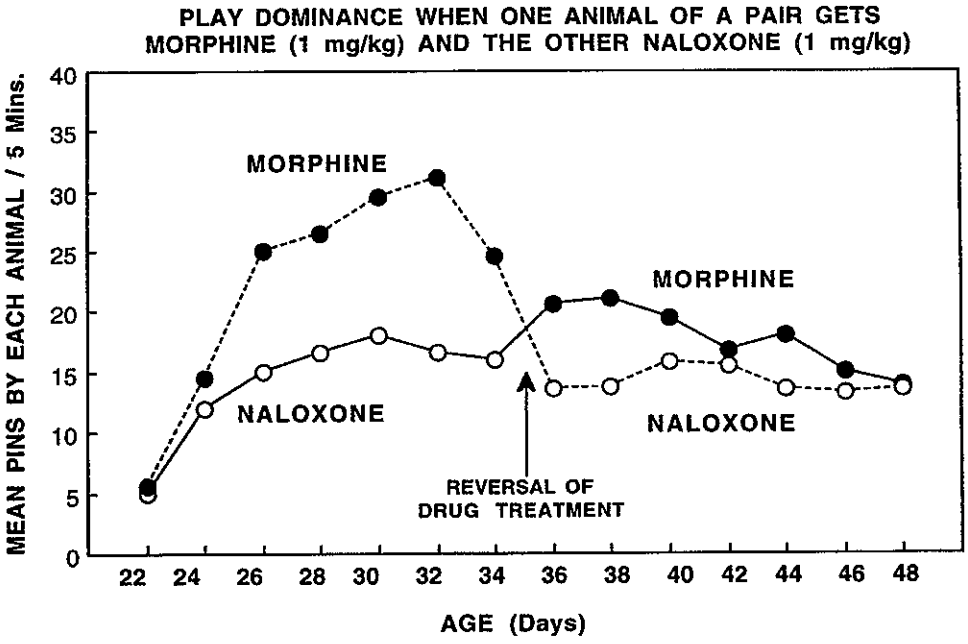

Figure 15.3. Pinning on successive days, with one animal getting naloxone (1 mg/kg) and the other getting morphine at the same dose. After seven days, drug conditions were reversed. (Adapted from Panksepp et al., 1985; see n. 22.)

Play and Aggression

RAT play in animals is often called *play-fighting*, and some believe it is little more than the juvenile expression of certain types of aggressive activity—for instance, intermale aggression. Although RAT play often has the outward behavioral hallmarks of aggressive fighting, a formal behavioral analysis indicates that the behavioral sequences exhibited during real fighting and play are remarkably different.[23] Resemblances between the two are only superficial. For instance, serious aggressive postures are rarely seen in play-fighting. In a real fight, rats often exhibit boxing, consisting of standing on their hind paws and paddling each other with their front paws, as well as laterally directed aggressive postures called "side-prancing," commonly accompanied by piloerection. These postures essentially never occur during social play. Sometimes play does end up in real fighting, but then the signs of behavioral rambunctiousness (frantic hopping, darting, and pouncing) immediately cease.[24] A behavioral tension emerges as RAGE and FEAR systems are presumably activated.

Moreover, true aggression and play follow different rules and are differentially sensitive to a variety of experimental manipulations: (1) In real intermale dominance fights, all other things being equal, the resident animal is invariably the winner if the behavioral test is conducted in the home territory of one of the animals. This is not the case during play-fighting.[25] (2) During play there are no *sustained* defensive postures in which one animal lies on its back while the other sustains a menacing top position for extended periods (i.e., thus, pins during play move along more gracefully and rapidly). (3) Play-fighting is a positive reward for both participants. The winners and the losers of previous play fights readily learn instrumental tasks, such as making an appropriate choice in a T-maze, in order to gain the opportunity to play together again, and they both run toward the opportunity to play with equal speed. The only difference is that the winners barge quickly into the play box, while the so-called losers are a bit more hesitant in making their entry into the play arena.[26] (4) Testosterone, which is quite powerful in promoting aggressive dominance, has relatively little effect except that in some pairs it reduces play-fighting.[27] In animals that exhibit reductions in play following several days of testosterone treatment, it seems that the play bouts quickly become too aggressive. When this occurs, the behavior loses its "carefree" quality, and overall playfulness becomes inhibited. (5) Highly specific antiaggressive drugs such as fluprazine and eltoprazine, which can markedly reduce various forms of fighting (see Chapter 10), do not clearly reduce play and in some instances appear to increase playful interactions.[28]

Although there are bound to be continuities between the skills learned during play-fighting and eventual adult dominance abilities, there is presently no clear evidence for powerful continuities in the executive brain mechanisms of roughhousing play and adult forms of aggression. The two seem to have distinct motivational substrates, although it remains possible that the play circuits of juvenile stages of development may take on a more adultlike luster as animals mature. Similarly, in humans, we see childhood play fading into ritualized dominance sports such as football or basketball.

It is unlikely that professional football or other sports require the participation of PLAY circuits in adult humans, but the quality of performance is probably increased when such circuits are aroused. On the other hand, it is possible that few spectators would consider professional sports to be fun were it not for the existence of PLAY circuits in their brains that are vicariously aroused by observation of play activities in others. Professional wrestling may be especially attractive to certain audiences because its choreography closely resembles the instinctual expressions of RAT play in humans.

Another dimension of sports that deserves attention is the possibility that it is an institutionalized way to dissipate intermale aggressive energies that might otherwise cause chaos in peaceful societies. Keeping "warrior energies" constrained within the guise of playfulness may help reduce the level of violence in peacetime. Indeed institutionalized forms of play, such as professional sports, have become big business around the world. This development casts a new and sometimes dark shadow over the spontaneous expressions of emotionality that should characterize the playing field.

Although there is still a great deal of joy and despair among those participating in professional sports, with spectators being overcome by waves of positive and negative emotions as the fortunes of their teams wax and wane, the new economic dimensions of professional sports have made us realize that in humans, games are simply no longer what evolution meant them to be. Instead of exercising various skills and having a good time, institutionalized play has become the arena for demonstrating one's acquired and aggressive skills. Although I do not dwell on such issues in this book, it is obvious that cultural forces in human societies have the ability to change emotional forces into new entities, both beautiful and horrific.

In sum, it seems evident that PLAY circuits are largely independent of aggression circuits, even though during development they may eventually contribute to the intermale types of aggression that were highlighted in Chapter 10. It is certainly possible that PLAY systems contribute to social dominance urges, which may help explain our love of rough professional sports, where such issues are paramount in the minds of players and spectators alike. Also, since sports provide the opportunity for expressions of symbolic dominance, it is little wonder that they are accorded such high esteem in our society, even by administrators of so many universities.

The Varieties of Play and Laughter, Especially in Humans

Humans are a uniquely playful species. This may be due in part to the fact that we are neotenous creatures who benefit from a much longer childhood than other species. For instance, our childhood and adolescence constitute about 20% of our life span, which is comparable to other great apes. However, the corresponding proportion for other primates is generally less than 10%; in dogs, cats, and rats, it typically approaches 5%. Another feature that adds to the complexity of our playfulness is the simple fact that our play instincts are modified so markedly by our cognitively focused higher brain areas. Although cortical processes surely add a great deal of diversity to our playful behaviors, especially as we develop, it is unlikely that the primal brain "energy" for playfulness emerges from those higher brain functions. These energies probably emerge from the same ancient executive systems that govern RAT play in other species. As those primitive playful impulses percolate through the brain, they assume new forms ranging from slapstick humor to cognitive mirth. Indeed, the hallmark of PLAY circuitry in action for humans is laughter, a projectile respiratory movement with no apparent function, except perhaps to signal to others one's social mood and sense of carefree camaraderie.[29] Some believe laughter is uniquely human, but we would doubt this proposition.

Ethologists have long distinguished two general types of happy or friendly faces: the social smile and laughter. The smile, with its prominent baring of teeth, probably harks back to ancient mammalian threat displays.[30] For instance, many creatures exhibit bare-toothed hissing in response to potential threats. In a social context, this may communicate that one possesses quite a dangerous set of teeth and is potentially willing to use them. No doubt, the probable evolutionary adaptation behind the display is that the potentially tense situation will require no further action if one smiles. The human smile may have evolved from such preexisting old parts to communicate that one is basically friendly but quite capable of dealing with any difficulties that may arise. Laughter, on the other hand, seems to have emerged largely from a different brain system; as some have cogently argued, it may emerge from PLAY motivation.

In children, laughter occurs most commonly in playful situations. Indeed, an openmouthed display characterizes the most intense forms of human laughter, and similar gestures are used as signals for play readiness in other species such as chimpanzees and dogs. Also, the rhythm of laughter has an outward resemblance to the rhythmic kicking and thrashing commonly seen in the roughhousing play of many mammals. Although we commonly associate the presence of laughter with the punch line of a joke, making it functionally similar to the pin position that is the terminal component of a RAT play episode, laughter certainly does not require much cognitive complexity. Physical tickling is one of the easiest ways to provoke laugher in young children; indeed, this response can be induced in infants within the first half year of life, even though it appears to be preceded by a period in which there is a strong tendency to smile in response to social interaction, starting at about 4 months of age.[31] A cyclical pattern, resembling laughter, with respiratory panting and grunting vocalizations can also be induced in chimpanzees and gorillas by tickling, and we have recently discovered a seemingly homologous process in young rats.

To evaluate whether rats laugh, we took a simple tickling approach. Listening in to the ultrasonic frequency range at which rats communicate, we rapidly found that friendly tickling induced very high-frequency chirping at about 50 KHz. This response could be provoked more effectively when the tickling occurred at the nape of the neck, where animals normally solicit play, than on the rump, and full body tickling was most effective of all (Figure 15.4). Animals that had been deprived of social contact from weaning at 24 days of age through puberty exhibited more chirping to tickling than littermates that had been allowed two play sessions each day through this interval. Also, the amount of playfulness in these animals correlated highly with the amount of tickling-induced "laughter," but while play declined with age, tickling effects did not (Figure 15.4). Tickling was a positive incentive for our animals: They would seek out this kind of stimulation and would rapidly begin to chirp to cues associated with tickling. If this vocalization pattern is truly homologous to basic human laughter, we may come to understand human joy by studying the circuits that generate such vocalization in rats.[32]

Apparently, laughter is not learned by imitation, since blind and deaf children laugh readily.[33] The ability to laugh precedes one's ability to comprehend the point of a joke; a great deal of children's laughter typically occurs in free play situations rather than in response to verbal jests. It is reasonable to suppose that the sources of human laughter go back to ancient social engagement systems that first mediated mammalian playfulness.

Laughter may now be one signal for victory within playful social encounters as the philosopher Thomas Hobbes argues,[34] just as being in the top position during pinning in RAT play is the preferred physical position (Figures 15.2 and 15.3). Indeed, this is the dark side of laughter, for it often occurs in response to seeing others hurt, humiliated, or embarrassed, and it indicates a recognition of the victim's slapstick predicament coupled with the feeling that one has been psychologically luckier and perhaps even smarter than the poor sod who is the brunt of some misfortune. In competitive playful encounters in humans, laughter is invariably exhibited more by victors than by losers. Likewise, the perpetrator of a practical joke is much more likely to

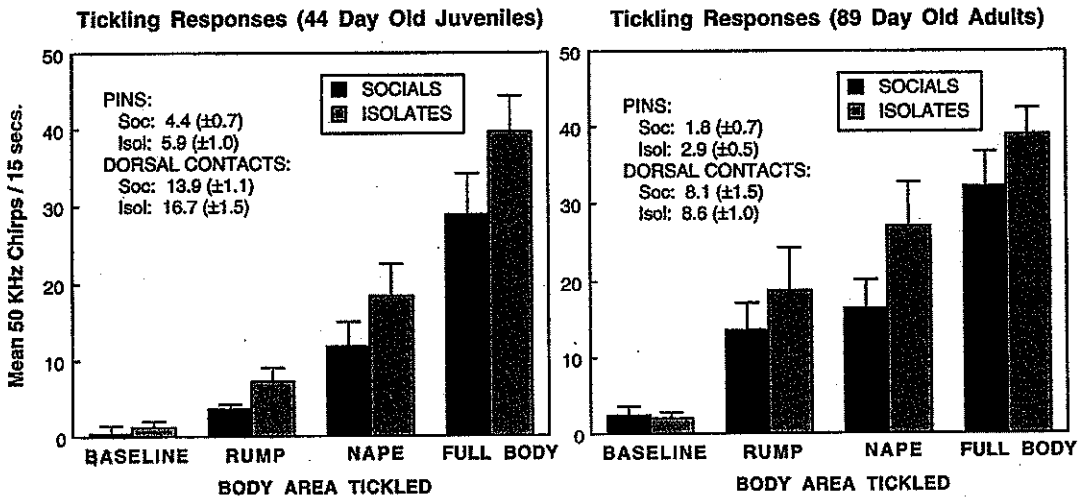

Figure 15.4. Mean (±SEM) levels of chirping in 44- and 89-day-old rats as a function of social history and the area of the body tickled. The animals had been weaned and individually housed at 24 days of age, and half the animals (socials) had received two 0.5-hour play sessions each day through puberty (50 days of age), while the others (isolates) had no opportunities for social interaction. Testing occurred in both groups following 48 hours without social interaction. Pinning and dorsal contacts during a 2-minute play session on an adjacent day are also provided. (Unpublished data, Panksepp & Burgdorf, 1997.)

laugh than the recipient. Groups of humans also often laugh together (it is infectious); this may help cement group solidarity, which is another popular view of the function of laughter.

Neurologically, laughter emerges from primitive subcortical areas of the brain as indicated by the types of brain damage that are correlated with pathological laughter. One disease process that can release impulsive laughter is amylotrophic lateral sclerosis (ALS), a demyelinating disease that affects the brain stem. Another is gelastic epilepsy, which is accompanied by bouts of laughter. Individuals with such a brain disorder can exhibit strong motor components of mirth without any accompanying experience of happiness. Although the victims of such disease processes look happy when they are spontaneously smiling and laughing, they often report no associated positive affect.[35] Most interestingly, such individuals typically exhibit pathological crying during earlier phases of the disease, again without any accompanying feeling of sadness. Not only does the onset of pathological crying typically precede the onset of laughter during the development of ALS, but the crying seems to emerge from lower levels of the neuroaxis than does the laughter.[36]

The apparent neural relationship between these two motor displays suggests that laughter and crying are intimately related in the brain, although the ability to cry appears to have preceded the ability to laugh in brain evolution. In other words, separation-distress mechanisms, and social-bonding ones, may have been essential prerequisites for the evolution of laughter. From this, one might suppose that the evolution of the social bond, and the consequent ability to cry, may also have been social prerequisites for the evolution of play. If so, we might suppose that both play and laughter still serve social-bonding functions, thereby helping to discriminate friends and family from strangers. Indeed, reunion "rituals" in chimpanzees, especially after long separations, are typically characterized by a lot of hooting, howling, and touching.[37] Also, in this context, it should be recalled that the preeminent sensory system, which both provides comfort after separation and most readily provokes play, is touch. Thus, in evolution, the pleasure of touch may have established a neural framework for the emergence of play.

Of course, in humans, play impulses can be manifested in many ways. As individuals mature, a great deal of human play may come to be focused on verbal interchange. The persistent verbal repartee that often characterizes nonserious interactions, such as teasing (e.g., when humans "rib" each other), seems to have the outward characteristics of dorsal contacts and pinning. One tries to arouse the other individual with some provocation, at times even sharp and biting comments; then, if others respond, there is often a desire to "sock it to them" with an especially clever response. If successful, this tends to yield peals of laughter among the young and chuckles among the elderly. This type of repartee may be repeated many times, with each individual trying to best the other—to be cleverest—until it is clear

that one is prevailing or until each is satisfied that he or she is a match for the other. Presumably, when that happens, the individuals have a high potential to establish a special respect and friendship. If one verbally "pins" the other too insistently, the relationship will probably be different, with a much greater sense of dominance and submission asymmetries, which may decisively guide future interactions. Perhaps this is one reason most humans are better talkers than listeners—humans are more likely to feel that they have prevailed if they sustain a high level of output instead of "wasting" time attending to inputs.

Such bonding and social-stratification functions of play and laughter are also especially evident in institutionalized sports. Perhaps for similar reasons, our culture has formalized "roasting" as a special occasion for individuals to exhibit their well-honed playfulness and dominance skills toward people they like. Apparently, the manifestations of PLAY circuitry have permeated human cultures and, perhaps, a great deal of higher brain organization.

Obviously, it would be presumptuous to reduce human playfulness to the operation of a single primitive system that controls RAT play in other animals. Too many layers of neural complexity have been added to the original PLAY instigation systems. In a hierarchically controlled structure like the brain (see Figure 2.2), each level of control has some consequences for the form of the final output. To use an analogy from physics, evidence about the basic emotional systems resembles our general knowledge of atomic structure, which constrains but does not readily allow us to predict the complexity of molecules and man-made materials that can be constructed from those basic structures. However, as we accept the complex reality of playfulness and other emotions in humans and their societies, an adequate analysis of the lower levels that we share with animals is essential for a satisfactory understanding of the complex manifestations that the higher levels permit. Thus, I will assume that the neural mechanisms of RAT play will ultimately prove illuminating for understanding play and joy in humans. It may also help us better understand certain childhood problems such as autism and attention deficit, hyperactivity disorders (ADHDs).[38] In any event, it is remarkable that the existence of this brain system has not yet been generally accepted in either neuroscience or psychology.

The Somatosensory Control of Play

Since RAT play ultimately emerges from powerful endogenous neural activities of the brain that interact with many forms of learning, it is especially difficult to study comprehensively. The motor features of RAT play are so complex that it is hard to imagine how one might trace the source mechanisms in a systematic manner. One approach is to consider that play is a socially contagious process. When playful urges arise in one animal, they seem to "infect" other animals via some type of sensory-perceptual influence. Accordingly, a reasonable question to ask would be: Which sensory systems are most important for social play? Studies that have selectively eliminated individual sensory influences clearly indicate that, at least in rats, neither vision nor olfactory senses (including vibrissae) are necessary for the generation of normal play. One can eliminate any one of these senses without reducing the overall *amount* of RAT play, even though the exact patterning of play has not yet been carefully analyzed in such animals. The auditory system contributes positively to play to some extent, since deafened animals play slightly less, and rats do emit many 50-KHz laughter-type chirps both during play and in anticipation of play.[39] However, the premier sensory system that helps instigate and sustain normal play is touch.

Indeed, certain parts of the body surface are more sensitive to play-instigation signals than others. This has been established by anesthetizing various zones of the body (Figure 15.5). Local anesthetization of the neck and shoulder area is highly effective in reducing the level of playful pinning in young rats, even though the motivation for play, as measured by dorsal contacts, is not reduced.[40] A substantially smaller effect on pinning is obtained if the anesthetic is applied to the rump, and no effect is evident if it is applied to either rostral or caudal areas of the animal's ventral surface, or when it is injected systemically. These findings correspond nicely to the tickling results summarized in Figure 15.4. This suggests that rats have specialized skin zones that send play signals into the nervous system when they are touched. In other words, mammals appear to have "play skin," or "tickle skin," with specialized receptors sending information to specific parts of the brain that communicate playful intentions between animals. Obviously, humans also have tickle skin. It is situated at the back of the neck and around the rib cage, where it is easiest to tickle young children and get them into a playful mood.

In rats the homologous play skin of the body seems to be on the rostral dorsal surface of the body, where most play solicitations (i.e., dorsal contacts) are directed. This is not the only target of play solicitation in rats, but PLAY circuitry of the brain does appear to receive especially potent somatosensory inputs from certain body zones. This helps answer the question that has perplexed so many children: Why can't I tickle myself? Apparently, the system is tuned to the perception of social stimulation partially by being sensitive to unpredictability. The underlying neural systems are designed so that one cannot easily be his or her own social partner or play companion. Tickling requires other selves to arouse playfulness. Thus, the ability to identify and perceive play partners is not a mere sensory phenomenon but a powerful, ingrained central

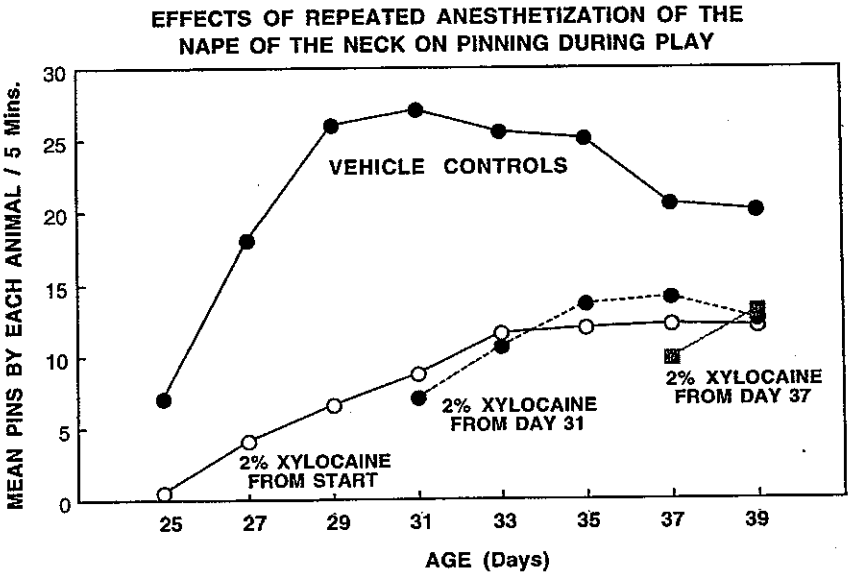

Figure 15.5. Play as a function of age in animals treated at the nape of the neck with xylocaine after 25, 31, and 37 days of life. The reduction of play exhibited no clear tolerance, suggesting that without appropriate somatosensory input, the consummatory aspects of play are seriously compromised. (Adapted from Siviy & Panksepp, 1987; see n. 40.)

nervous system concept (one that may have gone awry in autism).

Apparently, the broadly ramifying PLAY system of the brain can instigate rapid forms of learning. For instance, with some experience and the right ludic attitude, one can "tickle" a young child simply by wiggling a finger in midair or by intoning a "coochi-coochi-coo." Young rats also exhibit rapid conditioning to the cues associated with tickling. Presumably, this is because such rapidly learned play signals can generate the internal interpretation that one has a playful companion. Indeed, if a child is already in a playful mood, it is sometimes sufficient for them to simply look at another person to trigger laughter and playfulness. Indeed, children get into patterns of uncontrollable laughter rather easily, especially when sharing special mental games during culturally pretentious events—formal dinner tables and classrooms, where the abiding adult expectation is that ludic impulses should be controlled. In such circumstances, children's mutual "knowing" glances can generate great hilarity, often in an inverse relation to the level of self control that adults are expecting from them. This tendency indicates that, in humans, the visual system rapidly learns the patterns of behavior that are especially playful. Whether the visual system can generate playfulness without any prior participation of touch during earlier phases of development is unknown. At least in rats, vision is not essential; blind animals play with undiminished vigor.[41]

In sum, the existence of PLAY circuits in the brain probably explains the phenomenon of tickling and highlights the fact that the analysis of somatosensory stimulation of play skin may be a key to understanding the neural processes of PLAY systems. Parenthetically, it should be noted that the apparent expansion of play skin on the body surface when one is in a playful mood highlights a key property of an emotional system—namely, its ability to modify sensory and perceptual sensitivities that are relevant for the emotional behavior being exhibited.

It should be emphasized that anesthetization of the body surface (Figure 15.4) only reduces the animal's ability to perceive proximal play signals, which leads just to a reduction in pinning. It does not reduce the apparent desire to play, since the reduced pinning is not accompanied by a decrease in the emission of dorsal contacts, although it apparently results from diminished appreciation of such contacts. In other words, the anesthetized animal still exhibits normal play-solicitation tendencies. The basic desire to play is not dependent on sensory inputs. It is an endogenous urge of the brain.

The Neuroanatomy of Play

Analysis of the somatosensory projection systems of the brain yields a coherent way to address the neuroanatomy of play systems. Since anesthetization of the dorsal body

surface can reduce pinning, it is not surprising that similar effects can be obtained by lesioning the ascending somatosensory projection circuits from the spinal cord such as the spinothalamic tract.[42] However, this is also not a result of diminished play motivation, since such animals exhibit a normal desire to play. Only when somatosensory information enters the thalamic projection areas do we begin to get more specific motivational effects.[43] At that level, somatosensory information diverges into the *specific thalamic projection areas* of the ventrobasal nuclei that project discriminative information up to the parietal cortex and into *nonspecific reticular nuclei,* such as the parafascicular complex and posterior thalamic nuclei, that seem to elaborate a ludic motivational state within the animal. In other words, bilateral damage of the nonspecific reticular nuclei yields what appear to be specific play effects. Following such damage, pinning and dorsal contacts are both reduced, and the lesioned animals are no longer motivated to play. This effect is specific, since other relatively complex motivated behaviors, such as food seeking (foraging), are not diminished. This suggests that the parafascicular and posterior thalamic nuclei do specifically mediate play urges.

The parafascicular area is also thought to participate in pain perception because it contains neurons that respond to pinpricks and comparable noxious stimuli.[44] It may be, however, that these stimuli are closer to nipping or tickling ones than to painful ones. In this context, it is worth recalling that in humans, intense tickling is almost unbearable. Dorsal contacts may generate stimulus effects resembling the types of provocative stimuli that are especially effective in activating neurons in this brain area. It is of considerable import that human laughter systems have also been associated with these brain zones.[45]

Obviously, play recruits many brain abilities concurrently, and it is to be expected that many neural circuits are called into action during RAT play. There are bound to be powerful influences from the vestibular, cerebellar, and basal ganglia systems that control movement. However, little is known about the ludic functions of these brain areas, since extensive damage to them compromises virtually all of the animal's complex motor abilities. For instance, in some early unpublished work, we inflicted extensive bilateral damage to the caudate-putamen nuclei of several young rats; their play was abolished, but so was their appetite, curiosity, and desire to exhibit simple locomotor acts. They had to be sacrificed, since they were incapable of taking care of themselves. Obviously, that line of research could not have provided convincing evidence for the role of those brain areas in play. Large lesions in other areas, such as cerebellum, temporal lobe/amygdala and lateral hypothalamus, also markedly reduce play, but again, the overall behavioral competence of the animal is so impaired that it precludes any interpretation with respect to specific play circuitries. Smaller lesions are generally more interpretable, and early anectodal observations suggest that play circuitry is not heavily concentrated in the amygdala or temporal lobes. For instance, monkeys exhibiting the Klüver-Bucy syndrome, although emotionally placid and socially deranged, were "always eager to engage in playful activities with the experimenter."[46] Likewise, the comparable initial experiment with cats reported that the lesioned subjects exhibited augmentation of pleasure reactions and were generally playful, docile, and friendly.[47]

It is worth emphasizing that the neocortex is not essential for play.[48] Even though decortication, such as that depicted in Figure 15.6, can reduce pinning behavior to about half of control levels, those effects are not due to a reduced playfulness, since play solicitations and overall roughhousing, as monitored by direct activity measures, remain intact.[49] The reduced levels of pinning appear to be due to the animal's reduced willingness to respond to play solicitations by rolling over on its back. This may again reflect a heightened level of somatosensory and social insensitivity.

Contrary to the observations of other investigators, in our experience, massive lesions of the cingulate cortex also have little effect on the play of rats. Substantial increases in play can be produced with large frontal lesions[50] as well as septal ones, suggesting that those brain areas participate in the developmental processes that normally diminish play as animals mature. Other lesions may arouse emotional states that are incompatible with play. For example, VMH lesions, which make animals pathologically aggressive (see Chapter 10), will markedly reduce play.[51]

Clearly, the study of play circuitry remains in its infancy, and new techniques are needed to identify the relevant brain systems. One of the most promising techniques would be to analyze early gene markers of neuronal activity, such as *cfos* expression, described in Chapter 5. Using this approach, it becomes evident that wide fields of cells in the higher brain stem and telencephalon are activated during RAT play. This seems to be a common feature of all emotional processes—vast areas of the brain are aroused during each emotional state. As is evident in Figure 15.7, play elevates *cfos* expression in such medial thalamic areas as the parafascicular, in the hippocampus, and in many higher-brain areas, especially the somatosensory cortex.[52] Thus, even though decortication does not eliminate play motivation, it seems clear that play has powerful effects on the cortex. In other words, one of the adaptive functions of juvenile play may involve programming various cortical functions. In a sense, the cortex may be the playground of the mind, and PLAY circuits may be a major coordinator of activities on that field of play. Unfortunately, aside from such data as are summarized in Figure 15.6, there is presently no compelling evidence to support such a contention. A similarly unsatisfactory level of closure on key issues exists at the neurochemical level.

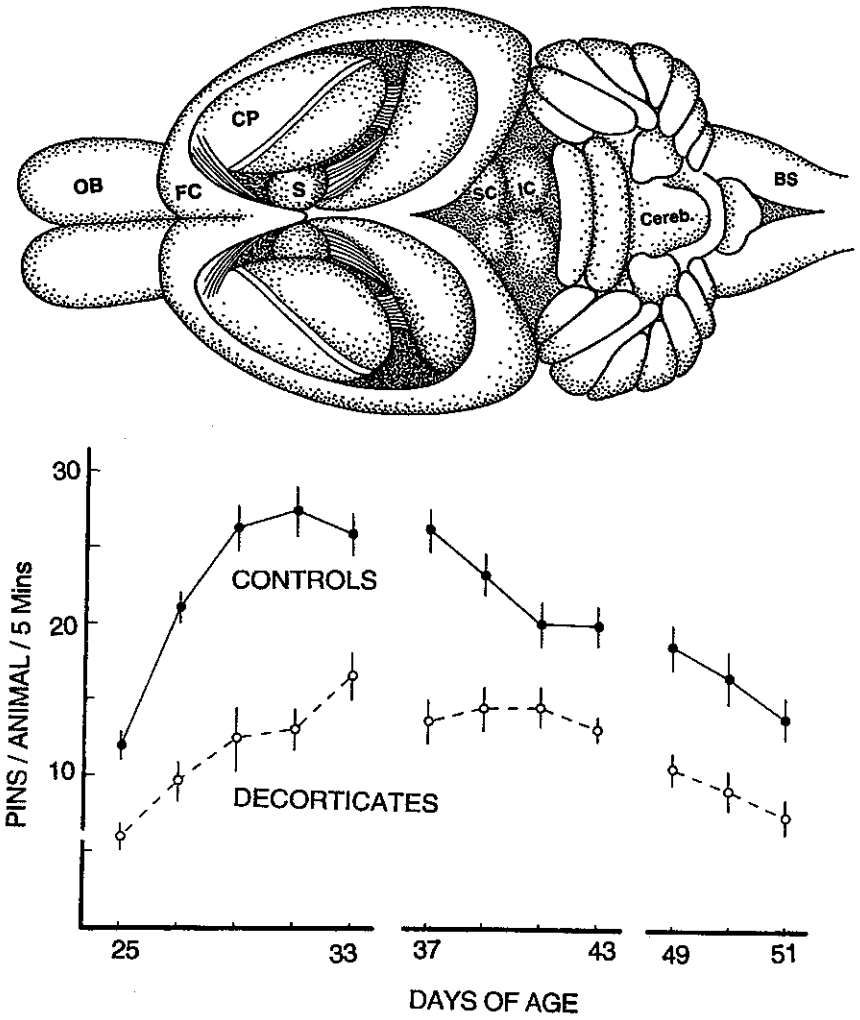

Figure 15.6. Play as a function of development in decorticate and control animals. (According to Panksepp et al., 1994; see n. 49.) The anatomical drawing depicts the appearance of the brain following the neonatal decortication, with many of the structures clearly visible that were described in Chapter 4, especially the hippocampus (HC), the caudate nucleus (CN), and the septal area (S).

The Neurochemistry of Play

It is remarkably easy to inhibit play using pharmacological manipulations, but it is very difficult to determine whether the effects reflect specific changes in the underlying regulatory mechanisms or merely the generally disruptive psychological and behavioral effects that many psychoactive drugs produce. Likewise, a great number of environmental manipulations can reduce play—including all events that evoke negative emotional states such as fear, anger, and separation distress. In addition, hunger is a powerful inhibitor of play,[53] as are many other bodily imbalances, including, of course, illness. In short, play is both a robust and a fragile phenomenon. When animals are healthy and feel good, play is an appealing psychobehavioral option. When they feel bad, it is not. Presumably many of these negative factors will have neurochemical underpinnings, and if we arouse them in a play context, play will be reduced (see Figure 1.1). Unfortunately, such ma-

nipulations do not measure the normal processes whereby an individual attains play satiety (i.e., reaches a healthy state of having played enough). Because of such specificity problems, which beset all behavioral experiments to some extent, it will be difficult to sort out those manipulations that reduce play because of physiologically important PLAY regulatory effects from those that reduce play for many other reasons.

One reasonable criterion for establishing that certain neurochemical systems have specific effects on play is to demonstrate that drugs that facilitate and inhibit neural transmission in a given system have opposite effects on play. With this criterion in mind, there is presently considerable evidence that opioids specifically modulate play. Low doses of morphine can increase play, and opiate antagonists can reduce play (even though, as highlighted in the previous chapter, these same manipulations decrease and increase the desire for social interaction, respectively).[54] Presumably, the reduction in play following opiate antagonists is a result of reduced activity and heightened negative emotionality, such as might be produced by mild arousal of separation-distress circuits. If the latter is the main cause, one would expect opioid blockade (at doses that normally reduce play) to increase play solicitation in animals that feel very secure about their social situation. Indeed, when tested against a totally nonthreatening, nonreciprocating partner who has been made unplayful via administration of cholinergic blocking agents such as scopolamine, animals treated with naloxone gradually begin to exhibit heightened play solicitations.[55]

In addition, as previously mentioned, play-dominance studies suggest that brain opioids may increase feelings of "social strength"; hence, animals treated with opiate antagonists are consistently submissive to normal control animals as well as those treated with low doses of morphine (Figure 15.3). Indirect evidence (from in vivo subtractive autoradiography studies)[56] suggests that there is widespread release of opioids in the nervous system during play, especially in such brain areas as the medial preoptic area, where circuitries for sexual and maternal behaviors are situated.[57] Of course,

Figure 15.7. Photomicrographs depicting Fos protein labeling in parietal cortex, hippocampus, and dorsomedial/parafascicular thalamus of rats that had played for half an hour and those that had been placed into the play chamber alone. Each dark dot represents a neuron that is expressing the neuronal activity marker cFos. I thank Steve Siviy (1997) for sharing these data; see n. 52.)

to facilitate play, opiate doses must be kept low. Higher doses of opioids reduce all social behaviors including play, and very high doses of opiates reduce all behaviors and induce a catatonic immobility. In any event, we can conclude that modest brain opioid arousal promotes play, and ongoing play promotes opioid release, which may serve to gradually bring the play episode to an end. However, opioids are surely not the only factor mediating play motivation, for it is not possible to restore playfulness in older rats or younger rats that are satiated with play by administration of low doses of opiate agonists or antagonists.

Many other neurochemical systems that have been studied also appear to have specific effects on play.[58] For instance, the muscarinic cholinergic receptor system appears to promote play; blockade of cholinergic activity with scopolamine or atropine markedly reduces play. Unfortunately, no one has yet been able to enhance play by activation of the cholinergic system. This may be partly because of the opposite roles of the nicotinic and muscarinic receptor components. Nicotinic receptor agonists reduce play, and antagonists mildly increase play.[59] Activation of serotonergic and noradrenergic systems also reduces play, while receptor blockade of certain of these systems can increase play somewhat.[60] Conversely, dopamine blockade reduces play, and most agonists do the same, which may indicate that animals need normal levels of synaptic dopamine activity in order to play. A comprehensive analysis of the many receptor subtypes within these biogenic amine systems should provide further clarity about their precise contributions.[61] Of course, all of these chemical systems participate in the control of a large number of brain and behavioral processes.

There may well be highly specific play-promoting neurochemicals in the brain, perhaps neuropeptides, but no such substance has yet been identified. Part of the problem in searching for relevant evidence is that virtually all of the neuropeptides must be administered directly into the brain, and we really do not know enough about play circuitry (especially about the relevant synaptic fields) to place the substances into the appropriate areas. However, we have evaluated the effects of a few neuropeptides, including oxytocin and CRF, both of which reduce play, while vasopressin does not appear to affect play.[62] We are still searching for that neurochemical system that will "turn on" playfulness in animals that are not psychologically ready to play, but this has proved to be a very difficult task (see the "Afterthought" of this chapter). It may require just the right combination of many manipulations. The fact that social deprivation increases the desire to play (Figure 15.1) suggests that it should be possible to produce such a state artificially. Only when someone has found a way to turn on play pharmacologically will we have achieved a profound neural understanding of playfulness, but even that may not reveal its adaptive functions.

The Functions of Play

The possible functions of play have been discussed extensively,[63] and the proposed ideas are remarkably wide-ranging. Suggestions fall into two categories: social and nonsocial. Among the first are the learning of various competitive and noncompetitive social skills, ranging from behaviors that facilitate social bonding and social cooperation to those that promote social rank and leadership, as well as the ability to communicate effectively. Among the potential nonsocial functions are the ability of play to increase physical fitness, cognitive abilities, skillful tool use, and the ability to innovate. Innovation can range from very generalized cognitive skills such as the ability to think creatively in a wide range of situations to very specific aptitudes such as learning to hunt among young predators and predator-avoidance skills in prey species. The collective wisdom is well summarized in the instruction sheet to Lego® toys that was quoted earlier. Unfortunately, there is no substantial scientific database for any of these ideas.

One could also propose a variety of additional fitness-promoting effects of RAT play, such as inoculation against social stress in future adult competitive encounters[64] or perhaps the facilitation of social attractiveness and skill so that reproductive potential is enhanced. Indeed, perhaps play even allows animals to hone deceptive skills, and thus in humans may refine the ability to create false impressions. It almost goes without saying that play must increase reproductive fitness in some way, but it should be noted that sexual-type behaviors are very infrequent during the course of RAT play in animals. Indeed, in unpublished work, we have been unable to find any evidence that male rats that had been socially deprived during the entire juvenile period (21 to 45 days of life) exhibited any deficiency at maturity in the latency and onset of sexual behaviors toward a hormonally primed female. However, if placed into a competitive situation, play-experienced animals were more effective in thwarting the advances of play-deprived animals than vice versa. Also, we have found that animals like to spend slightly more time with conspecifics that have had abundant play experiences than with those that have not.[65]

The best-documented beneficial effect of play discussed in the rodent behavioral literature is a mild increase in problem-solving ability in rats,[66] but in unpublished work we have not been able to replicate this effect. Other reported effects are decreased habituation to novelty in animals that have not experienced normal amounts of juvenile play and increased fearfulness in social situations.[67] Also, animals that have had much opportunity to play appear to be more effective in certain competitive encounters later in life,[68] but more data must be collected on these issues.

Although systematic work on this question is still in its infancy, there seems to be a growing consensus

that play is not superfluous, and that some distinct adaptive function should be demonstrable in a reasonably rigorous fashion. The issue of play functions in humans is muddied by the great variety of distinct forms of activities that are labeled as play, especially activities such as board games, where a great deal of previous learning is essential for the "play" to proceed. Indeed, it is generally believed that children learn more rapidly when they have fun, but the whole concept of play as it relates to educational ends remains murky.

By attempting to intentionally and formally recruit playfulness for educational ends, humans probably exercise many cortical potentials independently of PLAY-related functions. One is led to wonder to what extent the literature that has evaluated the role of play in facilitating learning and development of social competencies has simply evaluated the power of positive social interactions to facilitate desired educational goals.[69] It does seem that many of the supposed benefits of play that have been revealed by formal investigation simply reflect the beneficial effects of other types of social activities and supplemental tutoring.[70] There is presently no assurance that the many play interventions that have been studied in laboratory settings do in fact arouse primary-process PLAY circuits intensely. Of course, it remains very attractive to assume that the consequences of playful activities are beneficial for learning, but unfortunately, there are no robust and credible demonstrations of this in either humans or animals.[71] Once we have a clear understanding of basic PLAY circuits in the mammalian brain, it may be possible to monitor the development of behavioral and social competence in animals deprived specifically of normal activity in those circuits. Such experiments may be able to yield some definitive data.

Play and Dreaming

One straightforward perspective is that during play all of the natural (unconditional) emotional-behavioral potentials of the brain can be exercised. However, in addition to the relatively obvious functional hypotheses summarized here, only a few of which have even modest empirical support, it is to be expected that play may also be important in the functional control of brain organization. One molecular view might be that play promotes certain types of neuronal growth. A higher-level view is that play may serve to exercise and extend the range of behavioral options under the executive control of inborn emotional systems.[72] In fact, play may be the waking functional counterpart of dreaming.

As discussed in Chapter 7, a key function of rapid eye movement (REM) sleep may be to promote the processing of information that is especially important for complex emotional integration. PLAY systems may serve a similar function during waking. Since one of the characteristics of play is that many types of emotional behaviors are exhibited in the context of nonserious interactions, it is reasonable to hypothesize that play exercises the behavioral potentials of emotional circuits (Figure 15.8). According to this view, play may serve a function that is orthogonal to that of REM sleep: Namely, REM may exercise the potentials for organizing affective information in emotional circuits, while play exercises the emotive behavioral potentials of these same circuits in the relative emotional safety of a positive affective state. In other words, dreaming and play may have synergistic functions—providing special opportunities for exercising the psychobehavioral potentials of emotional operating systems within socially supportive environments. Thus, there could be as many behavioral variants of play as there are primary emotional systems within the brain.

A relationship between REM and PLAY processes is suggested by the fact that both are under strong cholinergic control. If there is, in fact, a neural continuity between REM and PLAY impulses, one might expect that

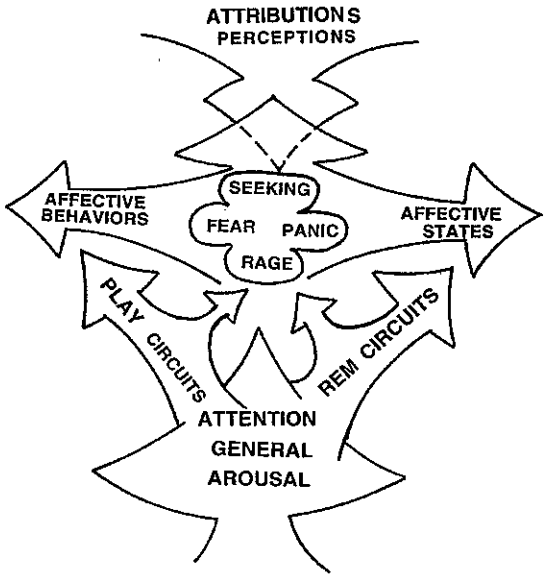

Figure 15.8. Emotional circuits are embedded in multiple convergent control processes such as startle, REM, and play circuits. REM may preferentially influence the higher affective consequences of emotive circuits, thereby helping process information that was collected during waking through the auspices of the various basic emotional circuits (see Chapter 7). Play may preferentially access the motor subroutines that are normally accessed by emotional circuits, thereby providing exercise and practice of instinctual motor patterns that are essential for competent emotive behavior patterns. (Adapted from Panksepp, 1986; see n. 72.)

RAT play may be characterized by unique EEG signals—for instance, large spikes during play jumpiness, which may resemble the PGO spikes of REM (see Chapter 7)—but this has never been evaluated. Also, since norepinephrine and serotonin neurons are silenced during REM, it is of interest that modest pharmacological reduction of activity in these two systems can modestly increase play.

Obviously, play will provide food for thought to scientists for a long time to come. Indeed, perhaps play provides "food for thought" for the brain, as recurring patterns become especially well consolidated into new habits during dreams. It will be interesting to determine to what extent preceding play periods affect subsequent REM periods, and how REM deprivation affects the information that is processed in the midst of play (e.g., whether play dominance, such as summarized in Figures 15.2 and 15.3, emerges without subsequent REM).

From this perspective, it would also seem likely that play may have direct trophic effects on neuronal and synaptic growth in many brain systems. Although the evidence is modest, environmental enrichment, including social dimensions, has been well studied in lab animals,[73] and there is some evidence that the observed social effects are due to RAT play. Neuronal effects of social enrichment (such as increased brain RNA and heavier cortices) can be observed after as little as 10 minutes of exposure for four days during sensitive periods of juvenile development.[74] Another basic hypothesis concerning play that deserves more experimental attention is the possibility that play is a "neuro-tonic" that can have antistress, health-promoting effects. We have evaluated this possibility via analysis of corticosterone secretion to mild stress in play-experienced versus nonexperienced animals. Unfortunately, we found no evidence to support the hypothesis that RAT play can regulate other stress responses.[75] Still, I believe more work along these lines will yield positive findings.

Distinctions between Play and Exploration

One psychobehavioral dimension that deserves special attention in future work is the role of SEEKING circuits in playful activities.[76] It has been quite common in the human literature to combine play and exploratory activities under the same rubric.[77] This is more problematic than is commonly realized. The mammalian brain probably contains distinct circuits for arousal of roughhousing types of social interactions and others for the arousal of exploratory and investigatory activities. These circuits may not always operate in synergistic ways. For instance, one highly effective way to reduce RAT play in animals as well as in humans (as indicated by the observation of "hyperkinetic" children) is to administer psychostimulants such as amphetamines, which concurrently increase attention and investigatory activities.[78] Such data raise the possibility that activities in PLAY and SEEKING systems may typically be antagonistic rather than synergistic.

Since the exploratory urge seems to be triggered to a substantial extent by brain dopamine activity,[79] it has been of some interest to determine whether dopamine systems are aroused during the course of RAT social play. To evaluate this, we once placed small amounts of the dopamine neurotoxin 6-hydroxydopamine into the nucleus accumbens at doses that did not debilitate the animal, but no clear effects on play were observed.[80] However, others have found some reduction in play from placing this same toxin into the dorsal striatum.[81] In more recent work, we measured the levels of forebrain dopamine and 3.4-dihydroxyphenylacetic acid (DOPAC), the metabolite that is commonly taken to reflect impulse flow in dopamine systems of the brain. Twenty minutes of RAT play led to an apparent increase of utilization of dopamine in the brain.[82] This result suggests that certain dopamine neurons are especially active during play, which is not surprising from the perspective that brain dopamine controls psychomotor arousal related to the perception of positive incentives (see Chapter 8). However, a determination of whether the same populations of dopamine neurons are active during the various forms of play and exploration will require a finer analysis of neural changes than has yet been achieved.

At present, it seems reasonable to provisionally conclude that basic exploratory and PLAY circuits in the brain are distinct, and that they normally operate antagonistically. However, we should remain open to the possibility that vigorous activity of the SEEKING system is a source process for what is typically called *object* or *manipulative play*. Because of concerns such as these, it will be difficult to determine to what extent the massive child development literature on the effects of "play" on psychological development reflects the functions of brain systems that control roughhousing play, as opposed to those that control exploration. These distinct psychobehavioral processes should not be placed under the same verbal construct.

There is presently an urgent need to determine what contributions to child development are in fact exerted by the PLAY circuits of the brain. Since roughhousing play cannot be readily studied in long-term and controlled experiments in human children, the use of animal models will be essential for adjudicating critical issues. Even more important, since this emotional system may be subject to pathologies, just like all the other emotional systems, it is worth considering how knowledge about the underlying circuits may relate to psychiatric disorders.

Play Disorders: Mania, Impulse Control Disorders, and Hyperkinesis

Since the PLAY circuitry of the brain appears to represent a fundamental emotional system, it is to be ex-

pected that there may be psychiatric disorders related to overarousal and underarousal of the system. Underarousal may well be related to certain types of depression and melancholic responses. Overarousal may be related to various manic symptoms, hyperkinetic or attention deficit disorders, and perhaps even Tourette's syndrome and other impulse-control disorders.[83] At present, there is no direct evidence for such assertions, but the symptoms of mania—expansiveness, unrealistic optimism, excessive happiness, and grandiosity—are the types of psychological symptoms one might expect from a highly playful brain.

Attention deficit hyperactivity disorders (ADHD) and impulse-control disorders are due in part to underarousal of cortical functions.[84] If we accept that heightened cortical activity can inhibit playfulness, it might well be that many children diagnosed with ADHD may, in fact, be exhibiting heightened play tendencies. Their hyperactivity, impulsiveness, and rapid shifting from one activity to another may be partly due to their unconstrained and unfocused playful tendencies. Indeed, the medications that are used to treat the disorder—psychostimulants such as methylphenidate (i.e., Ritalin®) and amphetamines—are all very effective in reducing playfulness in animals.[85] Moreover, parents of hyperkinetic children often complain that one of the undesirable side effects of such medications is the reduced playfulness of their children.[86] Obviously, parents value these childlike characteristics and are typically disturbed when the children's natural playfulness is pharmacologically diminished.

If at least part of ADHD is caused by excessive playfulness, it becomes a profound societal issue whether it is ethical to drug children for such traits (for more on this question see "Afterthought," Chapter 16). Obviously, it is essential to maintain attention to academic matters in the classroom, but is it appropriate to induce compliance in children through pharmacological means? At the very least, more benign interventions should be attempted first, such as provision of abundant RAT activity early in the morning prior to classes. This is especially important in light of the possibility that such drugs can produce long-lasting changes in the responsivity of brain catecholamine systems, as is seen in the psychostimulant-induced sensitization phenomenon (see "Afterthought," Chapter 8).

Finally, it is worth considering that Tourette's syndrome, with its bizarre nervous impulses—which lead to tics and sudden verbal expletives, commonly including "forbidden" expression such as curses and slurs[87]—may represent aberrant play impulses, or components of play impulses, circulating without restraint within the nervous system. Although this may seem far-fetched, pharmacological evidence provides some support for the hypothesis. Dopamine blocking agents, which presently are most effective in bringing Tourette's symptoms under control,[88] are also very effective in reducing playfulness in animals.[89]

Although these connections are highly speculative, if we keep our minds open to such possibilities, we may achieve a better understanding of both the nature of play and other perplexing disorders of childhood. In this context, it might be noted that autistic children typically like RAT play; if this is essentially the only type of social activity a child seems to favor, it is taken to be consistent with the diagnosis of autism.[90] This is also consistent with the idea that such children may have slightly excessive opioid activity, a brain chemical state that is compatible with abundant RAT play in animals (see Figure 15.3).

One thing is certain: During play, animals are especially prone to behave in flexible and creative ways. Thus, it is not surprising that play interventions have been used in educational and therapeutic settings (i.e., play therapy) to facilitate more efficient acquisition of new information and behavioral change. However, since play is fun, it could also be used more effectively as a reinforcer for desired behavioral change. To what extent would children be willing to discipline themselves with academic tasks if the availability of roughhousing play were made contingent on good academic performance? The benefits, for both classroom discipline and educational progress, might be enhanced if the availability of roughhousing was used to systematically reward scholarly achievement. But this would require us to begin viewing this ancient evolutionary brain function as a potentially desirable activity, rather than a disruptive force whose energies need to be suppressed or dissipated on the playground after the earnest business of education has been completed.

It is worth considering whether it might be possible to develop maneuvers to reduce disruptive RAT play impulses in the classroom, while utilizing opportunities to release those impulses as a reward for scholarly achievement. This approach is used by some high schools and colleges to increase the probability that athletes also become scholars, but it needs to be implemented in the earlier grades. Of course, the bottom line is that play is such fun. If we were able to make the process of learning more playful, the whole enterprise of education might become easier. The computer revolution now allows us to pursue such joyous modes of cultural development.

AFTERTHOUGHT: Future Research and the Search for Ludic Cocktails and Fountains of Youth

In addition to the many hypotheses that have been generated concerning the possible adaptive functions of play—ranging from the idea that play promotes muscular development to the possibility that play promotes the generation of new ideas—there are other provocative alternatives. For instance, it could still be the case that a major adaptive function of play is simply the generation of a powerful positive emotional state. This

could have direct health benefits by establishing a certain type of neurohumoral tone in the brain and body, which may promote more vigorous immunological patterns and other beneficial physiological responses. There are instances in the literature of prominent individuals claiming they have experienced remarkable medical benefits in the midst of serious illnesses by sustaining playful attitudes accompanied by abundant laughter.[91]

In a sense, play is an index of youthful health. From this vantage, the search for play transmitters can be thought of as a modern version of the search for the fountain of youth. It is the PLAY instinct that, more than any other, uniquely characterizes the joy of youth. Presumably there are brain chemistries, or combinations of chemistries, that can vigorously promote playfulness, but they have not yet been found. One way to discover them would be to identify neurochemical influences that gradually lead to the diminution of play as organisms grow older. The period of childhood has been greatly extended in humans and other great apes compared with other mammals, perhaps via genetic regulatory influences that have promoted playful "neoteny."[92] Indeed, we humans have the longest childhood of any creature on the face of the earth. One influence that might be irreversible is the maturation of the neocortex, which may tend to inhibit RAT ludic activities or at least channel those energies in different, more symbolic, directions.

Another way to understand playfulness might be to consider why playful impulses tend to return during adulthood, when one has offspring of one's own. Generally, parents seem to be more playful than nonparents, and it is reasonable to suppose that this tendency is promoted by neurobiological vectors in addition to the obvious cultural ones. In this context, it is worth reemphasizing that motherhood promotes specific neurochemical changes in the brain. For instance, oxytocin gene expression is increased, which certainly helps promote parenting behavior.[93] Perhaps this same neurochemical change promotes playfulness. For this reason, we evaluated the effects of intraventricular injections of oxytocin on play, but, as already noted, we only observed reductions in play. Vasopressin did seem to increase play slightly, but the results were not definitive. Thus, we presently only have hypotheses regarding which changes during parenthood may promote playfulness. Although we have an abundance of neuropharmacological data suggesting a variety of inhibitory influences on play circuitry,[94] we presently have no way to markedly increase playfulness in a nonplayful mature animal, except by play deprivation.

Over a decade ago, we took some of the more suggestive items from the list of available pharmacological manipulations that seemed to mildly promote play to see if we could generate some combinations that would facilitate play in a vigorous fashion. We were hoping to find a "ludic cocktail." The items selected included the opiate receptor agonist morphine, the serotonin receptor antagonist methysergide, and the dopamine receptor agonist apomorphine, each of which, given at low doses, had exhibited some tendency to increase play. These drugs were given in all possible permutations (a single drug, or two or three drugs concurrently), using various levels of social deprivation that should have allowed one to see both increases and decreases in play. These efforts were eminently unsuccessful. No combination of drugs seemed to clearly potentiate play, and each of the agents alone was, at best, marginally effective. However, we have recently had some modest success with cannabis-like molecules.

It remains possible that age-related decrements in play emerge from a diminished vigor of the underlying play circuits rather than a diminished availability of "play transmitters." If this is the case, it will be unlikely that a "ludic cocktail" can ever be generated, and the search for this "fountain of youth" will be as unproductive as the ones that have gone before. However, many lines of inquiry remain to be pursued.

Indeed, the pursuit of the neurochemical fountain of youth is becoming an active area of inquiry. Already, a series of agents have been found to exert powerful effects on longevity. I will discuss only one here—the antidepressant monoamine oxidase inhibitor deprenyl, which can selectively increase dopamine availability in the brain.[95] In fact, deprenyl is highly effective in reducing the symptoms of Parkinson's disease[96] and also provides neuroprotection against the progressive degeneration of dopamine systems that occurs with aging.[97] The vigor of brain dopamine declines markedly in most individuals after the age of 50, and most would become parkinsonian if they lived long enough.[98] Deprenyl, given daily in low doses, reduces this decline and seems to promote youthful vitality: It extends the maximal life span in animals by almost 30%, and male animals that have become sexually sluggish tend to regain their lustiness.[99]

It will be interesting to see how such agents influence the ontogeny and dynamics of play throughout juvenile and adult development. One would think that agents that can maintain psychological vitality would also tend to increase playfulness. Indeed, we should also consider the reciprocal idea—whether playful social companionship may actually extend life span. It is disconcerting how little work is presently being devoted to trying to understand the underlying mechanisms and the adaptive nature of this and other fundamental emotional processes of the mammalian brain.

Suggested Readings

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