CHAPTER 1

# THE SENSE OF DESIRE

*Smells are surer than sights or sounds to make your heartstrings crack.*

—NABOKOV

On November 22, 1997, Michael Hutchence, lead singer for the internationally renowned Australian band INXS, was found hanging naked in his hotel bedroom, the noose tied with his own leather belt. The last person to see him alive, onetime fling and longtime friend Kym Wilson, was at first suspected to be complicit. She insisted that she and her boyfriend had left Michael fully clothed the night before after a visit in his hotel room. Kym was soon relieved of suspicion and suicide confirmed as the mode of death. But why would the excessively successful and apparently want-free Michael Hutchence take his own life? What could have drawn him into such a deep depression that he would ultimately kill himself? Various accounts by friends and associates, including interviews with Michael Hutchence himself, point to a pivotal and life-altering event that could very well be the precipitating link to his suicide.

In September 1992, Michael Hutchence was in a freak traffic accident. Riding his bicycle home from a nightclub in Copenhagen, he was struck by a car and suffered a fractured skull. In an interview a few months after Michael's death, the journalist Robert Milliken reported in a feature in *The Independent* (March 1998), that: "His friends are convinced that the accident was a turning point that led to increasing bouts of depression and reliance on Prozac." Richard Lowenstein, the avant-garde Australian filmmaker, told Milliken that ever since the accident Michael was on a slow decline. He had never seen any evidence of depression, erratic behavior, or violent temper before, but saw all those things afterward, and he confessed that one night in Melbourne, Michael had broken down in his arms and sobbed: "I can't even taste my girlfriend anymore."

What was it about this accident that so scarred Michael Hutchence? Did he suffer undiscovered brain damage with pathological effects, or was it something more basic and obvious? Michael was a devout hedonist and completely sensual being.[1] A self-confessed decadent, Michael's gourmand tastes and lust for life were centered around consumption, and now these lascivious pleasures were irrevocably altered, because the accident had stolen his sense of smell.* Without the sense of smell, the temptations of food, the sweaty funk of sex, the

*Although his disability was described as losing "smell and taste," people frequently believe that they have lost their sense of taste when they have only lost their sense of smell. This is because flavor is predominantly produced by smell not taste per se (see Chapter 7). It is most likely the case that Michael Hutchence only lost his sense of smell.



essence of a walk on the beach, the feeling of nostalgia—the texture of life itself—were robbed from him. From all accounts, after this accident Michael fell into an increasingly debilitating depression from which he never emerged. As his melancholy progressed, he resorted more and more to both prescription and illicit drugs and alcohol, but these mind-numbing devices were in vain. Could it be that losing his sense of smell, which killed his most basic life pleasures, had such a cataclysmic effect on his well-being that he felt life no longer worth living? From all I know about the sense of smell and the consequences of its loss, this could very well be so.

My suspicion that loss of smell, medically referred to as *anosmia* (smell blindness), was a crucial factor in the suicide of Michael Hutchence is based on my insights into neurological, psychological, and clinical evidence. First, the neurological interconnection between the sense of smell (olfaction) and emotion is uniquely intimate. The areas of the brain that process smell and emotion are as intertwined and codependent as any two regions in the brain could possibly be. Smell and emotion are located in the same network of neural structures, called the *limbic system*. The limbic system is the ancient core of the brain, sometimes called the *reptilian brain* because we share it with reptiles, and sometimes called the *rhinencephalon*—literally, the "nose-brain." The key limbic structure to interact with our olfactory center is the *amygdala*. The amygdala is the brain's locus of emotion. Without an amygdala we cannot experience or process emotional experiences, we cannot express our own emotions, and we cannot learn and remember emotional events. Brain imaging studies have shown that when we perceive a scent the amygdala becomes

activated, and the more emotional our reaction to the scent, the more intense the activation is. No other sensory system has this kind of privileged and direct access to the part of brain that controls our emotions.

Clinical research on patients who have lost their sense of smell also suggests that Michael Hutchence's anosmia could have led him to suicide. After an acute trauma such as a head injury, which causes anosmia, patients often report a loss of interest in normally pleasurable pursuits, feelings of sadness, loss of appetite, difficulty sleeping, loss of motivation, inability to concentrate, and thoughts of suicide that can turn into action if not treated.[2] These symptoms are all key diagnostics for major depression as described by the *DSM-IV*,[3] the clinician's bible for classifying psychological disorders. The link between smell loss and depressive symptoms is correlational in humans, but cause and effect has been experimentally verified in laboratory animals. Rats who have had their olfactory bulbs surgically removed, and thereby can no longer perceive smells, display physiological and behavioral changes that are strikingly similar to those that occur in depressed people. They stop eating, lie around their cages, and are oblivious to the toys and activities that they normally vigorously enjoy.

Studies of people afflicted with anosmia also indicate that the development of depression is progressive. In one study that contrasted the trauma of being blinded or becoming anosmic after an accident, it was found that those who were blinded initially felt much more traumatized by their loss than those who had lost their sense of smell. But follow-up analyses on the emotional health of these patients one year later showed that the anosmics were faring much more poorly than the blind. The emotional health of anosmic patients typically continues to deteriorate with passing time, in some cases requiring hospitalization and in more tragic cases, such as Michael Hutchence's, ending in suicide.



I have been contacted by people worrying that their anosmia will *cause* them to commit suicide. They have read or heard a story like Michael Hutchence's and they are experiencing symptoms of depression themselves. The first person who asked me about this was Jessica Ross,* a woman I got to know well when I became the expert witness in her claim against an insurance company.

ONE DAY ABOUT TWO YEARS AGO I was working in my office when the telephone rang. The caller introduced himself as Bill Adams, a partner in a well-established law firm in Florida. He explained that they had a case where a woman had lost her sense of smell in a car accident, and they needed a scent expert to determine the validity and extent of her claims. The purpose of his call was to find out whether I would be willing to be an expert witness in this case.

Jessica Ross, twenty-eight years old, recently married and working at an accounting firm, was coming home from a party seated in the backseat of a car, when it collided with a truck on the highway. She was thrown forward and smashed against the

*The names and identifying features of individuals in this case have been changed.

front seats and windshield. Her face took the brunt of the impact, particularly the area around her eyes and forehead, and in less than a year she had already undergone three operations to correct the damage. Jessica's cranial fractures were at the level of the eyebrow, which would have severely affected the brain area behind it—the olfactory bulbs and the olfactory cortex, where our sense of smell is processed. The *olfactory bulbs* are two blueberry-shaped and -sized extensions of the brain, one for each nostril. Separating the neurons in our nose from the olfactory bulbs in our brain is a very thin and fragile bone called the *cribriform plate*. The cribriform plate is riddled with thousands of tiny holes through which the ends of the olfactory receptor neurons (called axons) pass through to get into the brain. When there is a violent blow to the front of the head, it knocks the cribriform plate out of alignment, causing the delicate olfactory axons that run through it to be sheared off. Imagine slicing a cobweb with a knife. With the axons cut off, the olfactory nerve is dead and the sense of smell is destroyed. The axons can never regenerate, and smell loss is permanent. Many types of frontal head injuries can easily lead to losing the sense of smell; for example, football players often suffer the same fate after a hard frontal tackle to the face. This is how Michael Hutchence's fractured skull made him anosmic.

After giving me this report, Adams asked if I thought it was possible that the car crash caused Jessica to lose her sense of smell. Indeed I did. He also explained that although her smell loss was apparent to her many months earlier, she was just now discovering how devastating this disability was. Jessica was now seeking to sue the responsible parties for her anosmia and



her loss of quality of life. Adams told me that prior to contacting me he had searched through available documentation but had been unable to find any legal precedent or medical validation for the severity of anosmia. The American Medical Association *Guides to the Evaluation of Permanent Impairment* currently gives the loss of smell and taste a value of only 1–5 percent of the total value of a person's life's worth, while loss of vision is given a value of 85 percent. In spite of this disregard for the importance of the sense of smell, could I help to justify how significant Jessica's disability was? Could it actually be comparable to losing sight?

When her lawyer called me, Jessica was cosmetically put back together. Her doctors had reported no central brain damage, and she was declared of sound mind and body—except for her sense of smell. To confirm her complaints, Jessica had been given a smell identification test* and received a score indicating pure anosmia. There was no doubt in my mind that her smell loss was total and permanent. I asked Adams to arrange a telephone interview so that I could find out how Jessica was coping.

For several hours I bombarded Jessica with a stream of questions concerning all aspects of her life. Jessica told me she felt disconnected from other people and, worse yet, disconnected from her *self*. She believed that she was now incompetent as a homemaker and caretaker, she had lost interest in sexual intimacy, and formerly gregarious, she now avoided

*The "UPSIT" (The University of Pennsylvania Smell Identification Test), a scratch-and-sniff odor identification test that is widely used in medical diagnoses of anosmia.

social contact as much as possible. More than anything else, Jessica complained that her emotional life had taken a markedly negative turn since the accident and that she felt generally depressed. "My husband says my personality has changed," she confided. When I asked her to explain these changes, she said that since the accident she was more irritable, was more ambivalent about other people and cared less what others thought of her, and that she felt sad a lot of the time and cried frequently. And haunting her through all of this was a growing worry that at any moment there might be something terribly wrong and that she wouldn't know it because she couldn't detect it as she might have done before with her nose: fire, spoiled food, and even her own body odor.

Throughout our conversation Jessica's voice was flat and dull. Her symptoms and her voice displayed all the classic characteristics of depression. Jessica was also distressed because she felt that her emotional state was getting worse with time. Unfortunately, Jessica was right. The derailing of our olfactory system caused by anosmia can have a progressively negative downstream effect on the healthy functioning of our emotional system.

## THE DEPRESSION-OLFACTION LOOP

Jessica Ross, Michael Hutchence, and clinical studies have shown us that anosmia can lead to depression. Conversely, it also turns out, depression can lead to loss of smell. Patients with serious depression often complain to their therapists that they think their ability to detect odors is suddenly weakening. Are they losing their minds? No. Depression can truly bring



about olfactory loss. Odor sensitivity tests on people who have been diagnosed with major depression show significantly diminished ability to detect scents at normal concentrations. The intimate and interdependent link between emotional and olfactory health is underscored even further by the fact that after treatment with antidepressant medication, smell sensitivity improves in these patients.[4]

Oddly, people who are plagued by a particular form of depression called *seasonal affective disorder* (SAD) show *increased* sensitivity to smells compared with their happy compatriots, and surprisingly this superior acuity exists yearround. This is surprising because SAD doesn't manifest like classical depression, which can happen at any time and usually lasts for many months or longer. Rather, SAD is a depression that only emerges in the winter, when light levels are low; in the springtime, people with SAD rebound to being normal happy people, or sometimes become manic—hence, the name *seasonal* affective disorder. The winter symptoms of SAD include: depressed mood, increased appetite, increased sleeping, and lack of motivation. Although SAD has prominent effects on mood, recent research suggests that it may primarily be a disorder of circadian rhythms, a suite of physiological rhythms that include when we feel sleepy and awake and that are highly influenced by light and fluctuate around a twenty-four-hour clock. When light levels are low, as they are in the winter, depression sets in, and when the hours of daylight increase in the spring, so does mood. The crucial role of light in SAD is further emphasized by the fact that people with SAD can be effectively treated with light

therapy.* It also may explain why the sense of smell is not diminished as it is in classical depression. My belief is that rather than amygdala interactions, which influence the relationship between smell and classical depression, in SAD another limbic structure is at the crux of the depression-smell relationship—the *hypothalamus*.

You may have already noted that some of the depressive symptoms of SAD are different from those seen in major depression; for example, increased versus decreased sleeping and eating. The hypothalamus has primary control over our drives for eating, sleeping, aggression, and sex. In animals who hibernate, it is also the area of the brain that controls hibernation. Several researchers have suggested that SAD may be a psychological echo of hibernation. In keeping with this idea it turns out that irregularities in the functioning of the hypothalamus occur among people with SAD. Like all limbic structures, the hypothalamus interacts with the olfactory system. I believe that the hypothalamic aberrations in SAD may make the olfactory cortex more sensitive to smells than it normally would be, rather than less responsive as it is when the amygdala is malfunctioning.

Another biological process controlled by the hypothalamus is the menstrual cycle. Women's sensitivity to smells also varies with menstrual cycle phase, becoming particularly heightened around the time of ovulation when they are most fertile. This has

*Recent research suggests that SAD is most effectively treated with 10,000 lux for half an hour each morning, with the patient sitting about sixteen inches away from the light source (see Dr. Robert Levitan, Center for Addiction and Mental Health, University of Toronto).



significant biological implications because it turns out that smell is a critical factor in heterosexual attraction and finding the "right" mate, particularly for women, as you shall see. Women are also much more likely to develop SAD than men, and this isn't simply a corollary of higher head counts of depressed women than men. The same hypothalamic mechanisms involved in smell sensitivity and women's sexual physiology may also be responsible for altering smell sensitivity in SAD.

MORE THAN ANY OTHER SENSORY EXPERIENCE, fragrances have the ability to trigger our emotions: to fill us with joy and rage, to bring us to tears and make our hearts ache, to incite us with terror, and to titillate our desires. Have you ever been stricken with a feeling of dread, not known why, and then noticed a strange smell in the air? Thousands of New Yorkers had this exact experience walking in the streets and riding through the subway stops near the World Trade Center during the months after September 11, 2001. The strange charred and dusty scent was an instant reminder of that historical terror. On a more pleasant note, have you ever experienced the wonderful feelings of comfort and serenity that the scent of fresh, damp earth and moss invites after the rain? These examples illustrate how every day, scents affect our emotional lives in exceptional ways, triggering moods and emotional memories.

Not only do odors trigger emotions, they can also *become* emotions. My studies have shown that odors can literally be transformed into emotions through association and then act as proxies for emotions themselves, influencing how we feel, how

we think, and how we act, I call this *odor-emotional conditioning*. In my laboratory we found that by pairing the feelings of frustration with an unfamiliar odor we were later able to make that odor alter behavior in accord with being in a frustrated mood. In one study, children who experienced an odor that had formerly been associated with trying to complete a frustrating maze showed less motivation and did more poorly on a simple test when exposed to that odor than children who had suffered through the frustrating maze but were exposed to a different odor or no odor during the test. In another study, adults exposed to a "frustration-associated odor" spent less time and were less motivated to solve challenging word problems compared with others who underwent the same procedures but were not exposed to this scent during the word problem test. For the disadvantaged participants, the scent had become conditioned to, and hence equated with, feelings of frustration, such that later the presence of the odor alone could influence the individuals to behave in a frustrated and unmotivated way. The odor caused the same behavior as experiencing the actual emotion would have.

Positive associations to odors can also lead to positive emotional conditioning, and there are many potential applications and benefits to society that could be developed from this connection. By linking feelings of intellectual competence to a specific odor and then using this odor when confronted with challenges at work or school, odor-emotional conditioning could be used to improve performance and productivity among individuals with low scholastic or job morale. Odor-emotional conditioning could also be used to improve social behavior in stressful settings. For example, by linking positive emotions to



a specific odor, the odor could then be used to reduce violent or antisocial behaviors. In an effort to determine the effect of fragrance on social behavior, Susan Schiffman, a psychologist at Duke University, sprayed pleasant scents, such as chocolate chip cookies, into New York City subway cars and then tested whether the presence of these ambient aromas would make riders less aggressive. And indeed she observed that riders in the scented cars pushed, shoved, and made rude comments with almost half the frequency as riders in the unscented cars.

## SCENT-EMOTION TRANSLATION

Why is it that our experience of scents and emotion are so interconnected, and what does this reveal about us and these two systems? I have a theory, based on evolutionary principles and insights into my own work, clinical findings, and research on emotion, that may explain why. A *chemical sense* was the first sense to appear in the mobile life-forms that emerged on the earth, and it is the only sense that the most primitive single-celled creatures share with us today. Its fundamental purpose was and is to detect chemicals to enable the organism to know what is good and what is bad "out there" for the basic goal of survival. Is this a good chemical (like food), or is this a bad chemical (like poison)? Do I approach or do I avoid? This is olfaction in its most basic form. From this very simple survival guide, the sense of smell has evolved to be a highly intricate go/no-go system for finding food and mates, establishing social hierarchies, assessing whether or not to be aggressive or fearful, avoiding predators, and many other complex behaviors. The sense of

smell is the primary sense by which most of our animal brethren negotiate the world, including our primate relatives, and it is the sense to which they owe their survival. For us, vision has taken over as the primary sense that facilitates our survival. Yet odors still evoke in us the remnants of this primeval survival code.

The most immediate reaction we have to a scent is an assessment of *good* or *bad*. Approach what smells good, avoid what smells bad. Emotions also convey a simple message that is similar. Positive emotions such as joy and interest tell us to approach, to go forth and multiply—and ultimately result in our successful procreation and survival. Negative emotions such as anger, fear, and disgust tell us to avoid—and facilitate our survival by triggering a flight-or-fight response. Our emotions impart the same approach and avoid codes that smell imparts to other animals.

The connection between smell and emotion is not only metaphorical but also is founded on the evolution of our brain. A primitive olfactory cortex was the first fabric of our brain and from this neural tissue grew the amygdala, where emotion is processed, and the parts of the brain that are responsible for basic memory and motivation—the collective structures of the limbic system. In other words, the ability to experience and express emotion grew directly out of our brain's ability to process smell. I have often wondered whether we would have emotions if we did not have a sense of smell; *I smell therefore I feel?*

In my opinion, the human emotional system is a highly evolved, abstract cognitive version of the basic behavioral motivations instigated by the olfactory system in animals. Emotions are to us what scents are to our animal cousins. Smell for animals informs survival in direct and explicit ways; for us its primary survival codes have been transformed, into our experience of emotions. I call this *olfactory-emotion translation*.



Olfactory-emotion translation proposes that the sense of smell and emotional experience are fundamentally interconnected, bidirectionally communicative and functionally the same. I believe that the human brain has co-opted the survival-guiding olfactory system of animals into our survival-guiding system of emotions. Why do I believe this?

As I indicated earlier, we have discovered from patient research that with smell loss comes depression and with depression comes smell loss. With anosmia the olfactory neurons that normally also excite the amygdala are no longer activated, and over time this cessation in activity from the olfactory cortex causes atrophy and/or dysfunction in the amygdala—sowing the seeds for clinical depression. When depression comes first, I surmise that because the amygdala is functioning abnormally, the healthy activity that would otherwise be stimulating the olfactory cortex is skewed or compromised. That is, dysfunction in the amygdala alters the normal functioning of the olfactory cortex, and dysfunction in the olfactory cortex alters normal functioning of the amygdala. The olfactory and emotion areas of our brain are dependent upon each other for their mutual health and integrity. When one side malfunctions, it affects the functioning of the other. There is ultimate synergy between smell and emotion, but it is not always positive.

From a psychological perspective, our immediate responses to scents are simple, almost instantaneous assessments of liking or disliking; and many emotion theorists consider this

fundamental "liking" response to be the basis of our highly complex emotional system. Moreover, the experiences of emotion and olfaction are similarly primal, visceral, and removed from verbal-semantic analysis. We have the same kind of difficulty using words to deconstruct our emotional experiences as we have articulating our experiences of smell. Try to explain why you love her, and what the attic smelled like. There is a fundamental analogy and bidirectional interaction between our experiences of smell and our experience of emotion.

The reason the bicycle accident that made Michael Hutchence anosmic was a crucial factor leading up to his suicide is because loss of the sense of smell brings with it severe disruption of mental health and happiness, while smell's intact state brings texture, richness, and a brilliant emotional quality to life in innumerable ways. The emotional enrichment imparted by scent is particularly striking for someone whose life is filled with its pleasures and vividness. Awareness and appreciation of scent actually increases our capability to smell. Recent studies at the University of California, Berkeley, have shown that when one is paying attention to and perceiving a scent, a part of the brain is activated that is otherwise quiet when that same scent is there, but you don't *notice* it.[5]

From biographical accounts it seems that Michael Hutchence was not simply aware of scents, he relied on them to intoxicate and transport him. Like Jessica Ross, life for Michael Hutchence without a sense of smell had become dark and dull, and the darkness grew larger and ever more enshrouding with the passage of time. For someone who took pleasure so seriously and could no longer indulge in one of its primary vehicles,



it was like being a drug addict and losing the high from heroin—never able to regain that high and constantly seeking it, I believe desperation finally got the better of him.*

## THE SMELL OF FEAR

The paradox for my proposition of a two-lane highway between smell and emotion is whether we can smell emotions. Are you afraid of dogs? If so, have you noticed that when you are around them, a so-called friendly pet has an uncanny tendency to single you out for torment? Or perhaps after being cajoled into horseback riding against your wishes, your horse, of all the ones in the ring, rears and tries to jump the fence. If these examples strike a chord, then you know that many animals seem to be able to detect our anxieties. What is it that these animals are sensing when they exploit our vulnerabilities? The answer is, they are smelling our fear.

The sweat on your tracksuit after a three-mile run is mainly composed of water, but when we are nervously waiting for medical test results, the sweat under our arms, and elsewhere, reflects the secretion of glands regulated by our nervous system and hence our emotional states. This sweat is more pungent than the sweat of exercise and has a characteristic odor, and like dogs and horses, we can recognize it, too.

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*I do not claim that anosmia was the sole reason for Michael Hutchence's suicide, but that it contributed to a depressive mind-set that made it more difficult for him to cope with a number of negative factors that were impinging on his life at the time.

Research by Denise Chen, at Rice University in Texas, has demonstrated that humans understand the scent of fear, as well as happiness, in one another. To investigate whether we can smell each other's emotions, she had college students watch excerpts from happy and frightening movies while they wore gauze pads in their armpits. The pads were then collected and aggregated by gender for happy and fearful male and female sweat. Another group of young men and women were then asked to use their noses to judge which emotional category the underarm pads were from. Women were able to judge happy sweat from both male and female donor pads, while men were better at identifying the scent of happy rather than fearful women. But both men and women were best at recognizing the scent of male fear. Female fear sweat was also recognizable but because male sweat is usually stronger than female sweat, it was easier to classify.[6]

## THE HARDWARE

What are these smells that can comfort us, frighten us, and inspire our deepest passions? Smells are chemicals, and they can be extremely complex and contain thousands of molecules, like the rose scent emanating from your flower bed, which is made up of between twelve hundred and fifteen hundred different molecules. Or they can be very simple and comprise just one molecule, like phenylethyl alcohol, the chemical that imparts the scent of rose in many commercial hand lotions. Surprisingly, my research has shown that we cannot reliably tell these two versions of an odor apart—the synthetic fake rose and the



rich, natural flower bed—and if asked which is the fake and which the real thing, we are more likely to err in favor of exclaiming the artificial aroma as being "the real thing." This is because we are often more familiar with the manufactured version of "natural" scents than the natural scents themselves and so these synthetic copies become the prototypes for what we believe these fragrances *should* smell like.

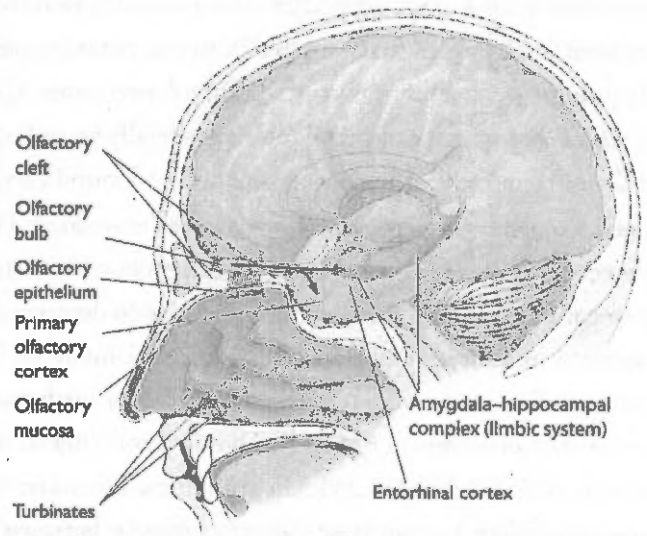
Not all chemicals, whether a single molecule or massive bundles, can be smelled. For humans to be able to smell a chemical, it must be of low molecular weight,* volatile, and able to repel water, so that it can stick to our olfactory receptors. However, there are also exceptions to the rule, and we don't detect all small, airborne, greasy chemicals. One common example is methane (natural gas). We also can't smell carbon monoxide, which is a by-product of methane. If you use natural gas in your home, you may think that it has a skunky/rotten-egg smell, but this is only because utility companies add a compound, tertiary-butyl mercaptan, to natural gas so that you can "smell" if your pilot light has gone out. In addition to not being able to smell carbon monoxide, we also cannot smell the gases that make up the air we breathe. Pure air is made up of about 78 percent nitrogen, 21 percent oxygen, and 1 percent a mixture of eight other gases including carbon dioxide, helium, and neon, none of which we can smell.

*Molecules that have a molecular weight of greater than 350 daltons cannot be perceived through our sense of smell. One dalton is equivalent to the weight of a hydrogen atom ($1.657 \times 10^{-24}$ grams). A simple water molecule has a molecular weight of 18.

Every day we inhale at least twenty-three thousand times, and with each breath comes the opportunity to perceive the chemicals whispered by a rose on a soft summer night, the chemicals nestled in the crook of your lover's neck, the cake baking in the oven, or the effluents of dirt, mold, gas, and decay—every scent that you have ever smelled. With each breath, air containing odor molecules enters our nostrils and is swept upward into the nasal passages of the nose (see page 21). In addition to being the location of the olfactory receptors, the function of the nose is to filter, warm, and humidify the air that we breathe. The interior of the nose is not simply a smooth cavity but possesses a complex geometry of ridges shaped by the underlying turbinate bones. The geometry of our nose, and the air convection system it generates, is more complex than the airflow around an aircraft wing. This special architecture allows the air containing odor molecules to pass through a narrow space called the olfactory cleft, where the chemicals then make contact with the olfactory receptors.

The olfactory receptors are buried in two patches of yellowish mucous membrane, called the *olfactory epithelium*, about seven centimeters up from each nostril. The olfactory receptors are on the tips of the olfactory neuron dendrites. We have approximately 20 million olfactory receptors covering the epithelium of both our right and left nostrils. However, our 20 million receptors would look paltry to a dog, like the bloodhound, who has about 220 million olfactory receptors. Olfactory receptors, unlike the receptors in any other sensory system, are directly exposed to the outside world, which is why among other things we can inhale drugs.






From Wolfe et al.: *Sensation and Perception*, 2006; Sinauer Associates, Inc.

After the chemicals that we smell make contact with the receptors on the olfactory epithelium, nerve impulses are passed from the olfactory receptor axons through the cribriform plate and into the olfactory bulbs. From the olfactory bulbs, scent information is then relayed to the olfactory cortex and the limbic system, and from there on to diverse regions of the brain, including the vision, taste, and touch centers—which is why, for example, we find that food artfully arranged on a plate "tastes" better than if it is all mashed up.

We have more receptors for smell than we have for any other sense except vision, yet the area of our brain that is devoted to olfaction is very small, about 0.1 percent of our total brain size.

The fact that such a small proportion of our brain is devoted to the sense of smell has led to the myth that we are not very good at smelling, but this statement is only true in relative terms. Compared with dogs and mice our noses aren't especially keen, but we can still sniff out most of the scents that a bloodhound can, we just need a much higher concentration of the odor to do so. Dogs can detect odors at concentrations nearly 100 million times lower than we can. This is the equivalent of being able to detect a drop of chocolate in a city the size of Philadelphia. Salmon are also amazing smellers and can detect the scent of their birth waters at a concentration as low as $3 \times 10^{-18}$. That is, 1 milliliter of scent diluted in 333,333,333,333,333,333 milliliters of water. The average nonchalant human nose can discriminate between ten thousand and forty thousand different odors, and professional smellers—literally called "noses" in the fragrance industry—and perfumers, whisky blenders, and chefs may be able to discriminate upward of one hundred thousand odors. The only real difference between an expert and an average nose is training. Technically, anyone with a healthy olfactory system can perceive an enormous number of aromas, the only limiting factor being the number of smellable molecules that are out there.

## FROM THE BAKERY DOOR TO CROISSANT CRAVING

The basic anatomy and physiology of our sense of smell is well understood, but oddly the deeper question of how a set of chemicals goes from wafting out of a bakery door to calling out "croissant" to you still remains a mystery. For all our other senses we



have a complete understanding of both physiological and psychological processes, but in olfaction we are lagging far behind.

The reason we are delinquent in understanding our most basic sense is that the sense of smell was not deemed worthy of study until very recently. The concept of "odor" was branded with negative connotations in the Victorian era. Then the notion that animals smell—that is, stink—but *civilized* people did not, or rather "should not," reigned, and this view has contaminated endeavors to explore and understand our sense of smell ever since. Research psychologists and neuroscientists are also to blame for why olfaction has been relegated to the sensory dustbin. The long-standing excuse has been that odors cannot be properly studied because their physical properties are very hard to precisely control and the responses to them are subjective; therefore, studying smell is "unscientific." It is true that to impose the same level of physical rigor in testing olfaction as one could when testing hearing, for example, is difficult, but that doesn't mean that the scientific investigation of olfaction is inherently compromised. It just means that you have to ask somewhat different kinds of questions, and use somewhat different techniques, as you will discover throughout this book. And the appropriate questions and methods to study olfaction aren't restricted to psychological science; they include fundamental biology and chemistry as well.

The decades of waving away the scientific legitimacy of olfaction ended with a breakthrough discovery in 1991 that not only made olfaction "scientifically" scrutinizable but made it one of the hottest and most desirable areas of research to be in. Linda Buck and Richard Axel, working at Columbia University,

versity, published a paper in the journal *Cell* that led to their winning the Nobel Prize for Physiology or Medicine in 2004.[7] It was the first time ever that a Nobel Prize had been awarded to anyone or anything having to do with the sense of smell. Buck and Axel discovered that there are about a thousand different odor receptor types, each coded for by a different gene. What this means is that in the nose (their test animal was the mouse) there are one thousand different types of receptors that decode the chemicals in the air into smells and that each of these receptors is represented by a different gene in the body. For comparison, in vision there are only four types of receptors—rods for black-white (night) vision and three types of cones, sensitive to short, medium, and long wavelengths of light, for color vision.* Buck and Axel also discovered that there were a lot of *pseudogenes*—DNA sequences that are remnants of genes that are no longer functional—in that family of one thousand olfactory receptor genes. In a dog, about 15 percent of its olfactory receptor repertoire are pseudogenes, the mouse has about 20 percent. In humans, the number of pseudogenes is much higher—about 65 percent. Precisely how many olfactory genes are functional in humans is currently under debate but it is on the order of three hundred to four hundred. Even though between 60 and 70 percent of our olfactory genes don't work, 1 percent of all the genes in our body is committed to regulating olfaction, which is far more genetic devotion than anything else in our bodies and brains gets.

*Short wavelength corresponds to seeing violet-blue, medium to green-yellow, and long to orange-red. A new photosensitive ganglion receptor cell has just been identified in the retina that modulates circadian rhythms.



Until very recently it wasn't understood why we would have so many olfactory genes that no longer do anything. But in January 2004, researchers at the Weisman Institute in Israel proposed an answer. Yoav Gilad and his colleagues observed that in Old World primate species such as gorillas and rhesus monkeys about 30 percent of their olfactory receptor genes were pseudogenes, whereas most New World species (e.g., squirrel monkeys) have a lower proportion—around 18 percent. The one New World exception is the howler monkey, which has around 30 percent of its olfactory receptor repertoire as pseudogenes. It turns out that the howler monkey and Old World primates have something in common with us—trichromatic (red, green, blue) color vision. Other mammals who lack trichromatic color vision, such as mice and dogs,* also have few olfactory receptor pseudogenes. The theory is that with the emergence of full color vision we lost the need for detecting the world so keenly with our sense of smell, and there was essentially an exchange in importance between these two senses in primate evolution. The better you can see, the less acutely you need to smell. Animals, including humans, either have excellent color vision or an excellent sense of smell, but not both. The finite size of the human brain is to blame. The human brain is limited to between 1,300 and 1,400 grams in weight. Having a highly complex sense of both smell and vision would take up too much brain space and so these functions had to compete with each other for which had the better survival value. It seems superior visual detection was better for our ancestors' survival than superior olfactory acuity, and the advantages offset the

*Dogs are red-green color-blind.

limitation to our sense of smell; hence, the large proportion of pseudogenes in our olfactory code.

Buck and Axel's seminal discovery of olfactory receptor genes produced a research explosion in the molecular biology and biochemistry of smell. With this energetic work we are now closer than ever to understanding how olfaction works, but a final theory for how molecules are translated into perceptible scents has still not been fully realized.

The most established current theory is referred to by both its advocates and detractors as *shape theory*. According to shape theory, the key to odor translation is the match between the shape of odorant molecules and odor receptors. This idea is strikingly similar to an ancient theory of smell involving shape that was suggested by the Roman philosopher Lucretius. In its modern form, shape theory got its legs in the 1960s from John Amoore, an independently minded biochemist, olfactory theorist, and entrepreneur. It was then further developed and refined by Gordon Shepherd and his colleagues at Yale University during the 1970s. In a nutshell, modern shape theory contends that odor molecules have different shapes, and odor receptors have different shapes; the ability for an odor to be detected is determined by how a specific molecule is recognized by specific olfactory receptors. From the most recent molecular research it seems that the detection of a scent is done by a combinatorial code. Different scents activate different arrays of olfactory receptors in the olfactory epithelia, producing specific firing patterns of neurons in the olfactory bulb. The specific pattern of electrical activity in the olfactory bulb then determines the particular scent we perceive. The scent of a mango elicits a different pattern of neural impulses than the smell of skunk.



One of the surprising results to emerge from the recent receptor work is that changing the concentration of an odor will also change the receptor code. That is, a weak concentration of baking butter sets off a different firing array than a high concentration, which explains why you need to be within a certain proximity of the bakery door to have the eureka moment of "croissant." The link between receptor code activity and intensity may also explain the differences in olfactory prowess between dogs and us. A dog is not necessarily detecting molecules that we cannot physically smell; rather, he is detecting them at a very much lower concentration than we are and therefore is capable of smelling things that are just "not there" to us. If all of our one thousand genes coded for functional olfactory receptors, our scent sensitivity would be completely overwhelming and all consuming, and I am sure human culture, civilization, and our experience of reality would be very different from what it is now.

In spite of growing support for shape theory, it is not accepted by all as the answer to how chemicals are detected by the cells in our noses and then become recognized by the brain as a croissant or kiwi. An alternate theory that has raised its head several times throughout history is that our perception of smells is based on the different vibrational frequencies of the molecules that compose various odors. In recent years this idea has been championed by Luca Turin, the emperor in Chandler Burr's *The Emperor of Scent*. In essence, *vibration theory* proposes that due to their atomic structure, there is a different vibrational frequency for every perceived odor molecule, and molecules that produce the same vibrational frequencies will have the same smell. Turin reported that chemicals that have predictably similar vibrations

due to their molecular composition also have similar smells. For example, all citrus-smelling odors are in the same class of vibrational frequency. But independent researchers testing this theory have not validated his claims. There has been much less research pursuing *vibration* than there has been for *shape,* so current research trends may unfairly bias our understanding. Nevertheless, vibration theory, unlike shape theory, cannot explain several conundrums of olfactory perception, such as "specific anosmias" and the different scents produced by stereoisomers.

Stereoisomers are molecules that are mirror-image rotations of one another, and although they contain all the same atoms, they can smell completely different. For example, *d-carvone* (the right-handed isomer) smells like spearmint, and *l-carvone* (the left-handed isomer) smells like caraway. Shape theory explains this as being because the rotated molecules do not fit the same receptors—like trying to put your right hand into your left-hand glove—and thus different receptors are activated for these two molecules and different scents are perceived. Vibration theory cannot explain why stereoisomers smell different from one another because the vibration of stereoisomers is the same.

Another mark in favor of shape theory comes from the study of specific anosmias. A *specific anosmia* is the inability to smell one specific compound with otherwise normal smell perception. Most specific anosmias, which appear to be genetic, are to steroidal musk compounds—animalic-sweaty scents within a specific chemical family. The most studied specific anosmia is an inability to smell the steroidal musk *androstenone.* Fifty percent of the population has a specific anosmia to androstenone. However, of the remaining 50 percent, about half describe



the smell as a *sweet musky-floral,* while the other half describe it as an unpleasant *urinous* odor. Vibration theory cannot explain why some people perceive the scent as a sweet-floral, others as urinous, and why yet others can't smell it at all. However, shape theory can account for these observations by proposing that in individuals with this specific anosmia the receptors that detect androstenone are nonfunctional, while among those who can smell it, different receptors are activated in those who perceive floral than in those who perceive urine.

From where we are now, shape theory has better support and explanatory value, but it is also true that all molecules vibrate, and all receptors are made of atoms that vibrate as well. This leaves room for vibrational interactions between odor molecules and receptors to play a role.

Beyond the unresolved theory for the translation between a chemical in our nose and a smell percept in our olfactory bulbs, the more profound question of how the pattern of activity in our olfactory bulbs becomes psychologically organized, processed, interpreted, and recognized by you as "croissant" with all its motivational and hedonic sequelae remains unknown. What we think a certain scent is, its connection to language and concept, what the scent means to us, what it makes us feel, and what it reminds us of—all interact in a complex multifaceted dance and determine our *perception* of that scent. Odor sensation happens at the level of our nose and olfactory bulb, but olfactory perception occurs in our mind, where our personal experiences with scents take over.