11

AUTONOMIC SPECIFICITY AND EMOTION

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Autonomic specificity refers to the notion that emotions can be distinguished in terms of their associated patterns of autonomic nervous system activity. This idea has a long history in psychology, tracing back at least to James's (1884) writings on the nature of emotion. Moreover, it is an idea that has always been shrouded in controversy, attracting many critics along the way (e.g., Cacioppo, Klein, Berntson, & Hatfield, 1993; Cannon, 1927; Schachter & Singer, 1962; Zajonc & McIntosh, 1992). The controversy has been framed by two immoderate assertions: (1) Every emotion is autonomically unique; and (2) every emotion is autonomically the same.

The uniqueness assertion is generally associated with Alexander's (1950) psychosomatic hypotheses. The second assertion of sameness arguably finds its clearest statement in Mandler's (1975) writings. Needless to say, these are both statements in extremis, and it would be difficult to find undiluted, unhedged versions of either in the contemporary literature. Nonetheless, they form the two poles around which participants in the debate over autonomic specificity have aggregated over the decades. In my view, both of these assertions are highly dubious.

Regarding the first assertion of uniqueness, as I hope this chapter makes clear, it is highly likely that reliable autonomic differences only exist for a small number of emotions. Moreover, where these differences do exist, they are likely to be "prototypical" in nature, with particular occurrences of a given emotion showing variation around these central tendencies.

Similarly, there is ample basis for rejecting the second assertion of no autonomic differences among emotions. An examination of the empirical literature reveals many studies that report evidence of autonomic specificity. A flurry of such studies appeared after Ax (1953) developed a paradigm for using "real life" inductions to study this issue in the laboratory. Another flurry appeared 30 years later following our report (Ekman, Levenson, & Friesen, 1983) that used directed facial actions and relived emotional memories to address the same question. In contrast, there are surprisingly few published empirical studies that report failures to find any evidence for autonomic specificity. Most of the support for the "sameness" position comes from a number of influential critiques that have either discounted autonomic specificity on a priori grounds (e.g., Cannon's argument that the autonomic nervous system was structurally incapable of supporting specificity; Cannon, 1927) or that have criticized existing data (e.g., Zajonc & McIntosh, 1992) without presenting any new data. The other source of "support" for the sameness position has derived from studies that have followed the paradigm introduced by Schachter and Singer (1962) in which the autonomic nervous system is activated by using some nonemotional agent (e.g., injection of epinephrine). Participants' emotional labeling of the resultant state is shown to be quite malleable, reflecting cues in the experimental environment. The original study and its progeny have been criticized on numerous grounds over the years (e.g., Plutchik & Ax, 1967; Reisenzein, 1983). However, beyond any methodological problems, the application of the findings from these studies to the question of auto-

nomic specificity is logically flawed. A study that manipulates autonomic nervous system physiology (that is, in which autonomic physiology is the independent variable) cannot be used to study the autonomic concomitants of emotion (in which autonomic physiology has to be the dependent variable).

It would be useful to consider the empirical literature on autonomic specificity as a whole. However, to my knowledge there have been no formal meta-analyses of the research findings in the area. Several informal aggregations of results do exist, but these are much less useful in settling controversies. For example, a number of years ago, I took the four most reliable autonomic differences among emotions that were found in our work and reviewed quite a large body of relevant research from other laboratories, concluding that there was a substantial amount of evidence in support of these four instances of specificity (Levenson, 1992). In the same volume, Zajonc and McIntosh (1992) published a paper highlighting other, much less reliable, findings in our work. Needless to say, the conclusions of these two papers regarding the evidence for autonomic specificity were quite different. Thus it is not surprising that more than a century after James's initial proposals and more than a half century after Ax's initial empirical forays, the issue of whether there is autonomic specificity in emotion is still far from settled.

Why Might Autonomic Specificity Exist?

The idea that emotions are likely to have different patterns of autonomic nervous system activity is grounded in an evolutionary view of emotion that suggests that emotions were selected for their ability to help the organism deal effectively and efficiently with a small set of problems that were critical for the species survival (for a thorough presentation of this position, see Tooby & Cosmides, 1990). Viewed from this perspective, emotions can be seen as time-tested solutions to timeless problems and challenges, such as defending what is ours, avoiding harm, attracting potential mates, regulating social distance, soothing and restoring equilibrium, and engendering help from conspecifics. With our emotions, evolution has provided us with at least one generalized response to these problems that has a high likelihood of being successful most of the time. In humans this emotional response encompasses multiple psychological and physiological systems, some of which serve to prepare the organism for action, some of which serve to regulate the behavior of conspecifics, and some of which do both. I have previously described these functions of emotion as follows:

> Emotions are short-lived psychological-physiological phenomena that represent efficient modes of adaptation to changing environmental demands. Psychologically, emotions alter attention, shift certain behaviors upward in response hierarchies, and activate relevant associative networks in memory. Physiologically, emotions rapidly organize the response of disparate biological systems including facial expression, somatic muscular tonus, voice tone, autonomic nervous system activity, and endocrine activity to produce a bodily milieu that is optimal for effective response. Emotions serve to establish our position vis-à-vis our environment, pulling us toward certain people, objects, actions and ideas, and pushing us away from others. Emotions also serve as a repository for innate and learned influences, possessing certain invariant features, and others that show considerable variation across individuals, groups, and cultures. (Levenson, 1994, p. 123)

This view presupposes an emotion system in which there exists some central mechanism that continuously scans the incoming stream of information from the external and internal worlds in search of certain configurations that represent a small number of problems and challenges that have significant consequence for the species's survival and well-being. Having recognized one of these prototypical configurations (e.g., being cheated; Tooby & Cosmides, 1990), the system activates the appropriate emotion (e.g., anger), which efficiently orchestrates a coordinated multisystem response that is highly likely to deal successfully with the problem. This response package is crafted from a number of disparate elements, drawn from a palette that may include perceptual/attentional systems (e.g., Mathews & Bradley, 1983), gross motor behavior, purposeful behavior (e.g., Frijda, 1986), expressive behavior (e.g., Ekman, 1984; Izard, 1971), gating of higher mental processes (e.g., Bower, 1981), and physiological support (e.g., Davidson, Ekman, Saron, & Senulis, 1990; Levenson, 1992). The elements of this package are mixed in their proper proportions, and the elements are choreographed in terms of the timing of onset, duration, and offset to produce a coordinated response.

Figure 11.1 presents a schematic for this kind of model of emotion (for a similar model, see Levenson, 1994).[1] The model has been simplified to emphasize systems primarily involved in studies of autonomic specificity. In this model, the emotion that is activated functions like a computer program that activates a set of subroutines for the various response systems (Tomkins, 1962). Thus there are sets of instructions for the facial muscles, the vocal apparatus, the skeletal muscles, and for various physiological systems, including the autonomic nervous system. These patterns of activation have two broad classes of functions: (1) preparing the organism to activate the behavioral response[2] that represents the generalized solution most likely to deal successfully with the eliciting situation, and

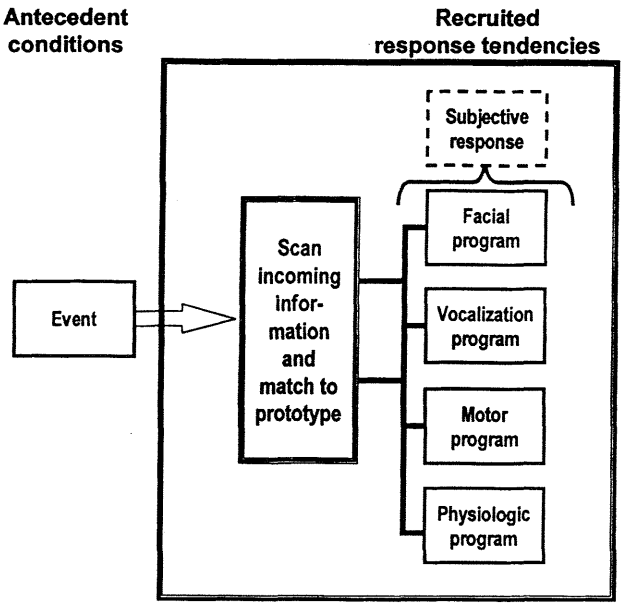

Figure 11.1 A schematic model of emotion.

(2) communicating the organism's emotional state to others in the service of altering their behavior. The likelihood that autonomic specificity exists derives support from both of these classes of functions.

The most commonly advanced argument for autonomic specificity derives from the first class of functions. If the emotion mobilizes a behavioral response or motor program (Frijda, 1986), it must also mobilize the configuration of autonomic nervous system activation necessary to provide optimal support for this particular set of behaviors. Moreover, this autonomic support should be produced on a "just in time" basis, so that the support will be there when needed and will not linger too long after the behavior is completed. Concluding the logic of this argument, if different emotions reliably call forth different patterns of behavior, and if these different behaviors require different configurations of autonomic support, then the pattern of autonomic nervous system activity should be different for different emotions.

The second argument for autonomic specificity, which is not emphasized as often as is the first, pertains to autonomically mediated appearance changes that accompany different emotions and that serve (along with changes in facial expression and in vocalization) as signals, communicating our emotional state to others. Many of these involve changes in coloration that result from alterations in local blood flow (e.g., flushing, blushing, blanching, bulging of arteries), whereas others involve additional detectable changes such as piloerection, sweating (and accompanying odors when apocrine sweat glands are involved), tearing and crying, and visible and audible changes in breathing. Of course, some of these autonomically mediated appearance changes are merely the observable manifestations of the previously discussed autonomic support for behavioral responses. Regardless, the specificity argument remains the same: If different emotions are reliably associated with different patterns of autonomically mediated appearance changes, then these should provide additional instantiation of autonomic specificity.

There is another aspect of this model that has important implications for autonomic specificity. Because the instructions associated with the emotion program create only tendencies to respond in certain ways, the observable emotional responses (which we measure in our laboratories in the form of changes in facial expression, vocalization, muscle tension, and autonomic and other forms of physiological activation) can vary in how closely they represent the response tendencies. If the onset gradient of the stimulus event is sharp, the match to prototype is close, and the stimulus intensity high, the activation of the emotion will be rapid and strong, with little opportunity for alteration of the response tendencies. Thus, in these situations, the observable emotional response would most closely resemble the generalized solution to the problem or challenge. In contrast, if the stimulus onset is gradual, the match to prototype only approximate, and the stimulus intensity low, then there will be ample opportunity for the person to alter (e.g., diminish, amplify, transform, mask) these response tendencies in accordance with learned emotional beliefs and practices (e.g., display and feeling rules; Ekman & Friesen, 1969; Hochschild, 1979). In these instances, the observable emotional response might be quite different from the prototype, depending on the person and situation. Thus, in terms of revealing autonomic specificity, stimulus situations of the first type, with sharp onset, close match to prototype, and high intensity, will be less vulnerable to interindividual "noise" and thus will be more likely to reveal any associated specificity of autonomic response. Unfortunately, in the laboratory, emotion elicitations are usually of the second type, with relatively gradual onset, approximate match to prototype, and mild intensity, and thus they are highly vulnerable to alteration, which works against finding evidence of autonomic specificity.

Why Is the Idea of Autonomic Specificity So Compelling?

Arguably, autonomic specificity is one of those ideas that is just too good *not* to be true. Consider the following thought experiments:

- Does your body feel the same way when you are afraid as when you are happy?
- Is your heart just as likely to race when you are disgusted as when you are afraid?

- Are you just as likely to cry when you are angry as when you are sad?
- Are you just as likely to say that your "blood is boiling" when you are afraid as when you are angry?
- Is your face just as likely to turn white and drain of blood when you are angry as when you are afraid?

For most of us, the answer to all five of these questions will be "no." Each of these "no" answers depends, in its own way, on the existence of autonomic specificity. Of course, these thought experiments may prove little other than that we share a set of common beliefs about the autonomic organization of emotion. But if emotions were truly undifferentiated autonomically, then what would be the source of these beliefs? And would not some of these beliefs, especially those that could be disconfirmed by casual observation, be called more frequently into question?

The notion of autonomic specificity is quite ubiquitous in our culture. Our metaphorical language of emotion is replete with references to differentiated bodily states. Linguists such as Lakoff (1987) and Kovecses (1989) have elaborated sophisticated theories based on careful analysis of the representation of the body in emotional language. In Lakoff's analysis of anger metaphors, the themes of "heat" ("burning up," "turning red") and "pressure" ("blowing my top") are most representative. The rising temperature and reddening themes in anger directly contrast with those of fear, in which themes of dropping temperature and turning pale are regularly found. As Lakoff has pointed out, the same temperature difference between anger and fear that is found in language has also been found in empirical work (e.g., Ekman et al., 1983; Levenson, Ekman, & Friesen, 1990). Moreover, the differences in the metaphorical language of anger and fear concur with consistent empirical findings of peripheral vascular differences between these two emotions (see Levenson, 1992, for a review). And finally, providing some preliminary support for a direct link between the nature of physiological metaphors and underlying physiology, we (Marchitelli & Levenson, 1992) reported a study of the use of anger metaphors of heat and pressure during discussions of marital conflict in married couples ($N = 144$ spouses). In this study, we found small but reliable correlations between greater use of heat and pressure metaphors and autonomic nervous system changes that indicate greater temperature (finger temperature: $r\,[140] = .17$, $p < .05$), greater blood flow (vasodilation: $r\,[142] = .37$, $p < .001$), and faster blood velocity (pulse transmission time: $r\,[143] = -.24$, $p < .01$).

Of course, such findings could merely reflect cultural conventions, socially constructed beliefs about emotion that would likely vary from culture to culture. However, recent cross-national surveys have also found marked consistencies in the autonomic sensations associated with different emotions, including the aforementioned temperature differences between anger and fear (Scherer & Wallbott, 1994).

The Importance of Autonomic Specificity

Implications for Emotion Theory

The question of whether emotions are associated with different patterns of autonomic nervous system activity is of fundamental importance to our understanding of the nature of emotion. Learning more about how the autonomic nervous system is organized in emotion would be of value in much the same way as would learning whether there are differences in patterns of regional brain activation or in patterns of facial muscle action in different emotions. Because emotions bridge mind and body and because they reflect both hardwired and learned influences, issues surrounding the biology of emotion often get caught up in larger theoretical controversies. This excess baggage has been a mixed blessing, drawing considerable attention to basic research on autonomic specificity but also increasing the likelihood that research findings will agitate one theoretical camp or another in a growing list of connected areas. I cite some examples here.

A case could be made that the "cognitive revolution" in psychology can be traced in some significant way to Schachter and Singer's (1962) study of cognition, physiology, and emotion. This study was widely interpreted as showing the primacy of cognition over emotion. If something as fundamental to the human condition as emotion could be made to dance like a puppet on the end of the strings of cognition, then cognition was clearly a force to be reckoned with. Schachter and Singer's two-factor model of emotion appeared to rest on the assumption of undifferentiated autonomic arousal in emotion. Thus, to many, any and all assertions that autonomic physiology was in fact differentiated in emotion would, at the very least, complicate the model and could in fact undermine one of its essential tenets.[3]

Autonomic specificity has also become involved in the controversy over the existence of "basic" emotions. In Ekman's model (Ekman, 1992), one characteristic of basic emotions is that they show differentiated autonomic nervous system activity (as well as unique facial signatures and other defining features). The entire notion of basic emotions was questioned by Ortony and Turner (Ortony & Turner, 1990; Turner & Ortony, 1992), thus setting off a spirited debate about whether there are some emotions that have special status by virtue of their unique biological features. Thus the issue of whether certain emotions have "autonomic signatures" whereas others do not has now become part of this controversy.

Another example is that of the universality of emotion.

After a long period in which emotions were considered to be socially constructed and thus culturally variable, evidence from cross-cultural studies suggested that facial expressions for emotions such as anger, fear, sadness, surprise, disgust, and happiness were universal (Ekman & Friesen, 1971; Ekman, Sorenson, & Friesen, 1969; Izard, 1971). A large part, but not all, of this evidence was based on studies in which participants in different cultures matched photographs of emotional expressions with emotion terms and emotion antecedents. The universalist position held sway in psychology until it was called into question by Russell (1994), setting off a heated controversy that still rages in some quarters. Evidence for the cross-cultural consistency of other aspects of emotion such as autonomic specificity would lend support to the universalist position. At this point there has been only one study that actually measured autonomic nervous system physiology during different emotions in more than one culture (Levenson, Ekman, Heider, & Friesen, 1992), and this study found evidence of cross-cultural consistency.

A final example derives from a number of controversies related to the parsing of emotional space. The major variant of this controversy is whether emotions are best organized in terms of *discrete* emotions or in terms of *dimensions* (usually a two-dimensional space consisting of valence and activation). Because most devotees of these models believe that they reflect the underlying biological organization of emotion, it is important to know whether autonomic nervous system differences reflect the discrete or the dimensional structure. Overwhelmingly, research on autonomic specificity has been carried out within the discrete-emotions tradition, but there have been a few exceptions, such as Winton and colleagues' work that attempted to map heart rate and skin conductance responses onto the pleasantness and intensity dimensions (Winton, Putnam, & Krauss, 1984). Evidence of autonomic differences among negative emotions, such as findings that disgust does not share the cardiac acceleratory characteristics of anger, fear, and sadness and that anger and fear differ in terms of peripheral vascular activity (Levenson, 1992), is more consistent with a discrete-emotions view than with a dimensional one.

Implications for Health

A great deal of the early interest in autonomic specificity was stimulated by the psychosomatic literature, exploring Alexander's (1950) speculations about the relationship between particular psychosomatic disorders, particular emotions, and particular patterns of autonomic physiology. These ideas were tested in research by Graham and others (e.g., Graham, Stern, & Winokur, 1958, 1960), who found that evoking attitudes (which roughly mapped onto different emotions) thought to be associated with psychosomatic diseases produced subclinical changes in organ systems relevant to the pathophysiology of the disease. Thus inducing an attitude thought to be associated with Reynaud's disease was found to produce decreases in hand temperature, whereas an attitude associated with hives was found to produce temperature increases.

Although these findings were quite promising, this line of research was not continued. Subsequent work on emotion and health, such as that showing the relationship between hostility and heart disease (e.g., Diamond, 1982; Williams et al., 1980), does not assume or test for autonomic specificity. Moreover, this subsequent work seems to mark a move away from the viewpoint that the *expression* of certain emotions is pathogenic toward the view that it is the chronic and repeated *restraint* of emotion that is most harmful. I base this assertion in part on the view that hostility is more closely associated with the suppression of anger than with its free-expression. Interestingly, there is very little experimental work on the cardiovascular impact of restraining emotion (there is, however, a great deal of correlational research). Experimental work from our laboratory indicates that suppressing emotion (i.e., restraining emotional behavior once an emotion is stimulated) has profound effects on the cardiovascular system, essentially doubling the magnitude of sympathetically mediated cardiovascular responses over the level present when the emotion is freely expressed (Gross & Levenson, 1993, 1997). Importantly, there appears to be little autonomic specificity in this aspect of emotion—the inhibition of three quite different emotions (sadness, disgust, and amusement) all have similar cardiovascular effects.

Research on Autonomic Specificity: Challenges and Obstacles

In previous work (Levenson, 1988), I addressed a number of methodological issues in autonomic specificity research in detail (see also chapter 12 in this volume). Here I address three more general problems that have historically plagued research in this area. Although I focus on the autonomic nervous system in this section, these same problems are also encountered in the emerging body of research on the role of other physiological systems in emotion (e.g., brain research using MEG, fMRI, and PET measures).

Emotion Elicitation

It is ironic that humans have emotions all of the time in their everyday lives, yet getting participants to experience a particular emotion at a particular time in the laboratory can be very difficult. To study the physiology of emotion regardless of whether it is autonomic, central, endocrine

Greater validity

ELICITOR	ECOLOGICAL VALIDITY	EXPERIMENTAL CONTROL	"DIFFICULT" EMOTIONS
Directed facial actions	Low	High	None (if subjects are good facial "athletes"); fear, sadness (if subjects are not)
Slides	Low-Medium	High	Anger, fear, sadness
Films	Medium	High	Anger
Relived emotions	Medium-High	Medium	None (if subjects are good imagers)
Staged manipulations	Medium-High	Medium	Sadness
Dyadic interaction among intimates	High	Low	None

Greater control

Figure 11.2 Emotion elicitation: Trade-offs between ecological validity and experimental control.

or immunological, we must have effective ways of producing what, for lack of a better term, might be called "real" emotions. In this regard, we must distinguish between situations in which participants make emotional judgments (e.g., rating a photograph of a crying child in ragged clothing as being "5" on a 7-point sadness scale) and situations in which they actually experience sadness. Of course, participants may experience sadness when viewing such a photograph, but they may not—their ratings in the latter case indicate that they perceive the photograph as having sad qualities but *not* that it makes them actually experience sadness. For the purpose of studying the physiology of emotion, we need to produce "real" emotional experience in our participants, but the structure and demand characteristics of our experiments are such that I believe we often fail in pursuit of this goal. Even more disturbing, even when participants say they are "feeling" emotion in our studies, they may, as in the earlier example, merely be providing a readout of the emotional characteristics of the experimental stimuli. If this happens, we are in effect studying the physiology of emotional judgments rather than the physiology of emotion—introducing a source of error that will serve as a major impediment to progress in this field.

Researchers who enter into the study of the physiology of emotion immediately encounter the problem of how to elicit emotions in the laboratory. It is tempting to simply adopt some method of elicitation that has a modicum of a priori face validity and assume that this method will produce the full range of emotions of interest. In our work, we have struggled with this issue of how to produce emotions in the laboratory. Over the years we have worked with directed facial actions (constructing emotional facial configurations), slides, films, relived emotions (emotional imagery), staged manipulations (e.g., threatened electrical shock at the hands of an incompetent experimenter; Ax, 1953), and dyadic interaction between intimates. Although historically most investigators have invented their own stimuli, investigators can now make use of standardized sets of slides (the International Affective Picture System [IAPS]; Lang, Greenwald, & Bradley, 1988) and films (Gross & Levenson, 1995).

Each of the methods commonly used for eliciting emotion has strengths and weaknesses. Figure 11.2 summarizes my experience with these methods. Invariably, the investigator is faced with a frustrating trade-off between ecological validity and experimental control. Thus tasks that are most similar to contexts in which human emotions typically occur (e.g., unrehearsed, minimally structured dyadic interactions between intimates) can be an experimental nightmare. In contrast, tasks with very tight experimental control (e.g., directed facial actions, which give the experimenter precise control of which emotion is displayed on the face and when) are not very representative of the ways in which emotions usually occur.

Furthermore, the six emotions commonly studied in autonomic specificity research (anger, disgust, fear, happiness, sadness, surprise) are not equally accessible using the different elicitors. Based on my own experience, and using a convergent criterion that considers an emotion most likely to have been successfully elicited if facial, autonomic, and subjective indicators are all present, I have included a column in Figure 11.2 that indicates the emotions that are particularly difficult to elicit using the various techniques.

Finally, there is the issue raised earlier about whether we are eliciting "real" emotions or not. With the directed facial action task, the experimental demand to report the emotion constructed on the face is high, even if that emotion is not actually felt. With external visual stimuli such

as slides and films, it is common for participants actually to feel no emotion but to report feeling emotions that in reality represent their judgments of the emotional qualities of the stimuli. The "real-life" elicitors (staged manipulations and dyadic interactions) seem most likely to produce "real" emotions but not without incurring a number of costs, including loss of experimental control, appearance of complex sequences of emotions, and, in the case of staged manipulations, serious ethical and human-participants issues.

Emotion Verification

Even if emotion elicitation tasks were usually successful in producing the desired emotion in most participants; even if the autonomic nervous system was inactive before and after being recruited in the service of emotion; even if emotion elicitations in the laboratory had the kind of sharp onset, close match to prototype, and high intensity that reliably produced full-blown emotional reactions; even if the autonomic concomitants of specific emotions were dramatically different; and even if effect sizes were huge, then it would still be critical to ensure that the autonomic physiology derived on a particular trial from a particular participant was in fact associated with the actual occurrence of the targeted emotion. In reality, none of these "ideal case" scenarios is likely to be true. Even the best of the available elicitation tasks often have unintended emotional outcomes; the autonomic nervous system is continually acting in the service of many masters other than emotion; laboratory-induced emotional elicitations are often pale comparisons of real-life ones; participants' emotional responses are often of low intensity and often include emotions other than the intended one; autonomic correlates of emotions are not unique but rather show complex patterns of overlap; and effect sizes are small.

For all of these reasons, it seems absolutely critical to verify the emotional state of participants on some independent basis and to derive conclusions about the autonomic correlates of emotional states from data obtained on trials on which participants are most likely to have actually experienced the targeted emotion. To fail to do this introduces a great deal of additional noise into an already noisy system and greatly undermines the value of such research in addressing the issue of autonomic specificity. The need for verification seems so compelling and so obvious that one would expect reasonable verification to be an important part of any study intending to assess autonomic specificity. With autonomic physiology serving as the dependent variable in these studies, verification of participants' emotional states needs to rely on some non-autonomic indicator such as emotional facial behavior or self-report or ideally both. And importantly, once the verification has been carried out, there must be some rational plan to use it to inform the subsequent data analyses. Has this in fact been done? Examination of the "classic" literature on emotional specificity reveals few, if any, studies that have adequately verified the emotional states of participants and used this information in a meaningful way in subsequent data analyses.

Consider these examples. Ax (1953) used an interview procedure to determine the emotional states produced by elicitors that targeted fear and anger. However, the interview was conducted 10 minutes after the second elicitor; thus, in the case of the first elicitor, participants were making retrospective statements about emotions that had occurred 30 minutes earlier. Moreover, the decision rules to deal with elicitation failures were not stated. Sternbach (1962) used a single film to study sadness, fear, pleasantness, and amusement. Participants were interviewed about their feelings 10 minutes after the film—all data were used in the subsequent analyses. Schwartz and colleagues (Schwartz, Weinberger, & Singer, 1981) used emotional imagery to study happiness, anger, sadness, and fear. Self-reports of emotion were obtained 7 minutes after each image ended (within this 7-minute period, a 1-minute physical exercise task also occurred)—all data were used in subsequent analyses. Roberts and Weerts (1982) also used imagery to study anger and fear. Self-report ratings were obtained following each trial, but again all data were used in subsequent analyses.

As pertains to verification, probably the best of the early studies of autonomic specificity was conducted by Funkenstein and colleagues (Funkenstein, King, & Drolette, 1954). Using criticism of performance on a math task to elicit anger-in, anger-out, and anxiety, they interviewed participants 10 minutes after the task to determine which emotions they experienced. Based on these interviews, 25% of the data were excluded. This procedure did have one questionable feature, however; they included in the analyses participants who experienced both anger and anxiety, which would seem to compromise their ability to distinguish between the two emotions.

Although verification sounds as if it should be useful, it is certainly reasonable to ask whether it makes any appreciable difference. There is not much published research that directly addresses this issue other than our work using the directed facial action task (Levenson et al., 1990). In this work, we used two kinds of verification: (1) facial measurement to determine if the targeted facial configurations were produced, and (2) self-report of emotional experience. To simplify presentation of these data, I use an index of the extent of specificity among negative emotions that we used in this research. This index is derived by computing "hit rates" (which represent whether an individual participant showed or did not show each of four differences between pairs of emotions found in group data—heart rate greater during anger than during disgust, heart rate greater during fear than during disgust, heart

rate greater during sadness than during disgust, finger temperature greater during anger than during fear).

In Figure 11.3 the hit rates are portrayed when the facial configurations in the comparisons are of low quality (i.e., they did not closely resemble the intended emotion prototype) versus high quality (i.e., they did closely resemble the intended emotion prototype). The hit rates (and thus the evidence of autonomic specificity) are significantly higher for data derived from the high-quality faces.

Figure 11.4 portrays hit rates when participants reported feeling the emotion associated with the facial configurations versus when they did not. Again, the hit rates are significantly higher for data derived when the targeted emotion is present.

These data, derived from one series of studies that used one eliciting task, are indicative of the potential clarity that can come from using reasonable verification procedures. Although we have not previously published these data, we found the same advantages when we used self-report verification criteria with the relived emotions task. Similar improvements over unverified data have been reported by others using verification procedures based on emotional facial behavior when examining EEG measures of regional brain activation obtained during a film eliciting task (Davidson et al., 1990)

Emotion Timing

There are several theoretical and methodological issues related to timing that are of particular importance for research on autonomic specificity. The first of these pertains to the time course of affective phenomena. Ekman (1984) has provided a useful discussion of this issue in which he arrays affective phenomena in terms of their increasing duration, starting with emotions, then moods, emotional traits, emotional plots, and, finally, emotional disorders. Emotions are the briefest of these phenomena, usually lasting only a matter of seconds. Compared with emotions, moods represent more enduring changes in affective tone, lasting from hours to days, with the stimulus conditions being less punctate and not always recognizable. Over the years, studies of autonomic correlations of affective states have addressed the entire range of affective phenomena. However, autonomic specificity, at least as it has been defined in the psychophysiological tradition, pertains to emotions and not to the longer affective phenomena. For that reason, it is important that attempts to aggregate data across multiple experiments do not mix apples and oranges (for example, comparing cardiovascular patterns during anger with those associated with being in an irritable mood or having a hostile personality).

The second issue is more practical, having to do with the temporal matching of autonomic measurement to the occurrence of emotion. As noted earlier, the autonomic nervous system is the slave to many masters, serving the needs of a host of bodily processes, including skeletal muscle demands, digestion, postural adjustments, thermoregulation, and so forth. According to the model I have proposed (Levenson, 1994, 1999), emotions such as anger, fear, sadness, and disgust briefly take the reins of the autonomic nervous system and alter its pattern of activation in service of behaviors that are likely to deal successfully with particular problems and challenges that face the organism. It is during this brief period in which the autonomic nervous system is under the control of emotion that we would expect to find autonomic specificity. Before the emotion asserts its influence and after it relinquishes control, autonomic activity will reflect other forces, and emotion-related patterning will not be found. In this regard, the autonomic nervous system is similar to other physiological systems that are taken over momentarily by emotion. For example, prior to the onset of emotion, the facial muscles may be serving a variety of functions, such as speech production and illustration; but in the throes of an emotion, a set of particular muscle actions can occur that signal the person's emotional state to others. After the emotion recedes, the facial muscle system, like the auto-

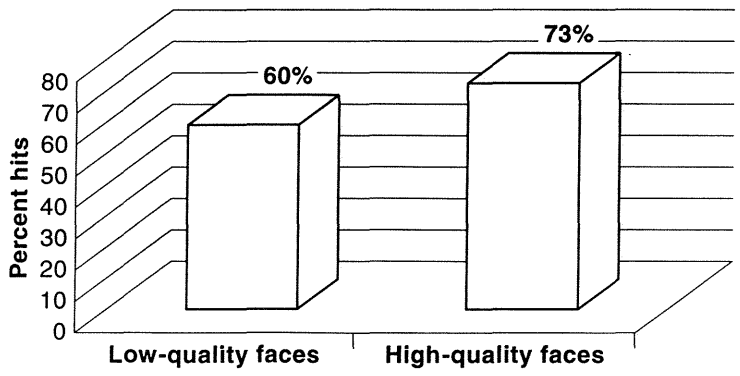

Figure 11.3 Impact of quality of facial configuration on extent of autonomic specificity.

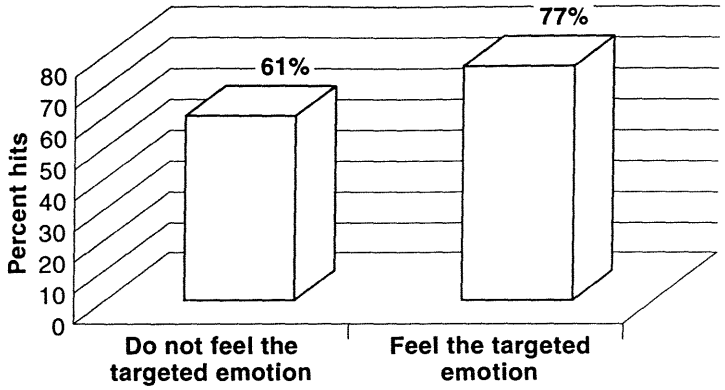

Figure 11.4 Impact of self-report of target emotion on extent of autonomic specificity.

nomic nervous system, goes back to its other activities with no residual sign that the emotion ever occurred.

In research using other physiological systems that show this kind of tight temporal linkage to the eliciting stimulus, great care is taken to ensure that measurements are time-locked to the stimulus onset. Thus, in measuring the electrocortical response evoked by a given stimulus, the EEG response is measured in a particular time window. Often with such systems, averaging over many stimulus presentations may be necessary to reveal the signal (e.g., a cortical event-related potential) against the background of noncontingent activity. In research on autonomic specificity, attempts to average across even two elicitations have been rare. Moreover, the temporal matching between measurement and emotion onset has often been lax, making it quite possible that the measured physiology has nothing to do with the elicited emotion.

Again, a look at the "classic" specificity literature will be illustrative. Ax (1953) induced fear and anger using complex manipulations (e.g., an incompetent experimenter) that stretched out over a 5-minute period. For each measured physiological function, the maximum and minimum levels reached during the 5-minute elicitation period plus the following 2 minutes were calculated and used to represent the targeted emotion. This is not an unreasonable approach, but it does have problems. For example, during the 7-minute period, one would expect a number of different emotions (targeted and nontargeted) to come and go. No attempt was made to match the autonomic responses to the occurrence of particular emotions. Nonetheless, the emotional induction and physiological measurement clearly had some amount of overlap.

In a number of other studies, the autonomic data that were analyzed were obtained *after* rather than *during* the emotion induction. Funkenstein and colleagues (Funkenstein et al., 1954) induced emotion by criticizing participants' performances, but all autonomic measures were obtained after the criticism was over. Similarly, Schwartz and colleagues (Schwartz et al., 1981) used imagery to induce emotion, but their autonomic data were obtained after the images ended. Stemmler (1989), in his "real life" inductions of fear, anger, and happiness, extracted physiological data during a 1-minute period following the end of the inductions.

In evaluating these studies, we unfortunately lack knowledge about the time course of any patterned autonomic response produced by an emotion. If this patterning continues long after the offset of the stimulus, then the reviewed studies were likely to be measuring the emotion-related autonomic response. If the patterning offsets rapidly, then they were likely measuring autonomic activity related to other, nonemotional activities of the organism.

The Future of Specificity Research

Specificity in the Autonomic Nervous System

Following the second flurry of research on this topic in the 1980s and early 1990s, activity in this area seems once again to have diminished. This is unfortunate for several reasons. First and foremost, it is my belief that despite more than 50 years of empirical work, we still do not have a definitive answer as to the extent of autonomic specificity in emotion. Despite the general belief in various cultures in autonomic specificity and a number of empirical findings that support its existence, there still does not exist a body of well-replicated, well-designed research that would settle this issue. Second, the extent of autonomic specificity is important to our understanding of the nature of emotion. As I pointed out earlier, autonomic specificity has profound implications for a number of theoretical formulations about the nature of emotion. Third, assuming that there is at least some degree of autonomic specificity for at least some emotions, then there are a host of interesting auxiliary issues that remain to be investigated.

Specificity for Positive Emotions

In research on autonomic specificity, the positive emotions have received far less attention than the negative

Often, when positive emotion has been studied, only a single positive emotion (e.g., happiness) has been included. In considering these issues a number of years ago (Levenson, 1988), I argued that the lack of association between positive emotions and behaviors that involve high-activity motor programs makes autonomic patterning for positive emotions less likely than for negative emotions. In that work, I proposed a model of positive emotions as efficient "undoers" of the autonomic activation provoked by negative emotion. Subsequently, Fredrickson and I were able to demonstrate that this was indeed the case for the positive emotions of amusement and contentment (Fredrickson & Levenson, 1998). Beyond this, the question of autonomic specificity for positive emotions remains unanswered. Subsequent work on this question should consider a broad range of positive emotions, including amusement and contentment, as well as calmness, excitement, joy, pride, awe, and love.

Temporal Organization and Interrelations of Emotion Response Systems

Many biologically oriented emotion researchers specialize in a single response system, such as visible facial expression, subvisible facial electromyographic activity, subjective emotional experience, acoustic properties of speech, autonomic nervous system activity, electrocortical or hemodynamic brain activity, or neurohormonal activity. What is needed is integrative research that advances our understanding of how these response systems are coordinated in emotion. Very basic questions, such as the temporal organization of facial response, autonomic response, electrocortical response, and subjective experience in emotion, remain largely unexamined. A related set of issues concerns the duration of autonomic specificity. Our work suggests that the activation of individual components of the autonomic responses can continue for some time (Fredrickson & Levenson, 1998), but we do not know how long the emotion-specific *pattern* is maintained.

Autonomic Concomitants of Blended and Sequential Emotions

In research on autonomic specificity, we often try to stimulate pure emotions—single emotions rather than a blend of emotions, and emotions that are isolated in time from other emotions. In this way, the autonomic activity associated with different emotions can be identified. In the natural world, however, emotions typically do not occur in such splendid isolation. Rather, they may occur in blends with other emotions or in sequences in which one emotion segues into another. Once we have identified emotions with different autonomic signatures, it will be important to determine what happens to this patterning when two emotions with different signatures combine or when they follow each other closely in time.

Alternative Motor Programs for Different Emotions

If the basis for autonomic specificity is a mapping between emotions and motor programs (with their need for a particular configuration of autonomic support), then the simplest model of specificity would be a single pattern of autonomic activity for each emotion. However, if emotions have multiple associated motor programs (e.g., "flight" and "freezing" in fear), each requiring a different configuration of autonomic support, this begs the question of whether, at least for *some* emotions, there are multiple associated autonomic patterns, depending on the particular motor program that is activated.

Generalizability Across Modes of Elicitation

The question of whether emotion-specific autonomic activity is consistent across modes of emotion elicitation has been nicely framed by Stemmler (1989), who argues for the importance of context in determining patterns of autonomic activity. In my review of four specific autonomic differences among negative emotions (Levenson, 1992), I found evidence for these same patterns in studies using quite different modes of elicitation. We (Levenson, Carstensen, Friesen, & Ekman, 1991) have also presented evidence in a single sample of participants exposed to multiple elicitors for this kind of generalizability across modes of elicitation for some autonomic differences among emotions. Generalizability to visual stimuli, such as films and slides, presents special challenges in this regard because there are strong autonomic correlates of orienting to the slides and films that can make it difficult to detect any emotion-specific autonomic activity. Clearly, this is an area in need of additional research.

Generalizability Across Sources of Individual Variation

In our work we have demonstrated generalizability of emotion-specific autonomic activity across gender (Levenson et al., 1990), young and old age (Levenson et al., 1991), and U.S. and West Sumatran cultures (Levenson et al., 1992). Clearly, this is just a beginning—there are many important issues pertaining to development, personality, and culture that remain to be explored.

Controlled Emotions

Emotions often occur in contexts in which the individual's learning history (e.g., cultural proscriptions) acts to limit free emotional expression. Such emotion control, whether it is conscious or unconscious, voluntary or involuntary,

inhibition or exaggeration, could alter the patterns of autonomic activity usually associated with that emotion. In our work on emotion suppression (Gross, 1998; Gross & Levenson, 1993, 1997), it appears that there may be a general pattern that is consistent across emotions when emotional behavior is voluntarily suppressed, but beyond that we know little about this issue.

Specificity in the Central Nervous System

In recent years, the spotlight in affective science has moved away from the autonomic nervous system and toward the brain. Localization of psychological functions in particular brain regions has always assumed an important role in brain research and continues to do so. New measurement methodologies offer the potential for studying activation sequences across different brain regions, which may go beyond mere localization and shed new light on the dynamics of the working brain.

Although human neuroscience maintains its longstanding interest in memory and other cognitive processes, there is clearly an explosion of interest in emotion and affective phenomena. At this juncture, most of this work has not been concerned with emotional specificity per se, perhaps reflecting the "traditional" undifferentiated model that places all emotional processing in the right hemisphere. However, a number of important challenges to this model now exist, especially models that situate certain classes of emotions (positive, approach-oriented) in left anterior brain regions (e.g., Davidson, 1993). To this point, however, most brain localization research has conceptualized emotion in terms of dimensions (positive-negative, approach-avoidance) rather than discrete emotions. Perhaps, with better experimental paradigms for eliciting discrete emotions repeatedly (or sustained over time), specificity of brain regions (and/or activation sequences) for some discrete emotions may be revealed. If not, we may be left with an interesting lack of parallelism, with emotional specificity in the central nervous system organized around dimensions and emotional specificity in the autonomic nervous system organized around discrete emotions.

Regardless of the ultimate organization, studies of specificity in the brain will have to address the same issues concerning emotion elicitation, verification, and temporal matching that beleaguer studies of autonomic specificity. As measures of brain functioning get more and more sophisticated and more precise in terms of temporal and spatial resolution, it may be tempting to become lax in the elicitation side of the research. However, the data derived from such studies will only be as good as their weakest link, and, as I hope this chapter has demonstrated, it is very easy to intend to study particular emotions but to end up missing the target. In fMRI studies, with participants lying on their backs in narrow tubes for hours on end, with their heads in vises, with loud hammering sounds in the background, and with major constraints on the kinds of emotional stimuli that can be used, the challenges for those wishing to conduct serious studies of the organization of brain function in emotion will be enormous.

NOTES

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1. In the earlier model, there was another subprogram for subjective emotional experience. In the ensuing years, I have come to view subjective emotional experience as deriving primarily from sensations associated with activation of the other response systems such as the face, physiology, vocal apparatus, and muscles. This sensory information can subsequently be integrated with appraisals of the environmental conditions when we engage in the act of labeling our emotional states.
2. In chapter 9 in this volume, Jänig discusses the function that the autonomic nervous system plays in protective reactions of the body during defensive behaviors such as flight, quiescence, and confrontation. Jänig suggests that the autonomic concomitants of these behaviors are integrated in the lateral and ventrolateral columns of the periaqueductal gray.
3. This "either/or" mentality is quite unfortunate. I believe that Schachter and Singer's model does in fact describe *one* way that emotion can be elicited. A system as important to our survival as emotion would likely be designed with multiple methods of activation. Thus, surely there are instances in which we find ourselves aroused for no immediately apparent reason and search for explanations as to why that might be. However, this is just one of a number of different ways in which emotions can be activated.

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