# 10 The Electrodermal System

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## THE ELECTRODERMAL SYSTEM

### Overview

Electrodermal activity (EDA), formerly called the Galvanic Skin Response (GSR), has been one of the most widely used response systems in the history of psychophysiology. Research involving EDA has been reported in practically all areas of psychology, psychiatry, and psychophysiology. EDA measures have been applied to a wide variety of topics ranging from basic research examining attention, information processing, and emotion, to more applied clinical research examining predictors and/or correlates of normal and abnormal behavior. The application of EDA measures to a wide variety of issues is due in large part to its low cost, relative ease of measurement and quantification, combined with its sensitivity to psychological states and processes.

The purpose of this chapter is to provide a tutorial overview of EDA for interested students, researchers, and practitioners who are not specialists in this particular system. We begin with a historical description of different methods of measuring EDA, and then discuss the physical, inferential, psychological, and social aspects of EDA.

### Historical Background and Measurement of EDA

***Exosomatic method.*** There are two fundamentally different methods of measuring EDA: (1) the "exosomatic method" which relies on the application of a small external electrical current across the skin and (2) the "endosomatic method" which measures internally generated electrical skin potentials without application of an external event. Both types of measure were first found to be sensitive to psychological changes in the later part of the nineteenth century.

The effect of psychological variables on the exosomatic measure of human EDA was demonstrated in the laboratory of Jean Charcot, the French neurologist famous for his work on hysteria and hypnosis (for interesting details see Neumann & Blanton, 1970; Bloch, 1993). Vigouroux (1879, 1888), a collaborator of Charcot, measured tonic skin resistance levels as a clinical diagnostic sign in patients with hysteria by passing a small electrical current across two electrodes placed on the surface of the skin. He reported that the skin resistance level changed from side to side of the body with changes in the side of the hysterical anesthesia. In the same laboratory, Féré (1888) found that skin resistance would momentarily decrease in response to a variety of discrete sensory stimuli (visual, auditory, gustatory, olfactory, etc.). The basic phenomenon discovered by Féré is that the skin momentarily becomes a better conductor of electricity when external stimuli are presented.

The electrical current used to measure exosomatic EDA can be either a direct current (DC) or an alternating current (AC). With DC one can measure either skin resistance when the current is kept constant or skin conductance when the voltage is kept constant. Skin conductance is much more commonly measured and is preferred for reasons to be described later under "Physical Recording Basis." AC is infrequently used and, although it may have some advantages, it is not recommended over DC (Boucsein et al., 2012).

***Endosomatic method.*** Shortly following the studies of Vigouroux and Féré, the Russian physiologist Tarchanoff (1890) reported that one could measure changes in electrical potential between two electrodes placed on the skin without applying an external current. Hence, Tarchanoff is said to have discovered the endosomatic method of recording EDA. However, the skin potential responses obtained with the endosomatic method are complex waveforms, often consisting of both positive and negative voltage changes. Due to this complexity, the scoring and interpretation of the skin potential response is difficult, and it is not widely used (Boucsein et al., 2012).

In summary, recording the skin resistance response (or its reciprocal, the skin conductance response) with the passage of an external DC across the skin is referred to as the *exosomatic* method, whereas recording the skin potential response does not involve an external current and

hence is referred to as the *endosomatic* method. The present chapter will focus on the DC exosomatic method of recording *skin conductance level* (*SCL*) and *skin conductance response* (*SCR*) because this clearly is the method of choice among contemporary researchers (Boucsein et al., 2012; Fowles et al., 1981). Additional information regarding the endosomatic method and/or the AC exosomatic method can be found in the comprehensive book on EDA by Boucsein (2012) and in the "Publication Recommendations for Electrodermal Measurements" written for the Society for Psychophysiological Research by Boucsein et al. (2012).

### Issues in the History of EDA Research

Several issues identified in this early research have been sources of considerable speculation and investigation throughout the history of research with this response system. One set of such issues concerns the mechanisms and functions of EDA. In terms of peripheral mechanisms, Vigouroux proposed what became known as the "vascular theory" of EDA (Neumann & Blanton, 1970). The vascular theory associated changes in skin resistance with changes in blood flow. Tarchanoff favored a "secretory theory," which related EDA to sweat gland activity. This theory was supported later by Darrow (1927), who measured EDA and sweat secretion simultaneously and found the two measures to be closely related, although the phasic SCR would begin about 1 sec before moisture would appear on the surface of the skin. Thus, it was concluded that activity of the sweat glands, not sweat on the skin per se, was critical for EDA. (Other lines of evidence indicating that sweat glands are the major contributors to EDA have been reviewed by Fowles, 1986, pp. 74–75.) It was generally known at the time that palmar sweat glands are innervated by the sympathetic chain of the autonomic nervous system, so EDA was said to reflect sympathetic activation. In terms of more central physiological mechanisms, work by early investigators such as Wang and Richter indicated that EDA was complexly determined by both subcortical and cortical areas (for a review of this early research, see Darrow, 1937). Darrow also proposed that "the function of the secretory activity of the palms is primarily to provide a pliable adhesive surface facilitating tactual acuity and grip on objects" (1937, p. 641).

Issues surrounding the proper methods of recording and quantifying EDA also have been important in the history of this response system. We would date the beginning of the modern era of EDA research to the early 1970s when Lykken and Venables proposed standardized techniques of recording skin conductance and standardized units of measurement. This was followed shortly by an edited book (Prokasy & Raskin, 1973) devoted entirely to EDA which contained several useful review chapters, including a particularly outstanding chapter by Venables and Christie (1973). Published around the same time were several other excellent reviews (Edelberg, 1972; Fowles, 1974; Grings, 1974). More recent reviews can be found in Boucsein (2012) and in Roy, Sequeira, and Delerm (1993), as well as in individual chapters by Andreassi (2007), Fowles (1986), Hugdahl (1995), and Stern, Ray, and Quigley (2001), and in Boucsein et al. (2012).

Another issue of central importance concerns the psychological significance of EDA. From the beginning, this response system has been closely linked with the psychological concepts of emotion, arousal, and attention. Carl Jung added EDA measurements to his word-association experiments in order to objectively measure the emotional aspects of "hidden complexes." An American colleague joined Jung in these experiments and enthusiastically reported that, "Every stimulus accompanied by an emotion produced a deviation of the galvanometer to a degree in direct proportion to the liveliness and actuality of the emotion aroused" (Peterson, 1907, cited by Neumann & Blanton, 1970, p. 470). About half a century later, when the concept of emotion was less in favor, Woodworth and Schlosberg (1954) devoted most of an entire chapter of their classic textbook in experimental psychology to EDA, which they described as "perhaps the most widely used index of activation" (p. 137).

Many of these issues have remained important for contemporary psychophysiologists and are discussed in the remainder of this chapter. In the next section we present a summary of the contemporary perspectives regarding the basic physiological mechanisms and proper recording techniques of EDA.

## PHYSICAL CONTEXT

### Anatomical and Physiological Basis

The skin is a protective barrier that aids in maintaining the body's water balance and constant core body temperature, functions accomplished primarily through vasoconstriction/dilation and through variation in the production of sweat. There are two forms of sweat glands in the human body: the apocrine, which have been less studied, and the eccrine, which have been of primary interest to psychophysiologists. The primary function of most eccrine sweat glands is thermoregulation. However, those located on the palmar and plantar surfaces are thought to be more related to grasping behavior than to evaporative cooling (Edelberg, 1972) and are more responsive to psychologically significant stimuli than to thermal stimuli. Although all eccrine glands are believed to be involved in psychological sweating, such sweating is usually most evident in these areas primarily because of the high gland density (Shields, MacDowell, Fairchild, & Campbell et al., 1987).

Figure 10.1 shows the basic peripheral mechanisms involved in the production of EDA. The extreme outer layer of the skin, the stratum corneum or horny layer, consists of a layer of dead cells that serves to protect the internal organs. Below the stratum corneum lies the stratum lucidum, and just below that is the stratum Malpighii.

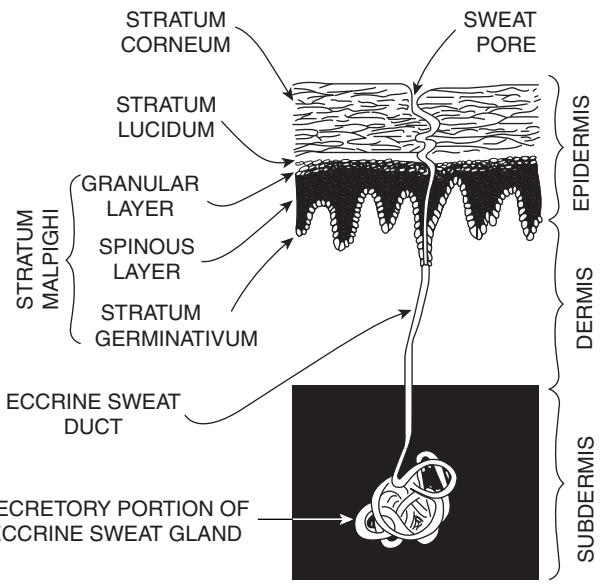


**Figure 10.1** Anatomy of the eccrine sweat gland in various layers of skin. (Adapted from Hassett, 1978.)

The eccrine sweat gland itself consists of a coiled compact body that is the secretory portion of the gland, and the sweat duct, the long tube which is the excretory portion of the gland. The sweat duct remains relatively straight in its path through the stratum Malpighii and stratum lucidum; it then spirals through the stratum corneum and opens on the surface of the skin as a small pore (Edelberg, 1972).

Many models have been suggested to explain how these peripheral mechanisms relate to the electrical activity of the skin and to the transient increases in skin conductance elicited by stimuli. Edelberg (1993) concluded that one can account for the variety of electrodermal phenomena, including changes in tonic SCL and phasic SCR amplitude, with a model based entirely on the sweat glands.

To understand how electrodermal activity is related to the sweat glands, it is useful to think of the sweat ducts (the long tubular portion of the gland that opens onto the skin surface) as a set of variable resistors wired in parallel. Columns of sweat will rise in the ducts in varying amounts and in varying numbers of sweat glands, depending on the degree of activation of the sympathetic nervous system. As sweat fills the ducts, there is a more conductive path through the relatively resistant corneum. The higher the sweat rises, the lower the resistance in that variable resistor. Changes in the level of sweat in the ducts change the values of the variable resistors and yield observable changes in EDA.

Human sweat glands have predominantly sympathetic cholinergic innervation from postganglionic fibers originating in the sympathetic chain (Shields et al., 1987). Convincing evidence for the sympathetic control of EDA has been provided by studies that measured sympathetic action potentials in peripheral nerves while simultaneously recording EDA. The results have shown that within normal ranges of ambient room temperature and thermoregulatory states, there is a high correlation between bursts of sympathetic nerve activity and SCRs (Wallin, 1981). Other evidence that SCRs reflect sympathetic nerve activity is reviewed by Bach (2014).

Excitatory and inhibitory influences on the sympathetic nervous system are distributed throughout the brain and therefore the neural mechanisms and pathways involved in the central control of EDA are numerous and complex. Boucsein (2012, pp. 32–42) followed the suggestions of Edelberg (1972) in describing at least two and possibly three relatively independent pathways that lead to the production of SCRs (see Figure 10.2). The *first* and highest level of central EDA control involves contralateral cortical and basal ganglion influences (Sequeira & Roy, 1993). One cortical pathway involves excitatory control by the premotor cortex (Brodmann area 6) descending through the pyramidal tract, and another involves both excitatory and inhibitory influences originating in the frontal cortex. The *second* level of EDA control involves ipsilateral influences from the hypothalamus and limbic system. There is considerable evidence of an excitatory hypothalamic descending control of EDA. Limbic influences are complicated, but there is evidence of excitatory influences from the amygdala and inhibitory effects originating from the hippocampus. The *third* and lowest level mechanism is in the reticular formation in the brainstem. Activation of the reticular formation by direct electrical stimulation or sensory stimulation evokes skin potential responses in cats, and presumably skin conductance responses in humans. An inhibitory EDA system has also been located in the bulbar level of the reticular formation.

Much of the evidence regarding the central pathways that control EDA described above was derived from animal studies, usually cats (e.g., Wang, 1964; Roy et al., 1993). However, knowledge of the central control of human EDA, particularly EDA associated with attention and emotional processes, has increased greatly in recent years. Three strategies have been used to investigate the neural substrates of EDA: (1) examination of EDA in patients with focal brain lesions, (2) stimulation of specific brain areas in human patients, and (3) examination of the relationship between patterns of brain activation and simultaneously recorded EDA.

In patients with brain damage, the brain areas found to be involved in the generation of EDA vary with the specific task or stimuli used to elicit EDA (Tranel, 2000). For example, patients with lesions in the lateral prefrontal cortex and anterior cingulate cortex (ACC) exhibit fewer SCRs to significant stimuli (reaction time cues) but not to simple non-significant tones (Zahn, Grafman, & Tranel, 1999). On the other hand there have been reports of consistent cerebral regions correlated with SCRs across different tasks: specifically, a gambling task, a working memory task, and a resting state (Patterson, Ungerleider, & Bandettini, 2002). The brain regions correlated with

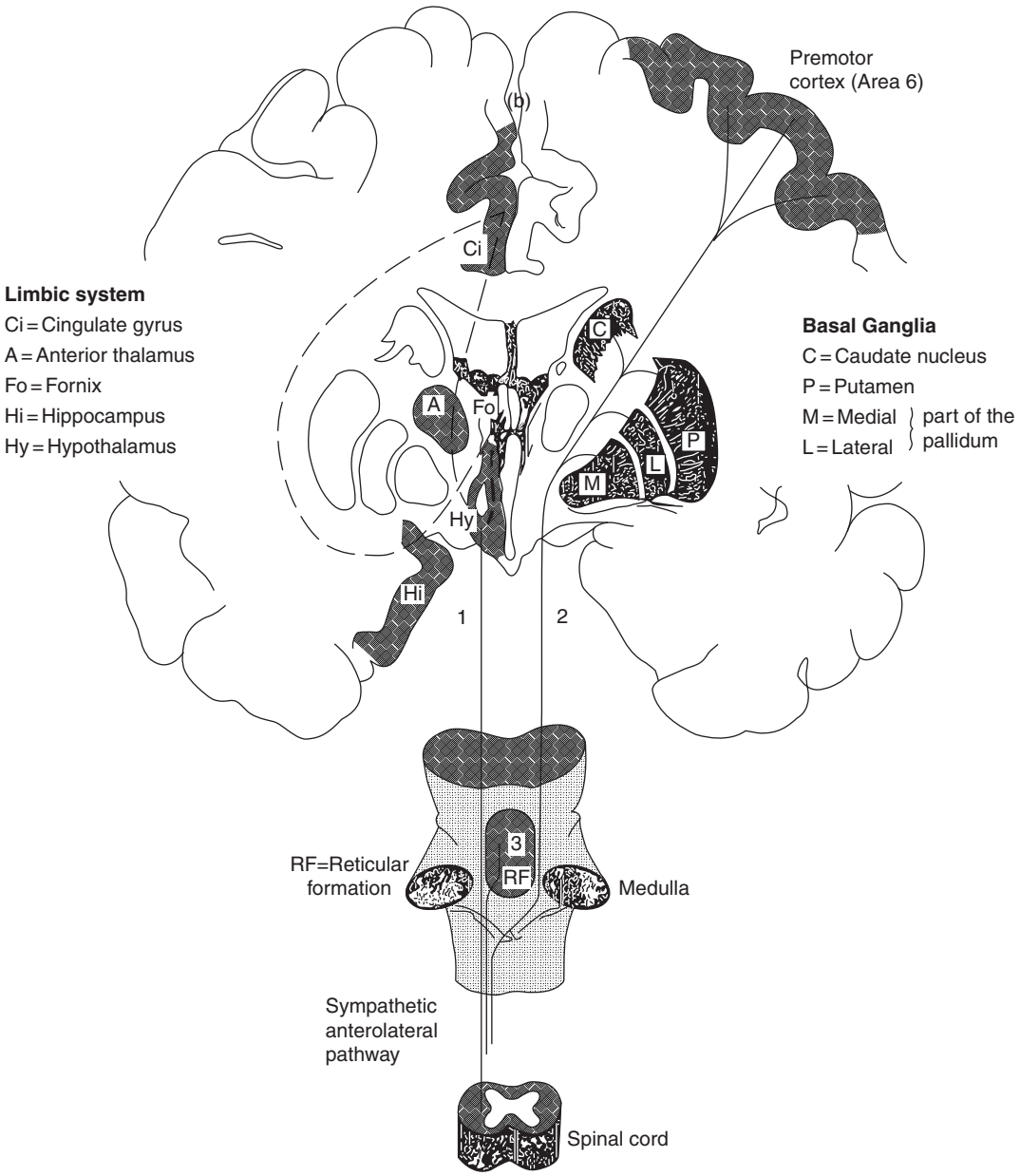


**Figure 10.2** Central nervous system determiners of EDA in humans. (From Boucsein, 2012.)

SCRs across tasks included the ventromedial prefrontal cortex (VMPFC), left inferior parietal cortex, cingulomotor cortex, posterior cingulate cortex, early visual areas, right cerebellum, and thalamus.

One of the tasks most commonly used to investigate the relationship between EDA and the central nervous system (CNS) has been fear classical conditioning, involving anticipation of aversive unconditioned stimuli. The amygdala has been consistently found to play an essential role in fear conditioning in both humans and lower animals. Several human studies have reported amygdala activation to occur concurrently with the SCR conditioned response (e.g., Cheng, Richards, & Helmstetter, 2007; Morris, Buchel, & Dolan, 2001). In fact, the size of the conditioned SCR has been found to be significantly correlated with the degree of activation of the amygdala, particularly during early learning trials and with the right amygdala (e.g., LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998; Phelps, Delgado, Nearing, & LeDoux, 2004). Moreover, Cheng, Knight, Smith and Helmstetter (2006) divided trials on which participants demonstrated a conditioned SCR from trials on which no SCR was apparent and found significant amygdala activity only on trials when the reinforced stimulus elicited an SCR.

One limitation of simultaneous recordings of CNS responses and SCRs is that it is not possible to draw a direct causal relationship between the brain activation and the EDA. It would be uncertain for instance whether

the appearance of amygdala activation and the SCR in fear conditioning reflects a causal relationship or parallel aspects of the fear response. Consistent with a causal effect, Gentil, Eskendar, Marci, Evans, and Dougherty (2009) found that stimulation of the transition region between the rostral and dorsal ACC produced reliable SCRs in a group of patients who were to undergo bilateral anterior cingulatomy. Also, Mangina and Beuzeron-Mangina (1996) studied a group of five patients with implanted electrodes for the treatment of intractable epilepsy. They found that direct electrical stimulation of the human amygdala, as well as the anterior and posterior hippocampus and the ACC, produced large SCRs on the ipilateral side of the stimulation with only very little contralateral effects. The authors conclude that EDA is ipsilaterally controlled by limbic structures. These brain stimulation studies strongly imply a causal relationship between activation of limbic areas and the production of SCRs.

Studies of brain-damaged patients also indicate that the amygdala is critical for SCR classical conditioning. For example, Bechara et al. (1995) found that a patient with selective bilateral destruction of the amygdala did show normal unconditioned SCRs to an aversive unconditioned stimulus but failed to show conditioned SCRs to the CS, although this patient was aware of the CS–UCS relation.

In addition to fear conditioning, brain activity has been measured simultaneously with EDA in a number of other types of tasks, including gambling tasks, aversive stimuli, significant stimuli, and during rest. For instance, SCRs and brain activity were measured concurrently to unlearned inherently aversive pain stimuli (Dubé et al., 2009). Dubé et al. found that subjects showing large SCRs elicited by thermal pain specifically displayed larger neural responses in the classic pain network including the ACC and thalamus, and also the left amygdala and hypothalamus.

Another type of task that has commonly been used to relate EDA with brain activity is a gambling task in which participants are required to choose from among options that vary in terms of their short-term and long-term rewards and punishments. As discussed later in this chapter, research has shown a relationship between the SCRs elicited during the decision-making phase of these tasks and the subsequent decisions made. In general, larger SCRs are shown prior to risky or bad decisions (e.g., Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Damasio, Damasio, & Lee, 1999). In terms of linking brain function with EDA, Bechara, Damasio, Damasio, and Anderson (1994) found that patients with bilateral damage to either the VMPFC or the amygdala failed to show normal SCRs in anticipation of a risky decision and they failed to learn to avoid options with poorer outcomes. Interestingly, patients with VMPFC damage generated SCRs when they received rewards and punishments (play money), whereas patients with amygdala damage failed to do so.

Critchley, Elliot, Mathias, and Dolan (2000) and Critchley, Mathias, and Dolan (2001) examined brain activity and EDA while healthy subjects performed somewhat similar gambling tasks. During an anticipatory period while subjects waited to see if they had won or lost money, activation of the ACC, areas of the frontal cortex, and other areas was correlated with generation of SCRs (for reviews of these and related studies see Critchley, 2002, 2009). As noted above, Patterson et al. (2002) also used a gambling task and found activity in the right orbitofrontal cortex as well as the VMPFC and posterior cingulate cortex to be positively correlated with SCRs. Another task, a go/no-go reaction time task, also produced correlations between ACC activation and heightened SCRs (Zhang et al., 2012). Broadly speaking, these results indicate that SCRs are elicited when brain areas are involved in an effortful task or activity. Moreover, Fan et al. (2012) found that spontaneous SCRs during rest were correlated with activity in the ACC.

Although there is not perfect overlap in the brain areas implicated across these studies, some consistent patterns have emerged. For example, activation of brain areas involved in evaluating stimulus significance/salience, particularly the VMPFC, right inferior parietal region, and ACC, have been found to be associated with elicitation of SCRs. In addition, when the stimulus has emotional significance, the amygdala and orbitofrontal cortex, in addition to the areas mentioned above, are also involved. Thermoregulatory sweating is controlled by the hypothalamus, which also integrates patterns of sympathetic activity in emotion, in conjunction with limbic structures.

## Physical Recording Basis

As briefly described earlier, exosomatic EDA is measured by passing a small current through a pair of electrodes placed on the surface of the skin. The principle invoked in the measurement of skin resistance or conductance is that of Ohm's Law, which states that skin resistance (R) is equal to the voltage (V) applied between two electrodes placed on the skin surface, divided by the current (I) being passed through the skin. This law can be expressed as R = V/I. If the current is held constant then one can measure the voltage between the electrodes, which will vary directly with *skin resistance*. Alternatively, if the voltage is held constant, then one can measure the current flow, which will vary directly with the reciprocal of skin resistance, *skin conductance*. Conductance is expressed in units of Siemens and measures of skin conductance are expressed in units of microSiemens (μS).

Lykken and Venables (1971) argued strongly for the direct measurement of skin conductance with a constant voltage system rather than measuring skin resistance with a constant current system, because skin conductance is linearly related to the rate of secretion of sweat. A description of constant voltage circuits that allow the direct measurement of skin conductance can be found in Lykken and

Venables as well as in Fowles et al. (1981) and Boucsein (2012), and most of the physiological recording systems currently on the market include constant voltage systems for the direct recording of skin conductance.

### EDA Recording Systems

Older recording systems, in operation ten or more years ago, output EDA to a paper record in analogue form. Most recording systems today are computer-based systems in which the analogue skin conductance signal is digitized and stored on a computer. With such systems, a researcher must select which time points the computer will sample from the continuous EDA waveform. Historically, this sampling window has been a few seconds following each presentation of an experimental stimulus. In these cases, EDA at all other time points is lost. Fortunately, with expanding computing capability, it is now generally feasible to sample EDA continuously, to allow an experimenter to flag critical events with a keypress or programmed signal, and to store a complete and continuous record of a participant's EDA within an experimental session. In choosing an EDA recording system one must consider computing capabilities and software issues. For example, some manufacturers offer software packages for the acquisition of EDA, some offer software for the quantification of EDA, and some offer both.

In addition to selecting an EDA recording system, special consideration must be given to the choice of recording electrodes, electrode paste, electrode placement, and general environmental considerations. Silver–silver chloride cup electrodes are the type most typically used in skin conductance recording because they minimize the development of bias potentials and polarization. With DC recording, polarization of electrodes can occur. In order to reduce the chance of polarization it is recommended that a polarity reversal switch be used that reverses the way the electrodes are plugged into the recording circuitry (Boucsein et al., 2012). Polarity can then be reversed between segments of an experiment and between subjects. Boucsein et al. (2012) provide detailed discussion of the bias potential issue, including description of a simple Bias Voltage Test that researchers can use to check their electrodes for polarization. The electrodes can be easily attached to the recording site through the use of double-sided adhesive collars which also serve the purpose of helping to control the size of the skin area that comes in contact with the electrode paste, an important parameter because it is the contact area, not the size of the electrode, that affects the conductance values.

The electrode paste is the conductive medium between the electrodes and the skin. Probably the most important concern in choosing an electrode paste is that it preserve the electrical properties of the response system of interest. Since the measurement of EDA involves a small current passed through the skin, the electrode paste interacts with the tissue over which it is placed. For this reason, the use of a paste which closely resembles sweat in its salinity is recommended (isotonic paste or gel; Venables & Christie, 1980). Instructions for making such paste are given in Fowles et al. (1981) and Grey and Smith (1984). Satisfactory paste is also available commercially. Commercial EKG or EEG gels should not be used because they usually contain near saturation levels of NaCl and have been shown to significantly inflate measures of skin conductance level (Grey & Smith 1984).

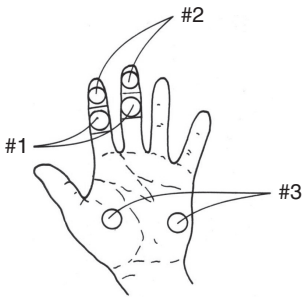


**Figure 10.3** Three electrode placements for recording electrodermal activity. Placement #1 involves volar surfaces on medial phalanges, placement #2 involves volar surfaces of distal phalanges, and placement #3 involves thenar and hypothenar eminences of palms.

Skin conductance is recorded using two electrodes, both placed on active sites (bipolar recording); hence it does not matter in which direction the current flows between the two electrodes. Skin conductance recordings are typically taken from locations on the palms of the hands, with several acceptable placements. The most common electrode placements are the thenar eminences of the palms, and the volar surface of the medial or distal phalanges of the fingers (see Figure 10.3). It should be noted that although electrodermal activity can be measured from any of these sites, the values obtained are not necessarily equal. Scerbo, Freedman, Raine, Dawson, and Venables (1992) made a direct comparison of EDA recorded from the distal and medial phalange sites simultaneously and found that both the elicited SCR amplitude and SCL were significantly higher from the distal recording site. The greater level of reactivity at the distal site was found to be directly related to a larger number of active sweat glands at that location (Freedman et al., 1994). Therefore, the distal phalange site is recommended unless there are specific reasons for not using the distal site (e.g., recording from children whose fingertips may be too small for stable electrode attachment, presence of cuts or heavy calluses on the fingertips, etc.).

Another recording issue concerns the hand from which to record. Many laboratories use the non-dominant hand because it is less likely to have cuts or calluses, and because it leaves the dominant hand free to perform a manual task. However, this begs the question of whether there are significant laterality differences in EDA. Although differences between left and right hand EDA recordings have been reported, the differences reported

across studies are often in opposite directions and the interpretations have been ambiguous (see reviews of early literature by Hugdahl, 1984). The aforementioned physiological model depicted in Figure 10.2 clearly predicts that emotional tasks involving the limbic structures would have ipsilateral effects on EDA whereas non-emotional motor tasks would have contralateral EDA effects. However, results of testing these differences have been complex and partly controversial (see Boucsein, 2012, p. 340). The simultaneous EDA recordings from the two hands are generally highly correlated, but there are usually small and unreliable differences between EDA recorded from the two hands, and occasionally large differences. Moreover, most of the evidence linking EDA asymmetries to specific psychological processes is inconclusive but this is an area that warrants further investigation.

In recent years, increasing interest in the possibility of recording EDA from subjects engaged in tasks requiring hand movements or who are completely ambulatory has led to the investigation of alternate sites for recording EDA other than the hands. Numerous anatomical sites were examined by Edelberg (1967), who reported good electrodermal reactivity from the medial side of the plantar surface of the foot over the abductor halluces muscle, as did van Dooren, de Vries, and Janssen (2012), Kappeler-Setz, Grobbenhorst, Scumm, Arnrich, and Tröster (2013), and Payne, Dawson, Schell, Singh, and Courtney (2013), although the latter found that the foot placement produced significantly fewer and smaller SCRs than did the distal phalanges of the fingers. Payne, Schell, and Dawson (2016) compared different foot sites and found that the toes were generally most comparable to the fingers in EDA, making them perhaps the best alternate site in the laboratory if the hands are not available. Surprisingly, it may be feasible to use a foot as well as the fingers for ambulatory recording. Kappeler-Setz et al. recorded EDA from both fingers and the foot from one subject while the subject was walking and reported what appeared to be strong point-by-point agreement of SCL across fingers and foot. However, artifacts due to changes in electrode contact pressure were evident in the foot electrode placement that would have made measurement of event-related SCRs very difficult.

With the emergence of wireless SC recording technologies that make ambulatory recording feasible, recording from the wrist has received considerable interest (Fletcher et al., 2010; Picard & Healey, 1997; Poh, Swenson, & Picard, 2010; Sano, Picard, & Stickgold, 2014). Using wrist-worn watch-like wireless devices with dry electrodes (without electrode paste), Poh et al. (2010) and Fletcher et al. (2010) reported the wrist produced generally small skin conductance responses but with strong within-subject correlations with responses recorded from the medial phalanges of the fingers, often on the order of 0.90. However, van Dooren et al. (2012) evaluated the wrist using a traditional laboratory-based recording apparatus and techniques, and found low SCLs and small SCRs at the wrist, as well as lower correlations between the wrist and the fingers (mean of 0.55). Payne et al. (2016) also used standard recording techniques in the laboratory and found the wrist to be much lower in SCL and SCRs than the fingers or foot sites, frequently failing to show a response to stimuli when the fingers did respond. Van Dooren et al. and Payne et al. calculated correlations across subjects, and found that individuals with high finger SCL and SCRs did not particularly show high wrist SCL or SCRs, while Poh et al. and Fletcher et al. calculated a point-by-point correlation for each subject across an entire recording period and found that for most individuals, when finger SCL increased, wrist SCL also tended to increase. However, Payne et al. also examined within-subject point-by-point correlations and found that the correlation between finger and wrist SCL averaged only 0.35. Further research is needed to determine when the wrist and other anatomical sites may be viable alternatives to the traditional palmar sites.

Another issue with respect to the wrist concerns whether the sweat glands at that site are primarily responsive to psychological stimuli as opposed to being thermoregulatory. The Society for Psychophysiological Research Publication recommendations for electrodermal measurements (Boucsein et al., 2012) cautioned that the wrist should be avoided because the sweat glands there may be primarily thermoregulatory in their functioning, as distinguished from sweat glands on the hands and feet that may be largely activated by emotional arousal. However, Picard, Fedor, and Ayzenberg (2015) cited evidence that changes in EDA recorded from the wrist may be evidenced in situations that elicit very strong activity (e.g., during and after epileptic seizures, during emotional arousal of autistic children, and during sympathetic storms during sleep).

Despite precautions about recording EDA from the wrist, there is a high degree of interest in recording ambulatory EDA in daily-life situations (see Chapter 14, this volume). One interesting example of recording ambulatory EDA from the fingers rather than the wrist in a life-situation was reported by Wilhelm and Roth (1998) and Wilhelm, Pflatz, Grossman, and Roth (2006). They recorded autonomic activity from participants with and without flying phobia while sitting during a short commercial flight. Measures of EDA were greater during the flight than at rest for all participants, and were greater during the flight among the flight phobics than among non-phobics. Schumm et al. (2008) also recorded ambulatory EDA from the fingers using a wearable recording device while subjects were walking on a treadmill. They found that the faster the subject walked, with associated arm movements, the more the number of "spontaneous" or "non-specific" SCRs (NS-SCRs) increased, making the detection of stimulus elicited SCRs more difficult. A somewhat different approach to ambulatory monitoring of electrodermal activity from the hand was reported by Tartz, Bartak, King, and Fowles (2015). These investigators developed a hand-held device the approximate size of a cell phone

which recorded EDA from dry electrodes touching the skin of the palm of the hand, at the thenar eminence and the tip of the middle finger. They found that, although there was some attenuation of SCRs measured by the device, only 15–20 percent of SCRs were missed when the grip force on the device was light to medium, compared to EDA simultaneously recorded from the distal phalanges of the opposite hand. Potentially, a wireless version of such a device could integrate electrodes directly into the housing of a smartphone or some other handheld device and be used for ambulatory measurement of EDA.

Because it is critical in exosomatic EDA recording that the electrical properties of the response system be preserved, the electrode sites should not receive any special preparation such as cleaning with alcohol or abrasion, which might reduce the natural resistive/conductive properties of the skin. However, since a fall in conductance has been noted following the use of soap and water (Venables & Christie, 1973), and since the length of time since the last wash will be variable across subjects when they arrive at the laboratory, these authors recommended that subjects wash their hands with a non-abrasive soap or just lukewarm water prior to having the electrodes attached and that the skin be kept clean and dry.

No matter what anatomical site is used, the standard methods of EDA measurement require that the recording sensors be in contact with the skin. However, interesting non-contact techniques have also been recently investigated. For example, Krzywicki, Berntson, and O'Kane (2014) measured eccrine sweat gland activity from the face and fingers of 20 participants with high resolution thermal imaging sensors that were placed 11.5 cm from the fingers and over 2 m from the face. This technique relies on the fact that pores with active sweat appear as localized areas of lower temperature. Phasic activity of the sweat pores of the finger tips was found to be positively correlated with the amplitude of SCRs recorded from the opposite hand (mean = 0.71). Although this non-contact thermal imaging technique is in the early stages of development, it illustrates the application of new technologies to the measurement of EDA.

Ambient temperature and time of day are two environmental factors that should be controlled (e.g., Hot, Naveteur, Leconte, & Sequeira, 1999; Venables & Mitchell, 1996). Because EDA is influenced by hydration of the corneum, SCL tends to rise with increases in ambient temperature in the normal room temperature range (see Boucsein et al., 2012, for an extended discussion of the corneum hydration issue). Boucsein (2012) recommends a room temperature of 23 degrees C. Likewise, room humidity should be kept as constant as possible. Because diurnal effects may influence EDA, this variable also should be controlled across experimental conditions. It is worth noting that these variables need to be taken into account when interpreting ambulatory recordings where environmental factors are likely to change.

## INFERENTIAL CONTEXT

### Quantification Procedures

Figure 10.4 shows tracings of two hypothetical skin conductance recordings during a 20 sec rest period followed by three presentations of a simple discrete stimulus (e.g., a mild tone). Several important aspects of EDA can be seen in Figure 10.4. First, it can be seen that tonic SCL begins at 10 µS in the upper tracing and at 5 µS in the lower tracing. While tonic SCL can vary widely between different subjects and within the same subject in different psychological states, the typical range is between 2 µS and 20 µS with the types of apparatus and procedures described here. Computing the log of SCL can significantly reduce skew and kurtosis in the SCL data and is recommended by Venables and Christie (1980).

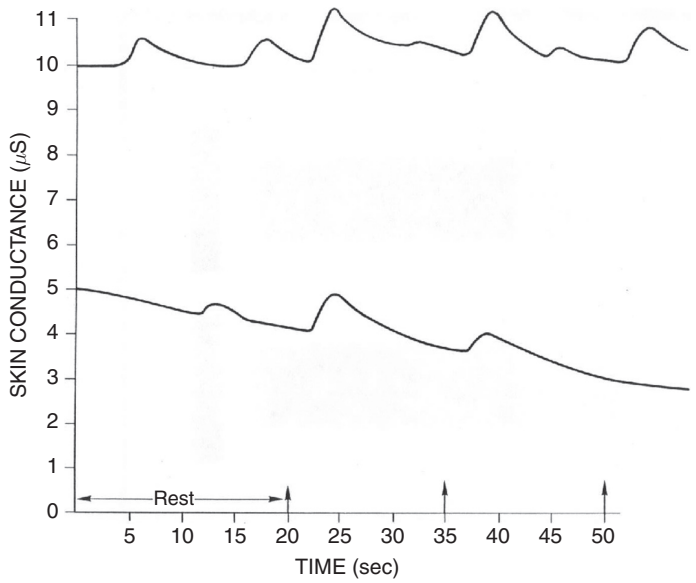


**Figure 10.4** Two hypothetical skin conductance recordings during 20 sec of rest followed by three repetitions of a simple discrete stimulus. Arrows represent the presentation of a stimulus. (From Dawson & Nuechterlein, 1984.)

**Table 10.1** Electrodermal measures, definitions, and typical values

| Measure | Definition | Typical values |
|---|---|---|
| Skin conductance level (SCL) | Tonic level of electrical conductivity of skin | 2–20 μS |
| Change in SCL | Gradual changes in SCL measured at two or more points in time | 1–3 μS |
| Frequency of NS-SCRs | Number of SCRs in absence of identifiable eliciting stimulus | 1–3 per min |
| SCR amplitude | Phasic increase in conductance shortly following stimulus onset | 0.2–1.0 μS |
| SCR latency | Temporal interval between stimulus onset and SCR initiation | 1–3 s |
| SCR rise time | Temporal interval between SCR initiation and SCR peak | 1–3 s |
| SCR half recovery time | Temporal interval between SCR peak and point of 50% recovery of SCR amplitude | 2–10 s |
| SCR habituation (trials to habituation) | Number of stimulus presentations before two or three trials with no response | 2–8 stimulus presentations |
| SCR habituation (slope) | Rate of change of ER–SCR amplitude | 0.01–0.5 μS per trial |

*Key*: SCL: skin conductance level; SCR: skin conductance response; NS-SCR: non-specific skin conductance response.

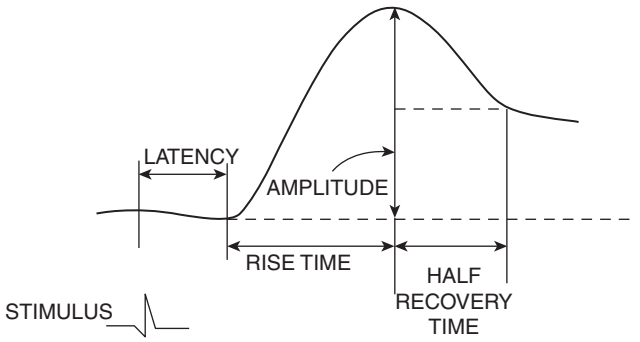


**Figure 10.5** Graphical representation of principal EDA components.

It can also be seen in the lower tracing of Figure 10.4 that the SCL drifts downward from 5 μS to nearly 4 μS during the rest period. It is common for SCL to gradually decrease while subjects are at rest, rapidly increase when novel stimulation is introduced, and then gradually decrease again after the stimulus is repeated.

Phasic SCRs are only a small fraction of the SCL and have been likened to small waves superimposed on the tidal drifts in SCL (Lykken & Venables, 1971). If the SCR occurs in the absence of an identifiable stimulus, as shown during the rest phase of Figure 10.4, it is referred to as a "spontaneous" or "non-specific" SCR (NS-SCR). The most widely used measure of NS-SCR activity is their rate per minute, which typically is between 1 and 3 per min while the subject is at rest. However, responses can be elicited by deep breaths and bodily movements, so unless these also are recorded, it is impossible to say which responses are truly NS-SCRs.

Presentation of a novel, unexpected, significant, or aversive stimulus will likely elicit an SCR referred to as a "specific" SCR. With the exception of responses elicited by aversive stimuli, these SCRs are generally considered components of the orienting response (OR). As is also the case with NS-SCRs, one must decide on a minimum amplitude change in conductance to count as an elicited SCR. Minimum values between 0.01 and 0.05 μS are generally used. Another decision regarding scoring of specific SCRs concerns the latency window during which time a response will be assumed to be elicited by the stimulus. Based on frequency distributions of response latencies to simple stimuli, it is common to use a 1–3 sec or 1–4 sec latency window. Hence, any SCR that begins between 1 and 3, or between 1 and 4 sec, following stimulus onset is considered to be elicited by that stimulus. It is important to select reasonably short latency windows so as to reduce the likelihood that NS-SCRs will be counted as elicited SCRs.

Having decided on a minimum response amplitude and a latency window in which a response will be considered a specific stimulus-elicited SCR, one can measure several aspects of the elicited SCR besides its mere occurrence and frequency. Definitions and typical values of the major EDA component measures are given in Table 10.1 and shown graphically in Figure 10.5. The measure that is most commonly used is the amplitude of the SCR, which is quantified as the amount of increase in conductance measured from the onset of the response to its peak as shown in Figure 10.5. The size of an elicited SCR typically ranges between 0.1 and 1.0 μS. The values in Table 10.1 are representative of healthy young adults. Readers interested in the effects of individual differences in age, gender, and ethnicity should consult Boucsein (2012). Although effects of these variables on EDA have been documented and linked to differences in skin physiology, the effects appear to interact with the nature of the eliciting stimuli (e.g.,

emotional or neutral), recording environment (e.g., season, time of day, etc.), and recording methodology (constant current or constant voltage) (Boucsein, 2012; Venables & Mitchell, 1996). In general, we advise that these individual differences be controlled across experimental conditions.

When a stimulus is repeated several times and an average size of the SCR is to be calculated, one may choose to compute mean SCR amplitude or magnitude. *Magnitude* refers to the mean value computed across all stimulus presentations including those without a measurable response, whereas *amplitude* is the mean value computed across only those trials on which a measurable (non-zero) response occurred. The magnitude measure is the most commonly used but Prokasy and Kumpfer (1973) noted that it confounds frequency and amplitude, which do not always covary. A magnitude measure can create the impression that the response size is changing when, in fact, it is response frequency that is changing. Hence, these authors recommend separate assessments of frequency and amplitude rather than magnitude. However, it is important to note that a complication with the amplitude measure is that the *N* used in computing average response size can vary depending on how many measurable responses a subject gives, and the data of subjects without any measurable response must be eliminated. Thus, a subject who responds on each of ten stimulus presentations with a response of 0.50 μS will have the same mean SCR amplitude as a subject who responds on only the first stimulus presentation with a response of 0.50 μS, and does not respond thereafter. We concur with Venables and Christie (1980) that there are arguments for and against both amplitude and magnitude and that although no absolute resolution is possible, it is important to keep the difference between the two measures clearly in mind. In some situations it may be reasonable to compute and compare results obtained with SCR frequency, amplitude, and magnitude.

Like SCL, SCR amplitude and magnitude are frequently found to be positively skewed and also leptokurtotic, so a logarithmic transformation can be used to remedy these problems. If measurements are being made of SCR magnitude, so that zero responses are included, then log of (SCR + 1.0) may be calculated, since the logarithm of zero is not defined (Venables & Christie, 1980). Another common practice is to use a square root transformation, $\sqrt{SCR}$, to normalize response amplitude data; this does not require the addition of a constant. In some cases the choice of the square root or logarithmic transformation should be guided by considerations of achieving or maintaining the homogeneity of variance across several groups (Ferguson & Takane, 1989). If skew, kurtosis, or homogeneity of variance problems do not exist in a particular set of data, no transformations need be performed.

In addition to response size, one can also measure temporal characteristics of the SCR including onset latency, rise time, and half recovery time. These temporal characteristics of the SCR waveform are not as commonly reported as magnitude, and their relationship to psychophysiological processes is not as well understood at this time. The possibility that SCR recovery time, for example, can provide information independent of other EDA measures and is uniquely responsive to specific psychophysiological processes remains unsettled (Fowles, 1986, pp. 84–87; Edelberg, 1993, pp. 14–15). This is not to say that SCR recovery time is without discriminating power; rather, only that its qualitatively different informational properties relative to other EDA components are an open issue.

The usual constellation of EDA components is for high SCL, frequent NS-SCRs, large SCR amplitude, short latency, short rise time, and short recovery time to cluster together. However, the correlations among the EDA components generally are not very high, usually less than 0.60 (Venables & Christie 1980; Schell, Dawson, & Filion 1988). The size and consistency of these relationships are compatible with the hypothesis that many of the EDA components may represent partially independent sources of information although, as indicated above with SCR recovery time, this is an unsettled hypothesis. The one exception to the modest relationships among EDA components is the consistently high correlation between SCR rise time and recovery time. Based on this relationship, Venables and Christie (1980) suggest that SCR rise time and half recovery time may be essentially redundant measures and that, since recovery time is not always as available as rise time (due to subsequent responses), rise time may be the preferred measure.

A problem with quantifying the SCR components occurs when the response to be scored is elicited immediately after a preceding response that has not had time to fully recover. It is customary to measure the amplitude of each response from its own individual deflection point (Grings & Lockhart, 1965; Edelberg, 1967). However, the amplitude and the temporal characteristics of the second response are distorted by being superimposed on the recovery of the first response. For example, the measurable amplitude of the second response will be smaller given its occurrence following the first response. The amount of distortion of the second response is a function of the size of the first response and the time since the first response (Grings & Schell, 1969). Although there is no perfect solution to the response interference effect when hand-scoring EDA, it can be pointed out that response frequency may be the least distorted component of the response in this situation.

Because of the challenges of scoring superimposed responses, the interstimulus intervals used in EDA research have historically been quite long, ranging from 20–60 sec. Scoring software is available from the manufacturers of several EDA recording systems, and customized software or shareware is frequently used as well. The "resources" portion of the Society for Psychophysiological Research (SPR) website contains a

software repository with downloadable versions of a number of programs for scoring SCR. The website (currently located at: www.sprweb.org/repository/index.cfm) includes descriptions of each program and contact information for the author/programmer. The key difference among available automated scoring programs is the algorithms used for identifying the onset and peak of individual responses. Traditional scoring algorithms are based on a trough-to-peak analysis in which waveform parameters such as slope change and rise time are used to identify an onset and peak for each response. Trough-to-peak methods appear most robust for scoring isolated single responses to discrete stimuli presented at long interstimulus intervals. Newer programs use waveform modeling algorithms to decompose EDA into separate tonic and phasic components and to more accurately isolate individual responses within overlapping waveforms such as responses to rapidly presented stimuli (for discussion and direct comparisons of some of these algorithms see Benedek & Kaernbach, 2010; Green, Kragel, Fecteau, & LaBar, 2014; Lim et al., 1997).

Another problem with quantifying the EDA components concerns the existence of large variability due to extraneous individual differences. Thus, whether an SCL of 8 μS is considered high, moderate, or low will depend upon that specific subject's range of SCLs. For example, one can see in Figure 10.4 that an SCL of 8 μS would be relatively low for the subject depicted in the upper tracing but would be relatively high for the subject depicted in the lower tracing. Similarly, an SCR of 0.5 μS may be relatively large for one person but relatively small for another. Lykken, Rose, Luther, and Maley (1966) proposed an interesting method to correct for this inter-individual variance called range correction. The procedure involves computing the possible range for each individual subject and then expressing the subject's momentary value in terms of this range. For example, one may compute a subject's minimum SCL during a rest period and a maximum SCL while the subject blows a balloon to bursting; the subject's present SCL can then be expressed as a proportion of his/her individualized range according to the following formula: (SCL – SCLmin)/(SCLmax – SCLmin). The rationale underlying these procedures is that an individual's range of EDA is due mainly to physiological variables unrelated to psychological processes (e.g., thickness of the corneum). It is the variation within these physiological limits that is normally of psychological interest (Lykken & Venables, 1971).

Although the range correction procedure can reduce error variance and increase the power of statistical tests in some datasets, it also can be problematic in others. For example, range correction would be inappropriate in a situation where two groups being compared had different ranges (Lykken & Venables, 1971). Taking a different approach, Ben-Shakhar (1985) has recommended using within-subject standardized scores to adjust for individual differences because this transformation relies upon the mean, a more stable and reliable statistic than the maximum response. Although these techniques may be useful under some circumstances, most investigators simply compare average values of SCL and SCR across groups, or compare difference scores within a group (e.g., SCL during a task minus SCL during rest).

Another important aspect of elicited SCRs is their decline in amplitude and eventual disappearance with repetition of the eliciting stimulus (SCR habituation). Habituation is a ubiquitous and adaptive phenomenon whereby subjects become less responsive to familiar and non-significant stimuli. There are several methods of quantifying habituation of the SCR (Siddle, Stephenson, & Spinks, 1983). One simple method involves counting the number of stimulus repetitions required to reach some predetermined level of habituation (e.g., two or three consecutive trials without measurable SCRs). This "trials-to-habituation" measure is useful and has been widely employed since its use by Sokolov (1963), but it is subject to considerable distortion by the occurrence of a single response. For example, whether an isolated SCR occurs on trial 3 can make the difference between a trials-to-habituation score of "0" (indicative of an atypical non-responder) and a "3" (indicative of a typical rate of habituation).

Another common measure of habituation is based on the rate of decline of SCR magnitude across trials as assessed by a "trials" main effect or interaction effect within an analysis of variance. However, this measure does not provide information about habituation in individual subjects and moreover can be distorted by differences in initial levels of responding.

A third measure of habituation is based on the regression of SCR magnitude on the log of the trial number (Lader & Wing, 1966; Montague, 1963). The regression approach provides a slope and an intercept score (the latter reflecting initial response amplitude), which are usually highly correlated with each other. Covariance procedures have been used to remove the dependency of slope on intercept, providing what Montague (1963) has called an "absolute rate of habituation." However, this technique rests on the assumptions that slope and intercept reflect different underlying processes and that the treatment effects under investigation do not significantly affect the intercepts (Siddle et al., 1983). Use of the slope measure also assumes that subjects respond on a sufficient number of trials to compute a meaningful slope, which may not be the case for some types of subjects with mild innocuous stimuli. Nevertheless, to the extent that these assumptions can be justified, the slope measure is often preferable because: (1) unlike the analysis of variance approach, individual habituation scores can be derived, (2) unlike the trials-to-habituation measure, isolated SCRs have less of a contaminating effect, (3) unlike trials-to-habituation, the slope measure makes fuller use of the magnitude data, and (4) unlike trials-to-habituation, the slope measure can discriminate between subjects who show varying

degrees of habituation but who fail to completely stop responding for two or three consecutive trials.

The temporal stability (test–retest reliability) of EDA measures such as the frequency of NS-SCRs, SCL, responsiveness to stimuli, and habituation has been fairly well investigated in normal healthy adults (see Freixa i Baque, 1982, for a discussion of early studies, and Schell, Dawson, Nuechterlein, Subotnik, & Ventura, 2002, for a later review). Test–retest correlations for periods extending up to one year or more have ranged from approximately 0.40 to 0.75 for NS-SCR frequencies, from 0.40 to 0.85 for SCL, and from 0.30 to 0.80 for number of SCRs elicited by a series of repeated stimuli. Stability of temporal measures (i.e., latency, rise time, etc.) is typically lower. Schell et al. (2002) found that as measures of overall responsiveness, simple counts of the number of SCRs elicited by a series of stimuli were more reliable than trials-to-habituation measures.

## ADVANTAGES AND DISADVANTAGES OF THE USE OF EDA

When one is considering use of EDA as an indicator of some psychological state or process of interest, it is well to remember that in the great majority of situations, changes in electrodermal activity do not occur in isolation. Rather, they occur as part of a complex of responses mediated by the autonomic nervous system.

Experimental treatments that have the effect of increasing SCL and/or NS-SCR rate also are expected to generally increase heart rate level and blood pressure and to produce peripheral vasoconstriction, to mention a few of the more commonly measured autonomic responses. The response or responses chosen for monitoring by a particular investigator should reflect considerations such as those discussed below.

For some researchers, EDA may be the response system of choice because, unlike most ANS responses, it provides a relatively direct and undiluted representation of sympathetic activity. As has been pointed out above, the neural control of the eccrine sweat glands is entirely under sympathetic control. Therefore, increases in SCL or the SCR are due to increased tonic or phasic sympathetic activation. In contrast, with heart rate as with most ANS functions (pupil diameter, gastric motility, blood pressure), a change in activity in response to stimuli of psychological significance cannot be unambiguously laid to either sympathetic or parasympathetic activity; it may be due either to one or to a combination of both. Thus, the researcher who wishes an unalloyed measure of sympathetic activity may prefer to monitor EDA, whereas the experimenter who wishes a broader picture of both sympathetic and parasympathetic activity may prefer heart rate, if constraints of instrumentation will allow only one to be recorded. Similarly, if for some reason (perhaps the use of medication with side effects on cholinergic or adrenergic systems) one wishes to monitor a response which is predominately cholinergically mediated at the periphery but which is also influenced by sympathetic activity, then EDA would be the choice.

Another advantage of measuring SCR is that its occurrence is generally quite discriminable. Thus, on a single presentation of a stimulus, one can determine by quick inspection whether or not an SCR has occurred. In contrast, the presence of a heart rate response on single stimulus presentation may be difficult to distinguish from ongoing variability in heart rate that reflects changes in muscle tonus or respiratory sinus arrhythmia.

For many investigators, an additional advantage of the use of EDA relative to other response systems is that of all forms of ANS activity, individual differences in EDA appear to be most reliably associated with psychopathological states. The correlates of some of these stable EDA differences between individuals are discussed in the next section.

Finally, it is important to note that, in comparison to many other psychophysiological measures, EDA is relatively inexpensive to record. After initial purchase of the recording system, expenses for each subject are trivial, involving electrode collars and paste and the occasional replacement of electrodes. Electrical shielding of the room in which the subject sits which is generally needed for noise-free recording of EEG or event-related potentials is unnecessary, and the costs of using EDA as a response measure are minuscule compared to those of hemodynamic techniques such as PET scans or functional MRI. Furthermore, the techniques used to record EDA are completely harmless and risk-free, and thus they can be used with young children and in research designs that require repeated testing at short intervals of time.

There are also potential disadvantages to the use of EDA as a dependent measure. First, EDA is a relatively slow-moving response system. As mentioned previously, the latency of the elicited SCR is between 1.0 and 3.0 sec, and tonic shifts in SCL produced by changes in arousal and alertness require approximately the same time to occur. Thus, an investigator who is interested in tracking very rapidly occurring processes, or stages within a complex process, may not find EDA useful. Although the SCR cannot index such rapidly occurring processes as sensory gating or stages of stimulus analysis on a real-time basis, it has been found to be correlated with real-time measures of these processes. For example, Lyytinen, Blomberg, and Näätänen (1992) observed that the parietal P3a was larger when an SCR was elicited by a novel tone than when no SCR was elicited.

Another potential disadvantage is that EDA has multiple causes; the elicited SCR is not specific to a single type of event or situation (as, for instance, the N400 ERP component appears to be specifically influenced by semantic expectancy; see Chapter 23 by Kutas et al. in the present volume). However, the multiple influences on EDA may actually be as much an advantage as a disadvantage. As described throughout this chapter, EDA can be used to

index a number of processes: activation, attention, and significance or affective intensity of a stimulus. Because of this complex causality, in using EDA as a response measure, one must take care to control experimental conditions – that is, be sure that one is varying only one process that may influence EDA at a time. Such experimental control is essential for all attempts to draw clear inferences from results, whether one is recording EDA, electrocortical activity, or a hemodynamic measure, given the number of processes that may influence these measures as well.

Thus, like any single response system, EDA has distinct advantages and disadvantages. The ideal situation, of course, is one in which the researcher can record more than one response measure. When ANS activity is of primary interest, EDA and heart rate are probably the two most common choices: EDA for its neuroanatomical simplicity, trial-by-trial visibility, and utility as a general arousal/attention indicator and heart rate for its potential differentiation of other psychological and physiological states of interest to the researcher.

## PSYCHOLOGICAL AND SOCIAL CONTEXT

In this section, we review the psychological and social factors that have been shown to influence EDA in three types of paradigms: (1) those that involve the presentation of discrete stimuli, (2) those that involve the presentation of continuous stimuli, and (3) those that involve examining the correlates of individual differences in EDA.

### Effects of discrete stimuli

Properties of stimuli to which the SCR is sensitive are wide and varied: they include stimulus novelty, significance/salience, surprisingness, intensity, and arousal content. It might be argued that, because EDA is sensitive to such a wide variety of stimuli, it is not a clearly interpretable measure of any particular psychological process (Landis, 1930). This view is certainly correct in the sense that it is impossible to identify an isolated SCR as an "anxiety" response, or an "anger" response, or an "attentional" response. However, the psychological meaning of an SCR becomes interpretable by taking into account the stimulus condition or experimental paradigm in which the SCR occurred. The better controlled the experimental paradigm, the more conclusive the interpretation.

One discrete stimulus paradigm that relies on the SCR's sensitivity to stimulus significance is the so-called Guilty Knowledge Test (Lykken, 1959) which is also known as the Concealed Information Test (CIT) (Verschuere, Ben-Shakhar, & Meijer, 2011). The CIT is a type of detection of deception test (popularly known as "lie detection" or "polygraph testing") that involves recording SCRs (as well as other physiological responses) while presenting subjects with a series of multiple-choice questions. For instance, a suspect in a burglary case might be instructed to answer "no" to each of the alternatives given for a question concerning details about the burglary (e.g., the specific item that was stolen). For each question, the correct alternative would be intermixed among other plausible alternatives. The guilty (knowledgeable) subject is expected to respond electrodermally more to the correct alternatives than neutral control alternatives, whereas the innocent (unknowledgeable) subject is expected to respond randomly. The theory behind the technique is that the correct answer to each question is more psychologically significant to a guilty subject than are other alternatives, whereas for the innocent subject all of the alternatives are of equal significance. For a discussion of the differing views of psychophysiological techniques of detecting deception, see Chapter 26, this volume.

Meijer, Selle, Elber, and Ben-Shahkar (2014) conducted a meta-analysis of the validity of the CIT, based on laboratory mock-crimes and personal-items paradigms, and using four physiological variables – skin conductance response, respiratory changes, changes in heart rate, and enhanced amplitude of the P300 event-related potential. They found that the CIT was a highly accurate method of detecting concealed information with all four physiological measures. The P300 was the most accurate measure followed by the SCR. It should be noted that the advantage of the P300 over the SCR was mediated by the type of paradigm: it was highly significant for studies using the personal-items paradigm but not for the mock-crime studies. The authors were careful to emphasize that the results of the meta-analysis cannot be generalized to real-life field studies. They noted a number of differences between the typical laboratory experiment and criminal investigations and called for research to bridge this gap in the future. However, for present purposes the results clearly demonstrate the sensitivity of the SCR to specific significant stimuli.

Another discrete stimulus paradigm in which EDA is commonly measured that highlights the influence of stimulus significance, processed at either a conscious or unconscious level, involves discrimination classical conditioning. For example, Dawson and Biferno (1973) employed a discrimination classical conditioning paradigm, in which college student subjects were asked to rate their expectancy of a brief electric shock (unconditioned stimulus, UCS) following each presentation of a CS+ (a conditioned stimulus regularly followed by the shock) and a CS– (a control stimulus never followed by shock). A distracting cognitive task was used to delay the subject's awareness of the CS+ – UCS contingency. There was no evidence of SCR discrimination conditioning prior to the development of subject's awareness of the CS+ – UCS contingency; however, once the subject became aware, the CS+ became more significant than the CS–, and there was an abrupt increase in the magnitude of the SCRs elicited by the CS+. Moreover, SCR discrimination conditioning failed to occur when the subject never became aware of the contingency. These results have

been frequently replicated (see reviews by Dawson & Schell, 1985; Lovibond & Shanks, 2002) and suggest that awareness of the CS–UCS relation, that is, awareness of CS+ significance, is necessary for human discrimination SCR conditioning under at least some conditions.

The conditions under which subjects need *not* be consciously aware of the stimulus significance in order to elicit an SCR have been a topic of considerable research. For example, SCR discrimination conditioning has been reported to occur without subjects becoming aware of the CS–UCS relationship under special circumstances when "prepared" stimulus relationships are conditioned. The concept of "preparedness" is that certain stimulus associations (e.g., taste with nausea and snakes with pain) are more quickly, easily, and automatically learned than are others (e.g., an arbitrary tone and a shock) and are more resistant to extinction because they have been correlated in our evolutionary past (Seligman, 1970).

Öhman and his colleagues extended Seligman's concept to human autonomic conditioning, using types of CSs that have been termed "biologically prepared," "potentially phobic," or "fear-relevant": pictures of spiders, snakes, and angry faces (for a review see Öhman, 2009; Öhman & Mineka, 2001). Öhman and his colleagues have demonstrated that SCRs conditioned with fear-relevant CSs and a shock UCS are more resistant to extinction than are SCRs conditioned with neutral CS–UCS relations and also were more resistant to cognitive manipulations such as extinction instructions informing subjects that the UCS would no longer be delivered (Hugdahl & Öhman, 1977). They were also retained past the point of cognitive extinction (no greater expectancy of the UCS after the CS+ than after the CS–) following the presentation of many nonreinforced trials (Schell, Dawson, & Marinkovic, 1991). Thus, the SCR may be elicited by stimuli that the subject does not consciously consider to be salient.

In later studies of this series, backward masking was used to prevent awareness of the CS–UCS relation by preventing conscious detection of the fear-relevant CSs. In this paradigm, visual CSs are presented very briefly (30 ms) and immediately followed by a masking stimulus. These procedures prevent recognition of the CSs in the vast majority of subjects on the vast majority of trials (Öhman, Dimberg, & Esteves, 1989a).

Esteves, Para, Dimberg, and Öhman (1994) paired a masked angry face (CS+) or masked happy face with shock. During subsequent extinction, unmasked CSs were presented and conditioned SCRs were elicited to the previously masked angry face CS+, but not to the happy face CS+. Thus, electrodermal conditioning was established "non-consciously" to a threatening angry face, but not to a smiling face. Conditioning to other masked biologically fear-relevant CSs was replicated in subsequent experiments by Öhman and Soares (1998) using pictures of snakes and spiders rather than angry faces. Studies using functional brain imaging techniques have replicated these SCR results and demonstrated the importance of the amygdala, extended regions of the amygdala complex, and sensory cortex in such unaware conditioning (Morris et al., 2001).

All in all, these findings indicate that SCR conditioned responses may be acquired without the subjects' awareness of the CS–UCS relation or conscious awareness of stimulus significance in some circumstances. The nature of these circumstances (only with biologically prepared fear-relevant stimuli or with certain types of brain damage?) is a topic of ongoing research.

SCRs elicited by discrete non-aversive stimuli are generally considered to be part of the orienting response (OR) to novel or significant stimuli, what Sokolov called "signal stimuli." The data reviewed in this section are consistent with this theoretical position. The task of subjects exposed to the CIT is to conceal knowledge, and the correct item is more relevant to this task than are incorrect alternative items. Thus, guilty subjects orient more to the crime-related significant items than the crime-irrelevant nonsignificant items. As Lykken (1974, p. 728) stated, "for the guilty subject only, the 'correct' alternative will have a special significance, an added 'signal value,' which will tend to produce a stronger orienting reflex than that subject will show to the other alternatives." Verschuere and Ben-Shakhar (2011) reviewed evidence that strongly supports the OR theory of responding in the CIT (e.g., greater heart rate deceleration and peripheral vasoconstriction occur following relevant items than irrelevant items in the CIT which is consistent with the orienting hypothesis). Likewise, the signal of an impending shock (CS+) is more significant than the signal of no shock (CS–) and therefore elicits larger conditioned ORs. All in all, the results observed here are consistent with the notion that the SCR is highly sensitive to stimulus significance, even under certain conditions where the reasons for that significance may not be consciously processed.

Other discrete stimuli capable of eliciting SCRs are those with either strong positive or negative affective valence. We orient to stimuli that are significant because they are either very positive or very negative in terms of their emotional properties. However, unlike responses such as the startle eye blink, the SCR does not distinguish arousing positive stimuli from equally arousing negative stimuli. Lang, Bradley, Cuthbert, and their colleagues have developed a set of widely used photographs (the International Affective Picture System, IAPS, Lang, Bradley, & Cuthbert, 1998; see Chapter 20, this volume) that are rated for both their arousal-producing quality and valence on a strongly positive to strongly negative scale. SCRs elicited by these pictures have reliably been found to be related to the arousal dimension, with responses increasing in magnitude as arousal rating increased for both positively valenced pictures (greater for erotic pictures than for beautiful flowers) and negatively valenced pictures (greater for striking snakes than for tombstones in a cemetery) (Lang, Greenwald, Bradley, & Hamm, 1993; Cuthbert, Bradley, & Lang, 1996). Bradley (2009) has

argued that the SCR response occurs whenever a stimulus activates either the appetitive or defensive motivational system and reflects an OR component indicative of a readiness to take action.

Other affective stimuli shown to evoke SCRs are those associated with internal processes involved in making decisions. As noted earlier, SCRs have been observed during the decision-making phase of gambling tasks in which participants must select among options that vary in their likely reward/outcome. This finding is consistent with what Damasio (1994) termed the "somatic marker" hypothesis, the main point of which is that decision-making is influenced by emotional somatic responses. As noted earlier, normal subjects, as opposed to brain-damaged patients, generate SCRs in anticipation of making "bad" decisions and begin to avoid those decisions over time. These results were interpreted as indicating that SCRs in response to decision-making processes reflect somatic markers that help the person make advantageous decisions.

In conclusion, in this section we have described some of the discrete stimulus paradigms in which EDA is most often measured and has proven to be most useful. We have emphasized that determining the psychological meaning of any particular SCR is dependent on a well-controlled stimulus situation. Finally, these areas of research examining the SCR to discrete stimuli underscore the point made previously that one advantage of the SCR is that the response can easily be measured on individual presentations of a stimulus. Thus, one may determine whether (1) the response to a "guilty" relevant stimulus in a group of stimuli is greater than that to "innocent" irrelevant stimuli, (2) the SCR elicited by a CS+ is greater on the first trial after awareness of the CS–UCS relationship occurs than on the last trial before that awareness occurs, (3) the eliciting stimulus is highly arousing due to either a positive or negative valence, and (4) arousal states that occur during decision-making guide decisions when risk is involved.

### Effects of continuous stimuli or situations

We turn now to an examination of the effects of more chronic, long-lasting stimuli or situations as opposed to the brief, discrete stimuli reviewed above. Chronic stimuli might best be thought of as modulating increases and decreases in tonic arousal. Hence, the most useful electrodermal measures in the context of continuous stimuli are SCL and frequency of NS-SCRs, because they can be measured on an ongoing basis over relatively long periods of time.

One type of continuous stimulus situation that reliably produces increases in electrodermal activity involves the necessity of performing a task. The anticipation and performance of practically any task will increase both SCL and the frequency of NS-SCRs, at least initially. For example, Lacey, Kagan, Lacey, and Moss (1963) recorded palmar SCL during rest and during the anticipation and performance of eight different tasks. The tasks ranged from those requiring close attention to *external* stimuli, such as listening to an irregularly fluctuating loud white noise, to those requiring close attention to *internal* information processing, such as solving mental arithmetic problems. The impressive finding for present purposes was that SCL increased in each and every one of the task situations. Typically, SCL increased about one μS above resting level during anticipation and then increased another one or two μS during performance of the task. Heart rate, unlike SCL, discriminated between tasks involving attention to external stimuli and tasks requiring attention to internal information processing.

Munro, Dawson, Schell, and Sakai (1987) observed that large increases in SCL and NS-SCR frequency were induced by a different task situation. In this case, college student subjects were tested during a five-minute rest period and then during performance of a continuous performance task. The task stimuli consisted of a series of digits presented visually at a rapid rate of one per second with exposure duration of 48 ms; the subject's task was to press a button whenever the digit "0" was presented. Both the number of NS-SCRs and SCL initially increased sharply from the resting levels during this demanding task and then gradually declined as the task continued.

Video games constitute another highly popular type of continuous task in which electrodermal activity has been usefully measured (see review by Kivikangas et al., 2011). Psychophysiological measures such as electrodermal activity provide relatively unobtrusive measures of continuous real time psychological experiences during the game play. Significant increases in SCL occur during the playing of video games compared to resting levels, particularly in games with high auditory and visual fidelity (greater realism of character images and sounds) (Ivory & Kalyanaraman, 2007). The representation of "self" (the avatar) is an important part of many contemporary games where participants choose their avatar's physical characteristics (e.g., gender and race) and personal characteristics (e.g., strength and intelligence). Lim and Reeves (2009) manipulated whether participants selected their own avatar. In one condition they chose their avatar from a set of six different characters and in a second condition the avatar was assigned by the experimenter. In addition, the characters' point-of-view (POV) was also manipulated at two levels: first person in which the camera was the eyes of the character and third person in which the camera showed both the avatar and the surroundings. Players displayed more frequent NS-SCRs and higher SCLs when playing with an avatar of their choice compared to playing with an assigned avatar. SCL was also higher in the third person condition when the avatar could be seen. Interestingly, these EDA differences were found even though there were no significant effects of avatar choice on the users' self-report of arousal.

In some video games the alternative characters are controlled by other humans and in other games the characters are controlled by computer software. Lim and Reeves (2010) examined the effects of participants being told they were playing with a computer-generated character or a human-controlled character under two different conditions: competition (dueling the opponent character) or cooperation (trading game items with the other player). The frequency of NS-SCRs, but not SCL, was significantly higher when the participants believed they were playing against a human than when playing against a computer-controlled agent. Both NS-SCR frequency and SCL, as well as heart rate, were significantly higher when participants were playing a competitive game than a cooperative game, suggesting that participants were more cognitively and emotionally engaged while carrying out a competitive game and while playing with a fellow human.

The finding that electrodermal activity is reliably elevated during task performance suggests that tonic EDA can be a useful index of a process related to "energy regulation" or "energy mobilization." An information processing interpretation of this finding might be that tasks require an effortful allocation of information resources and that this is associated with heightened autonomic activation (Jennings, 1986). A different, but not necessarily mutually exclusive, explanation would invoke the concepts of stress and affect rather than, or in addition to, attention and effortful allocation of resources. According to this view, laboratory tasks are challenging stressors, and a reliable physiological response to stressors is increased sympathetic activation, particularly EDA arousal.

Non-task related continuous stimulus situations that elicit strong emotions also increase tonic EDA arousal, as would be expected from the finding discussed above that SCR magnitude is affected by the arousal value of discrete stimuli with emotional valence. In a classic experiment, Ax (1953) created genuine states of fear and anger in subjects by causing them to feel in danger of a high-voltage shock due to equipment malfunction or by treating them in a rude and inconsiderate fashion. SCL, number of NS-SCRs, and several other measures of sympathetic nervous system activity rose during both the fear and the anger conditions, with the patterns for fear and anger differing to some degree (SCL rose more in fear than in anger, while NS-SCRs and diastolic blood pressure rose more in anger than in fear). Levenson, Gross, and their colleagues have used films in a number of studies to elicit emotional states (Gross & Levenson, 1993; Gross, 1998). SCL and other measures of sympathetic activation in these studies were higher during the films than during a baseline period, and the rise in SCL was influenced by the emotional regulation strategy that subjects were instructed to use. Participants instructed to suppress their facial display of emotion, to try to behave as though anyone observing them would not know what they were feeling, showed greater increases in SCL than participants who simply watched the films or who were instructed to reappraise what they were seeing, to watch the film with a detached, objective, and unemotional attitude.

Social stimulation constitutes another class of continuous stimuli that generally produces increases in EDA arousal. Social situations are ones in which the concepts of stress and affect are most often invoked. For example, early research related EDA recorded during psychotherapeutic interviews to concepts such as "tension" and "anxiety" on the part of both patient and therapist (Boyd & DiMascio, 1954; Dittes, 1957). In one such study, Dittes (1957) measured the frequency of NS-SCRs of a patient during 42 hours of psychotherapy. The results of this study indicated that the frequency of NS-SCRs was inversely related to the judged permissiveness of the therapist, and Dittes concluded that EDA reflects "the anxiety of the patient, or his 'mobilization' against any cue threatening punishment by the therapist" (p. 303).

There are other social interaction situations in which intense cognitive and affective reactions may occur that precipitate large changes in EDA and other physiological responses. For instance, EDA was recorded during stressful marital interactions (Levenson & Gottman, 1983, 1985). The researchers measured SCL (in addition to heart rate, pulse transmission time, and somatic activity) from married couples while they discussed conflict-laden problem areas. It was found that couples from distressed marriages had high "physiological linkage"; i.e., there were greater correlations between husbands' and wives' physiological reactions in distressed marriages than those in satisfying marriages during the discussions of problem areas. Moreover, greater physiological arousal, including higher SCL, during the interactions and during baselines was associated with a decline in marital satisfaction over the ensuing three years. Subsequent research has continued to use SCL and other autonomic measures to identify characteristics of individuals that make them most prone to being highly physiologically reactive during relationship conflicts (e.g., those with general attachment insecurity, Roisman, 2007; those who experience low maternal sensitivity to their needs as children, Raby, Roisman, Simpson, Collins, & Steele, 2015).

## Individual Differences in EDA

We have discussed the utility of EDA as a dependent variable reflecting situational levels of arousal/activation or attentiveness/responsiveness to individual stimuli. In this section we consider EDA as a relatively stable trait of the individual, as an individual difference variable. Individual differences in EDA are reliably associated with behavioral differences and psychopathological states of some importance, and we will examine some of these.

***EDA lability.*** Individual differences in the rate of NS-SCRs and the rate of SCR habituation have been used to define a trait called "electrodermal lability" (Mundy-Castle & McKiever, 1953; Lacey & Lacey, 1958; Crider, 1993).

Electrodermal "labiles" are subjects who show high rates of NS-SCRs and/or slow SCR habituation, whereas electrodermal "stabiles" are those who show few NS-SCRs and/or fast SCR habituation. Electrodermal lability is an individual trait that has been found to be relatively reliable over time, as the high test–retest correlations over time for the frequency of NS-SCRs, SCL, responsiveness to stimuli, and habituation discussed above would indicate. This stability is no doubt in part due to the degrees to which such lability is genetically controlled. Crider et al. (2004) studied several hundred monozygotic and dizygotic twins and found significant correlations between twins, much higher for the monozygotic twins, on NS-SCR rates and measures of habituation. They concluded that EDR lability as assessed by NS-SCR levels and measures of habituation represented a single latent phenotype equally influenced by genetic and unique environmental factors. The same conclusion was reached by Isen, Iacono, Mallone, and McGue (2012) with respect to frequency of SCR responding to non-signal tones. Vaidyanathan et al. (2014) studied a sample of over 4,000 cases from the Minnesota Twin Family Study and concluded that at least 50 percent of the variance in an EDA latent factor derived from SCL, SCR amplitude during a habituation task, and SCR response frequency was heritable. Their results suggested a polygenetic influence on EDA.

Labiles differ from stabiles with respect to a number of psychophysiological variables, including measures of both electrodermal and cardiovascular responsiveness (Kelsey, 1991; Schell et al., 1988). In the following, we review behavioral and psychological differences associated with this individual difference in both normal and abnormal populations.

***EDA lability in normal populations.*** Electrodermal lability is a trait of interest in psychological research in part because many investigators have reported that labiles outperform stabiles on tasks which require sustained vigilance. When individuals perform a signal detection task that is sustained over time, deterioration across time in the accurate detection of targets is frequently observed, a phenomenon referred to as vigilance decrement (Davies & Parasuraman, 1982). Several experimenters have reported that when vigilance decrement occurs, it is more pronounced among electrodermal stabiles than among labiles. This appears to be particularly true when EDA lability is defined by differences in SCR OR habituation rate (Koelega, 1990). As time on and the task goes by, labiles are apparently better able to keep attention focused on the task and to avoid a decline in performance (Crider & Augenbraun, 1975; Hastrup, 1979; Munro et al., 1987; Vossel & Rossman, 1984). With a difficult continuous performance task, Munro et al., for instance, whose study was mentioned previously, found that stabiles showed a significant decrement over time in performance, whereas labiles did not. The degree of task-induced sympathetic arousal as measured by increases in NS-SCR rate was negatively correlated across subjects with performance decrement.

Researchers investigating these sorts of behavioral differences between electrodermal stabiles and labiles have concluded that lability reflects the ability to allocate information processing capacity to stimuli which are to be attended to (Lacey & Lacey, 1958; Katkin, 1975; Schell et al., 1988). As Katkin (1975, p. 172) concluded, "electrodermal activity is a personality variable that reflects individual differences in higher central processes involved in attending to and processing information." Viewing electrodermal lability in this way suggests that labiles should differ from stabiles in a variety of information processing tasks. Consistent with this view, EDA labile children have been found to generally outperform stabiles on a variety of tasks that require perceptual speed and vigilance (Sakai, Baker, & Dawson, 1992).

Lability may also be correlated with broad personality characteristics. Crider (2008), after an extensive review of personality correlates of EDA lability, concluded that greater lability was associated with lower levels of overt expression of emotion and antagonistic impulses, so that the labile person is more inhibited in emotional expression and is more agreeable, whereas the stabile person is more expressive and antagonistic. He concluded that EDA lability reflects the effortful control of such expression and the allocation of cognitive resources to that control, a formulation consistent with the findings of Levenson, Gross, and their colleagues (cited above) that persons instructed to inhibit facial expression of emotion showed increased SCL, and with the observation of decreased EDA among psychopaths discussed below.

In addition to the differences between stabiles and labiles in the normal population, reliable abnormalities in electrodermal lability are associated with diagnosable psychopathology. We will next summarize EDA abnormalities reported in schizophrenia, psychopathy, and anxiety disorders. A more general discussion of psychophysiological abnormalities in these and other psychopathologies can be found in Chapter 25, this volume.

***EDA lability in schizophrenia.*** In general, two types of electrodermal abnormalities have been reported in different subgroups of patients with schizophrenia. First, between 40 percent and 50 percent of schizophrenia patients fail to show any SCR orienting responses to mild innocuous tones (termed "non-responders"), compared to approximately 10 percent non-responders in the normal population (see reviews by Bernstein et al., 1982; Dawson & Nuechterlein, 1984; Iacono, Ficken, & Beiser, 1993; Öhman, 1981). More recent data reported and reviewed by Venables and Mitchell (1996) suggest the percentage of SCR non-responders in normal groups may be closer to 25 percent.

The second electrodermal abnormality, found in the "responder" subgroup of patients, is the presence of higher

than normal levels of tonic arousal, indicated by high SCLs and a high frequency of NS-SCRs (Dawson & Nuechterlein, 1984; Dawson, Nuechterlein, & Schell, 1992a; Öhman, 1981). In effect, the non-responder group is characterized by hypo-responsivity to stimuli and EDA stability whereas the responder group is characterized by tonic hyper-arousal and EDA lability. Both types of abnormalities have been found to be reliable across time. For example, in a group of 56 chronic schizophrenia patients classified as non-responders on an initial test, 87 percent remained non-responders two weeks later and 91 percent were non-responders four weeks later (Spohn, Coyne, Wilson, & Hayes, 1989), and in a group of 29 young, recent onset non-responder schizophrenia outpatients, 62 percent remained non-responders one year later (Schell et al. 2002).

The hope associated with the identification of responder and non-responder EDA subgroups is that it will identify meaningful subgroups in terms of different symptomatic types of schizophrenia or different prognoses, or that one or both abnormalities might constitute a vulnerability marker for schizophrenia. Unfortunately, the results relating EDA abnormalities with current symptoms, future prognosis, and vulnerability have not always been consistent. As we point out later, the reasons for these inconsistencies may have to do with different populations of patients and control comparison groups, or with combined influences of more than one risk factor for poor outcome.

Non-responder and responder subgroups of patients have been reported by some investigators to show different symptomology at the time of testing, with responders displaying more symptoms of excitement, anxiety, manic behavior, and belligerence, whereas non-responders tend to show more emotional withdrawal, conceptual disorganization, and negative symptoms (e.g., Bernstein et al., 1981; Straube, 1979; Fuentes, Merita, Miquel, & Roja, 1993). Furthermore, SCR hypo-responsivity has been related to a more severe form of illness (Katsanis & Iacono, 1994), poor premorbid adjustment (Öhman et al., 1989b), and more psychiatric symptoms overall (positive and negative) (Green, Nuechterlein, & Satz, 1989; Kim, Shin, Kim, Cho, & Kim, 1993). Other investigators, however, have found the hyper-aroused responders to display the greater level of overall symptomatology (Brekke, Raine, Ansel, Lencz, & Bird, 1997; Dawson, Nuechterlein, Schell, & Mintz, 1992b).

Abnormally elevated EDA arousal also has been found particularly during periods of psychotic symptomatology, compared to the same patients during periods of remission. In within-subject comparisons of patients during both a period of symptomatic remission and during a period when psychotic symptoms were present, SCL and NS-SCR levels were not different from normal controls during remission but increased significantly to be greater than controls when the patients were symptomatic (Dawson, Gitlin, Schell, Nuechterlein, & Ventura, 1994). Moreover, heightened EDA arousal has been found to occur within a few weeks prior to an impending psychotic relapse, compared to control periods of stable remission within the same patients (Hazlett, Dawson, Schell, & Nuechterlein, 1997; Dawson et al., 2010). This finding is consistent with a theoretical model that hypothesizes that heightened sympathetic activation is associated with a "transient intermediate state" that precedes psychotic episodes in vulnerable individuals (Nuechterlein & Dawson, 1984). According to this theoretical model, these states constitute periods of heightened vulnerability with an increased risk of relapse, with the actual occurrence of relapses or exacerbation being influenced by environmental stressors.

The predominant finding in terms of predicting clinical outcome is that EDA hyper-arousal is associated with poor short-term symptomatic prognosis (Brekke, Raine, & Thomson, 1995; Frith, Stevens, Johnstone, & Crow, 1979; Zahn, Carpenter, & McGlashan, 1981; Dawson et al., 1992b; see review by Dawson & Schell, 2002). However, a minority of studies have reported that EDA hypo-responsivity, not hyper-arousal, is associated with poor prognosis (Hultman et al., 1996).

In a longer-term study (Tarrier & Barrowclough, 1989), the number of NS-SCRs and the change in SCL measured during interactions with relatives at the time the patients were hospitalized were found to be related to symptomatic relapse over the next two years. The direction of the effect, greater frequency of NS-SCRs and greater rise in SCL among the patients who later relapsed, is consistent with the hypothesis that patients at high risk of relapse have a predisposition to autonomic hyper-arousal to certain environmental or social stimuli. It has been well documented that patients are at increased risk for relapse if their family members are critical, hostile, or emotionally overinvolved with them (Brown, Birley, & Wing, 1972; Vaughn & Leff, 1976; Vaughn, Snyder, Jones, Freeman, & Falloon, 1984). The term *expressed emotion* (EE) is used to designate this continuum of attitudes on the part of the relative. Tarrier, Vaughn, Lader, and Leff (1979) found that schizophrenia outpatients who had a high EE relative showed higher levels of EDA activity in the presence of that relative than did patients with low EE relatives in the presence of such a relative, consistent with the finding that such patients are more likely to relapse.

Subotnik et al. (2012) examined the interaction of EDA lability and presence/absence of an EE relative in a patient's family to determine the joint effect of these two predictors of long-term outcome in schizophrenia. They assessed family EE, the number of SCR orienting responses to a series of tones when patients were stabilized as outpatients, and positive symptoms and negative symptoms both at outpatient stabilization and at a one-year follow-up. The number of orienting responses was greater among the patients with high EE relatives. A combination of number of orienting responses and presence of a high EE relative was found to have a unique effect in increasing

levels of negative symptoms both initially and at follow-up. More labile subjects (more reactive to environmental stimuli) who also had a high EE relative had higher levels of negative symptoms that all other groups. High lability in the absence of a stress-producing relative, or the presence of such a relative for a patient low in lability (typically a non-responder), did not increase negative symptoms.

The studies of prognosis reviewed above relied primarily upon measures of psychotic symptoms or hospital re-admission. However, some studies have measured prognosis as functional outcome, such as holding a job or having friends, instead of psychotic symptoms. Öhman et al. (1989b) reported that skin conductance non-responding and lower levels of tonic EDA activity taken at the beginning of a follow-up period predicted poor social and employment outcome over a two-year period in a subgroup of male schizophrenia patients. Conversely, Wieselgren, Öhlund, Lindstrom, & Öhman (1994), using an identical methodology to that used by Öhman et al. (1989b), reported an opposite relation for female schizophrenia patients, with high tonic electrodermal activity predicting poor social and work outcome. More recently Schell et al. (2005) used the same measure of outcome and reported results consistent with Wieselgren et al. That is, high SCL and NS-SCRs (as well as number of SCR ORs) were associated with poor social and occupational outcome and negative symptoms measured one-year later. Moreover, this was true for both males and females.

Schell et al. (2005) also raised the possibility that both EDA abnormalities in patients with schizophrenia (non-responsiveness and hyper-arousal) may predict poor functional outcome. Whether a particular study finds non-responders or responders to have the poorer outcome may depend upon whether the sample as a whole is more or less responsive or aroused than normal. Many of the studies reviewed above did not include comparison of patients to normal controls, instead selecting their EDA subgroups based solely on the distribution within the patient group. However, interesting differences are present among those that did report comparisons to normal. For example, Öhman et al. (1989b), who reported poorer functional outcome among non-responders, had a sample of patients who were much more likely to be non-responders and to have lower SCL than normal controls. However, Wieselgren et al. (1994) and Schell et al. (2005), both of whom reported poor outcome associated with the hyper-aroused responders, had groups of patients who did not differ from normal on SCR responsivity but did as a whole have higher than normal EDA arousal. Thus, Öhman et al.'s more abnormal non-responders had the poorer outcome, whereas Wieselgren et al.'s and Schell et al.'s more abnormal hyper-aroused responders had the poorer outcome. It may be that either abnormality, hypo-responsivity or hyper-arousal with respect to controls, is associated with poor outcome.

Finally, the issue of vulnerability to schizophrenia has been addressed in some EDA studies, again not always with consistent results, by examining first degree relatives of schizophrenia patients, usually the children of schizophrenia patients, who are not manifesting schizophrenic symtomatology. The most common finding in the early research using this methodology was abnormal hyper-arousal and/or hyper-reactivity to aversive stimuli in the offspring of schizophrenia patients (see reviews by Dawson & Nuechterlein, 1984; Öhman, 1981). Subsequent research has generally supported this finding (e.g., Hollister, Mednick, Brennan, & Cannon, 1994; Iacono, Ficken, & Beiser, 1999). However, the latter study reported the same abnormality in first degree relatives of patients with major depressive illness, a finding that suggests that electrodermal hyper-arousal may not be a vulnerability marker specific to schizophrenia.

In addition to being studied as a symptomatic correlate and a predictor of outcome in schizophrenia, the study of EDA has also clarified the nature of a symptom of schizophrenia, anhedonia (the reduced ability to experience pleasure). Persons with schizophrenia are often less expressive of emotion (both in terms of facial expression and vocal expression) than are persons without schizophrenia, both in terms of positive and negative emotion. This led to the view that schizophrenia patients had reduced emotional processes – affective blunting. However, Kring and Neale (1996) found that while patients with schizophrenia showed fewer facial responses to emotionally evocative film clips than did normal controls, they actually showed greater SCL increases, and their subjective experience was equal. Most subsequent studies have also found either greater EDA responsiveness to emotional stimuli among patients or no differences from controls (Kring & Elis, 2013), and startle eye blink studies have also found normal emotional expression among schizophrenia patients. Thus the view that patients do not process emotional stimuli to a normal degree has been found to be an oversimplification.

***EDA lability in psychopathy.*** Abnormalities in tonic EDA and SCR responsiveness have also been reported in other psychopathologies, particularly psychopathy. Psychopaths are usually characterized as low in arousal and deficient in feelings of fear and anxiety, leading to their thrill-seeking and antisocial behavior (Lykken, 1957; Quay, 1965). It would be expected that both of these abnormalities should be reflected in EDA abnormalities, in particular in lower tonic measures of arousal such as SCL and NS-SCRs, and in smaller SCRs given in response to stimuli that would be associated with fear or anxiety in normal individuals. Both such abnormalities have been reported among psychopaths.

Fowles (1993), in a review of EDA during resting conditions, concluded that lower levels of SCL were occasionally found among psychopaths, although effect sizes were small, and less evidence existed for lower NS-SCR levels. Lorber (2004), in a meta-analysis of 95 studies of EDA and HR in psychopathy, concluded that psychopaths were

characterized by reduced tonic EDA at rest, although again the effect sizes were small. Clearer differences from normal controls appear in tonic EDA levels as arousal increases, as, for instance, when simple orienting stimuli are presented (Fowles, 1993). Tonic EDA differences between psychopaths and normals clearly maximize when stressful stimuli are present (Fowles, 1993).

In one very well-known study which assessed not only tonic EDA but also responses to anxiety-provoking stimuli, Hare (1965) measured SCL in psychopathic and non-psychopathic prison inmates and college student controls during rest and while they watched the numbers 1–12 presented consecutively on a memory drum at 3 sec intervals. A strong electric shock was given as the number 8 was presented. Psychopathic subjects had lower SCL during rest and during the task than the other groups, and psychopathic inmates showed smaller increases in skin conductance from numbers 1 to 8 than did non-psychopathic inmates, which was interpreted as indicating less fear elicited in the interval prior to anticipated punishment. This finding with the "count-down" procedure has been replicated several times (for reviews, see Fowles, 1993 and Lykken, 1995).

As would be expected from Hare's findings, numerous investigators have reported that psychopaths show impaired SCR conditioning with aversive UCSs (usually electric shocks) (Lykken, 1957; Hare, 1965; Fowles, 1993; Rothemund et al., 2012). Veit et al. (2013) studied a sample of highly psychopathic incarcerated subjects and normal controls and also found reduced fear conditioned responses using a shock UCS among the psychopaths. Among these subjects the effect was specific to an affective factor of psychopathy (as opposed to lifestyle or antisocial factors).

Psychopaths also exhibit abnormal SCRs to other affective stimuli. Verona, Patrick, Curtin, Bradley, and Lang (2004) presented positively and negatively affectively valenced and neutral sounds (e.g., laughing baby, crying baby, clucking chicken) from the International Affective Digitized Sounds (IADS; Bradley & Lang, 1999) system to prison inmates assessed with the Psychopathy Checklist – Revised (PCL-R; Hare, 1991). The PCL-R assesses what are generally regarded as two factors of psychopathy, emotional detachment (e.g., egocentricity, shallow affect, and absence of remorse) and antisocial behavior (e.g., frequent trouble with the law, pathological lying, and substance abuse). Similar to the finding by Veit et al., those inmates scoring high specifically on the emotional detachment factor showed smaller responses to both pleasant and unpleasant sounds than did those who scored low on the factor, indicating that abnormalities in emotional processes in psychopathy extend beyond the realm of fear and anxiety.

More subtle abnormalities in emotional processes in psychopathy have also been suggested by EDA studies. An interesting study by Blair, Jones, Clark, and Smith (1997) presented psychopathic and non-psychopathic prison inmates with IAPS slides from three categories: non-threatening (e.g., a book), threatening (e.g., a very angry face), and distress (e.g., a crying child). The two groups did not differ in SCR magnitude to threatening and non-threatening stimuli, but the psychopaths responded less to the distress cues than non-psychopaths.

In addition to these abnormalities in EDA seen in adults diagnosed with psychopathy, lower levels of tonic EDA have been reported in children and adolescents who show psychopathic traits or who later exhibit antisocial behavior. Isen et al. (2010) found that 9–10-year-old boys (but not girls) from a community sample scoring high on the interpersonal factor of the Child Psychopathy scale gave smaller SCRs in response to a variety of non-signal auditory stimuli than boys who did not score high. Working with adolescents, Fung et al. (2005) found that in a count-down task like that used by Hare leading to an aversively loud noise, a psychopathy-prone group scoring high on the Child Psychopathy scale were more likely to be non-responders during the anticipatory period and were also less likely to respond to the noise itself.

In a prospective study, Raine, Venables, and Williams (1990) recorded EDA, heart rate (HR), and EEG during rest and several tasks from a sample of unselected 15-year-old schoolboys, and at a 10-year follow-up identified those who during the follow-up period had committed serious criminal offenses. As adolescents, the offenders had a lower rate of resting NS-SCRs, indicating lower arousal levels. The lower resting HR and greater EEG power in low frequency bands seen in the offender group also were consistent with lower arousal. Working with younger children, Gao, Raine, Venables, Dawson, and Mednick (2010) measured electrodermal fear conditioning using an aversive loud noise in a large sample of 3-year-olds and assessed criminal offending at age 23. When a group of predominantly male serious offenders was compared with a control group of non-offenders, SCR conditioning at age 3 was found to be absent in the future offender group.

The above studies make clear that deficits in electrodermal responding, particularly conditioning, precede the diagnosis of psychopathy or the development of criminal offending and thus constitute a risk factor for such behavior. However, it is worth noting that studies of the psychophysiological correlates of psychopathy have typically used only male subjects. Little if anything is known about psychophysiological abnormalities among female psychopaths.

***EDA liability in anxiety disorders*** Another area of clinical research in which electrodermal activity is proving to be useful is in the area of anxiety disorders. Electrodermal measures are being used to elucidate both state and trait differences in anxiety-related stimulus processing (e.g., Aue, Hoeppli, Piguet, Sterpenich, & Vuilleumier, 2013; Mosig et al., 2014; and see Pole, 2007, for a review), as well as to test important hypotheses about the etiology and treatment of anxiety disorders by investigating anxiety-related differences in the acquisition and extinction of

conditioned fear. A comprehensive review of the conditioned fear literature provided by Lissek et al. (2005) highlights the value of this approach. Their meta-analysis, which includes a comparison of findings across different dependent measures, suggests that EDA may provide unique information to these investigations. Although Lissek et al. note important limitations to using only SCR to index conditioned fear, they suggest combining SCR with other measures, such as the fear-potentiated startle, to help discriminate general arousal and attentional processes from processes that are fear-specific. Indeed, recent studies in which EDA measures have been used in combination with neuroimaging, ERP, and eye-tracking measures, have contributed important information toward our understanding of stimulus processing differences in trait anxiety (e.g., Haddad, Pritchett, Lissek, & Lau, 2012;), social anxiety (e.g., Moscovitch, Suvak, & Hofmann, 2010), and phobic disorders (e.g., Aue et al., 2013). In addition, an exciting avenue in this area of research is the use of electrodermal measures as a component of treatment outcome assessments. For example, Heeren, Reese, McNally, & Philippot (2012) included EDA in a study to assess the effects of an attention training protocol in social phobia, and several recent studies have used EDA to assess the effect of exposure therapies in the treatment of spider phobia (e.g., Dethier, Bruneau, & Philippot, 2015; Matthews, Naran, & Kirby, 2015; Shiban, Pauli, & Mühlberger, 2013; Van Bockstaele et al., 2011). Inclusion of EDA in these studies allows comparison of self-reported reactivity and distress to phobia-related stimuli with objective measures of arousal both pre- and post-treatment.

## EPILOGUE

EDA is a sensitive peripheral index of sympathetic nervous system activity that has proven to be a useful psychophysiological tool with wide applicability. Social and behavioral scientists have found that tonic EDA is useful to investigate general states of arousal and/or alertness, and that the phasic SCR is useful to study multifaceted attentional processes, as well as individual differences in both the normal and abnormal spectrum. We believe that future research will continue to support the use of EDA in a variety of situations and stimulus conditions.

An important direction for future research involves sharpening the inferential tool characteristics of EDA itself. That is, basic research is needed to address the specific conditions under which specific EDA components reflect specific psychological and physiological processes and mechanisms. For example, under what stimulus conditions does the SCR amplitude component of the orienting response reflect automatic preattentive cognitive processes versus controlled cognitive processes? Likewise, under what test situations do tonic and phasic EDA components reflect different brain systems? New technologies for ambulatory measurement as well as technologies that allow EDA recording within neuroimaging environments (see Boucsein et al., 2012) will facilitate exciting new applications for EDA measures. We expect that incorporating EDA measures into the expanding use of neuroimaging techniques in cognitive and affective neuroscience will elucidate these issues, making EDA an even more interesting and valuable psychophysiological tool.

## DEDICATION

We dedicate this chapter to William W. Grings, one of the pioneers in electrodermal activity who served as mentor to the first two authors, and friend to all three.

## REFERENCES


Andreassi, J. L. (2007). *Psychophysiology: Human Behavior and Physiological Response*, 5th edn. Hillsdale, NJ: Lawrence Erlbaum Associates.

Aue, T., Hoeppli, M., Piguet, C., Sterpenich, V., & Vuilleumier, P. (2013). Visual avoidance in phobia: particularities in neural activity, autonomic responding, and cognitive risk evaluations. *Frontiers in Human Neuroscience*, 7: 194. doi: 10.3389/fnhum.2013.00194

Ax, A. (1953). The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine*, 15: 433–442.

Bach, D. R. (2014). Sympathetic nerve activity can be estimated from skin conductance responses: a comment on Henderson et al. (2012). *NeuroImage*, 84: 122–123.

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50: 7–15.

Bechara, A., Damasio, H., Damasio, A. R., & Lee, G. P. (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *Journal of Neuroscience*, 19: 5473–5481.

Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, 275: 1293–1295.

Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C., & Damasio, A. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science*, 269: 1115–1118.

Benedek, M. & Kaernbach, C. (2010). Decomposition of skin conductance data by means of nonnegative deconvolution. *Psychophysiology*, 47: 647–658.

Ben-Shakhar, G. (1985). Standardization within individuals: a simple method to neutralize individual differences in skin conductance. *Psychophysiology*, 22: 292–299.

Bernstein, A. S., Frith, C., Gruzelier, J., Patterson, T., Straube, E., Venables, P., & Zahn, T. (1982). An analysis of the skin conductance orienting response in samples of American, British, and German schizophrenics. *Biological Psychology*, 14: 155–211.

Bernstein, A. S., Taylor, K. W., Starkey, P., Juni, S., Lubowsky, J., & Paley, H. (1981). Bilateral skin conductance, finger pulse volume, and EEG orienting response to tones of differing intensities in chronic schizophrenics and controls. *Journal of Nervous and Mental Disease*, 169: 513–528.

Blair, R. J., Jones, L., Clark, F., & Smith, M. (1997). The psychopathic individual: a lack of responsiveness to distress cues? *Psychophysiology*, 34: 192–198.

Bloch, V. (1993). On the centennial of the discovery of electrodermal activity. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Research* (pp. 1–6). New York: Plenum Press.

Boucsein, W. (2012). *Electrodermal Activity*, 2nd edn. New York: Springer.

Boucsein, W., Fowles, D. C., Grimnes, S., Ben-Shakhar, G., Roth, W. T., Dawson, M. E., & Filion, D. L. (2012). Publication recommendations for electrodermal measurements. *Psychophysiology*, 49: 1017–1034.

Boyd, R. W. & DiMascio, A. (1954). Social behavior and autonomic physiology: a sociophysiologic study. *Journal of Nervous and Mental Disease*, 120: 207–212.

Bradley, M. M. (2009). Natural selective attention: orienting and emotion. *Psychophysiology*, 46: 1–11.

Bradley, M. M. & Lang, P. J. (1999). *International Affective Digitized Sounds (IADS): Stimuli, Instruction Manual and Affective Ratings*. *Technical Report No. B-2, Center for Research in Psychophysiology*, University of Florida, Gainesville.

Brekke, J. S., Raine, A., Ansel, M., Lencz, T., & Bird, L. (1997). Neuropsychological and psychophysiological correlates of psychosocial functioning in schizophrenia. *Schizophrenia Bulletin*, 23: 19–28.

Brekke, J. S., Raine, A., & Thomson, C. (1995). Cognitive and psychophysiological correlates of positive, negative, and disorganized symptoms in the schizophrenia spectrum. *Psychiatry Research*, 57: 241–250.

Brown, G., Birley, J. L. T., & Wing, J. K. (1972). Influence of family life on the course of schizophrenia. *British Journal of Psychiatry*, 121: 241–248.

Cheng, D. T., Knight, D. C., Smith, C. N., & Helmstetter, F. J. (2006). Human amygdala activity during the expression of fear responses. *Behavioral Neuroscience*, 120: 1187–1195.

Cheng, D. T., Richards, J., & Helmstetter, F. J. (2007). Activity in the human amygdala corresponds to early, rather than late period autonomic responses to a signal for shock. *Learning & Memory*, 14: 485–490.

Crider, A. (1993). Electrodermal response lability-stability: individual difference correlates. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Research* (pp. 173–186). New York: Plenum Press.

Crider, A. (2008). Personality and electrodermal response lability: an interpretation. *Applied Psychophysiology and Biofeedback*, 33: 141–148.

Crider, A. & Augenbraun, C. B. (1975). Auditory vigilance correlates of electrodermal response habituation speed. *Psychophysiology*, 12: 36–40.

Crider, A., Kremen, W. S., Xian, H., Jacobson, K. C., Waterman, B., Eisen, S. A., . . . & Lyons, M. J. (2004). Stability, consistency, and heritability of electrodermal response lability in middle-aged male twins. *Psychophysiology*, 41: 501–509.

Critchley, H. D. (2002). Electrodermal responses: what happens in the brain. *Neuroscientist*, 8: 132–142.

Critchley, H. D. (2009). Psychophysiology of neural, cognitive and affective integration: fMRI and autonomic indicants. *International Journal of Psychophysiology*, 73: 88–94.

Critchley, H. D., Elliot, R., Mathias, C. J., & Dolan, R. J. (2000). Neural activity relating to generation and representation of galvanic skin conductance responses: a functional magnetic resonance imaging study. *Journal of Neuroscience*, 20: 3033–3040.

Critchley, H. D., Mathias, C. J., & Dolan, R. J. (2001). Neural activity in the human brain relating to uncertainty and arousal during anticipation. *Neuron*, 29: 537–545.

Cuthbert, B. N., Bradley, M. M., & Lang, P. J. (1996). Probing picture perception: activation and emotion. *Psychophysiology*, 33: 103–111.

Damasio, A. R. (1994). *Descartes' Error: Emotion, Reason, and the Human Brain*. New York: Grosset/Putnam.

Darrow, C. W. (1927). Sensory, secretory, and electrical changes in the skin following bodily excitation. *Journal of Experimental Psychology*, 10: 197–226.

Darrow, C. W. (1937). Neural mechanisms controlling the palmar galvanic skin reflex and palmar sweating. *Archives of Neurology and Psychiatry*, 37: 641–663.

Davies, D. R. & Parasuraman, R. (1982). *The Psychology of Vigilance*. London: Academic Press.

Dawson, M. E. & Biferno, M. A. (1973). Concurrent measurement of awareness and electrodermal classical conditioning. *Journal of Experimental Psychology*, 101: 55–62.

Dawson, M. E., Gitlin, M., Schell, A. M., Nuechterlein, K. H., & Ventura, J. (1994). Autonomic abnormalities in schizophrenia: state or trait indicators? *Archives of General Psychiatry*, 51: 813–824.

Dawson, M. E. & Nuechterlein, K. H. (1984). Psychophysiological dysfunctions in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, 10: 204–232.

Dawson, M. E., Nuechterlein, K. H., & Schell, A. M. (1992a). Electrodermal anomalies in recent-onset schizophrenia: relationships to symptoms and prognosis. *Schizophrenia Bulletin*, 18: 295–311.

Dawson, M. E., Neuchterlein, K. H., Schell, A. M., & Mintz, J. (1992b). Concurrent and predictive electrodermal correlates of symptomatology in recent-onset schizophrenic patients. *Journal of Abnormal Psychology*, 101: 153–164.

Dawson, M. E. & Schell, A. M. (1985). Information processing and human autonomic classical conditioning. In P. K. Ackles, J. R. Jennings, & M. G. H. Coles (eds.), *Advances in Psychophysiology, Volume 1* (pp. 89–165). Greenwich, CT: JAI Press.

Dawson, M. E. & Schell, A. M. (2002). What does electrodermal activity tell us about prognosis in the schizophrenia spectrum? *Schizophrenia Research*, 54: 87–93.

Dawson, M. E., Schell, A. M., Rissling, A., Ventura, J., Subotnik, K. L., & Nuechterlein, K. H. (2010). Psychophysiological prodromal signs of schizophrenic relapse: a pilot study. *Schizophrenia Research*, 123: 64–67.

Dethier, V., Bruneau, N., & Philippot, P. (2015). Attentional focus during exposure in spider phobia: the role of schematic versus non-schematic imagery. *Behaviour Research and Therapy*, 65: 86–92.

Dittes, J. E. (1957). Galvanic skin response as a measure of patient's reaction to therapist's permissiveness. *Journal of Abnormal and Social Psychology*, 55: 295–303.

Dubé, A., Duquette, M., Roy, M., Lepore, F., Duncan, G., & Rainville, P. (2009). Brain activity associated with the electrodermal reactivity to acute heat pain. *NeuroImage*, 45: 169–180.

Edelberg, R. (1967). Electrical properties of the skin. In C. C. Brown (ed.), *Methods in Psychophysiology* (pp. 1–53). Baltimore, MD: Williams & Wilkens.

Edelberg, R. (1972). Electrical activity of the skin: its measurement and uses in psychophysiology. In N. S. Greenfield & R. A. Sternbach (eds.), *Handbook of Psychophysiology* (pp. 367–418). New York: Holt.

Edelberg, R. (1993). Electrodermal mechanisms: a critique of the two-effector hypothesis and a proposed replacement. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Research* (pp. 7–29). New York: Plenum Press.

Esteves, F., Parra, C., Dimberg, U., & Ohman, A. (1994). Nonconscious associative learning: Pavlovian conditioning of skin conductance responses to masked fear-relevant facial stimuli. *Psychophysiology*, 31: 375–385.

Fan, J., Xu, P., Van Dam, N. T., Eilam-Stock, T., Gu, X., Luo, Y., & Hof, P. R. (2012). Spontaneous brain activity relates to autonomic arousal. *Journal of Neuroscience*, 32: 11176–11186.

Féré, C. (1888). Note on changes in electrical resistance under the effect of sensory stimulation and emotion. *Comptes Rendus des Séances de la Société de Biologie, (Series 9)*, 5: 217–219.

Ferguson, G. A. & Takane, Y. (1989). *Statistical Analysis in Psychology and Education*, 6th edn. New York: McGraw-Hill.

Fletcher, R. R., Dobson, K., Goodwin, M. S., Eydgahi, H., Wilder-Smith, O., Fernholz, D., ... & Picard, R. W. (2010). iCalm: wearable sensor and network architecture for wirelessly communicating and logging autonomic activity. *IEEE Transactions on Information Technology in Biomedicine*, 11: 215–223.

Fowles, D. C. (1974). Mechanisms of electrodermal activity. In R. F. Thompson & M. M. Patterson (eds.), *Methods in Physiological Psychology, Part C: Receptor and Effector Processes* (pp. 231–271). New York: Academic Press.

Fowles, D. C. (1986). The eccrine system and electrodermal activity. In M. G. H. Coles, E. Donchin, & S. W. Porges (eds.), *Psychophysiology: Systems, Processes, and Applications* (pp. 51–96). New York: Guilford Press.

Fowles, D. C. (1993). Electrodermal activity and antisocial behavior: empirical findings and theoretical issues. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Research* (pp. 223–237). New York: Plenum Press.

Fowles, D. C., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology*, 18: 232–239.

Freedman, L. W., Scerbo, A. S., Dawson, M. E., Raine, A., McClure, W. O., & Venables, P. H. (1994). The relationship of sweat gland count to electrodermal activity. *Psychophysiology*, 31: 196–200.

Freixa i Baque, E. (1982). Reliability of electrodermal measures: a compilation. *Biological Psychology*, 14: 219–229.

Frith, C. D., Stevens, M., Johnstone, E. C., & Crow, T. J. (1979). Skin conductance responsivity during acute episodes of schizophrenia as a predictor of symptomatic improvement. *Psychological Medicine*, 9: 101–106.

Fuentes, I., Merita, M. G., Miquel, M., & Rojo, J. (1993). Relationships between electrodermal activity and symptomatology in schizophrenia. *Psychopathology*, 26: 47–52.

Fung, M. T., Raine, A., Loeber, R., Lynam, D. R., Steinhauer, S. R., Venables, P. H., & Stouthamer-Loeber, M. (2005). Reduced electrodermal activity in psychopathy-prone adolescents. *Journal of Abnormal Psychology*, 114: 187–196.

Gao, Y., Raine, A., Venables, P. H., Dawson, M. E., & Mednick, S. A. (2010). Association of poor childhood fear conditioning and adult crime. *American Journal of Psychiatry*, 167: 56–60.

Gentil, A. F., Eskandar, E. N., Marci, C. D., Evans, K. C., & Dougherty, D. D. (2009). Physiological responses to brain stimulation during limbic surgery: further evidence of anterior cingulate modulation of autonomic arousal. *Biological Psychiatry*, 66: 695–701.

Green, M. F., Nuechterlein, K. H., & Satz, P. (1989). The relationship of symptomatology and medication to electrodermal activity in schizophrenia. *Psychophysiology*, 26: 148–157.

Green, S. R., Kragel, P. A., Fecteau, M. E., & LaBar, K. S. (2014). Development and validation of an unsupervised scoring system (autonomate) for skin conductance response analysis. *International Journal of Psychophysiology*, 91: 186–193.

Grey, S. J. & Smith, B. L. (1984). A comparison between commercially available electrode gels and purpose-made gel, in the measurement of electrodermal activity. *Psychophysiology*, 21: 551–557.

Grings, W. W. (1974). Recording of electrodermal phenomena. In R. F. Thompson & M. M. Patterson (eds.), *Bioelectric Recording Technique, Part C: Receptor and Effector Processes* (pp. 273–296). New York: Academic Press.

Grings, W. W. & Lockhart, R. A. (1965). Problems of magnitude measurement with multiple GSRs. *Psychological Reports*, 17: 979–982.

Grings, W. W. & Schell, A. M. (1969). Magnitude of electrodermal response to a standard stimulus as a function of intensity and proximity of a prior stimulus. *Journal of Comparative and Physiological Psychology*, 67: 77–82.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74: 224–237.

Gross, J. J. & Levenson, R. W. (1993). Emotional suppression: physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology*, 64: 970–986.

Haddad, A. D. M., Pritchett, D., Lissek, S., & Lau, J. Y. F. (2012). Trait anxiety and fear responses to safety cues: stimulus generalization or sensitization? *Journal of Psychopathology and Behavioral Assessment*, 34: 323–331.

Hare, R. D. (1965). Temporal gradient of fear arousal in psychopaths. *Journal of Abnormal Psychology*, 70: 442–445.

Hare, R. D. (1991). *The Hare Psychopathy Checklist – Revised*. Toronto: Multi-Health Systems.

Hassett, J. (1978). *A Primer of Psychophysiology*. San Francisco: W. H. Freeman and Company.

Hastrup, J. L. (1979). Effects of electrodermal lability and introversion on vigilance decrement. *Psychophysiology*, 16: 302–310.

Hazlett, H., Dawson, M. E., Schell, A. M., & Nuechterlein, K. H. (1997). Electrodermal activity as a prodromal sign in schizophrenia. *Biological Psychiatry*, 41: 111–113.

Heeren, A., Reese, H. E., McNally, R. J., & Philippot, P. (2012). Attention training toward and away from threat in social phobia: effects on subjective, behavioral, and physiological measures of anxiety. *Behaviour Research and Therapy*, 50: 30–39.

Hollister, J. M., Mednick, S. A., Brennan, P., & Cannon, T. D. (1994). Impaired autonomic nervous system habituation in those at genetic risk for schizophrenia. *Archives of General Psychiatry*, 51: 552–558.

Hot, P., Naveteur, J., Leconte, P., & Sequeira, H. (1999). Diurnal variations of tonic electrodermal activity. *International Journal of Psychophysiology*, 33: 223–230.

Hugdahl, K. (1984). Hemispheric asymmetry and bilateral electrodermal recordings: a review of the evidence. *Psychophysiology*, 21: 371–393.

Hugdahl, K. (1995). *Psychophysiology: The Mind–Body Perspective*. Cambridge, MA: Harvard University Press.

Hugdahl, K. & Öhman, A. (1977). Effects of instruction on acquisition and extinction of electrodermal responses to

fear-relevant stimuli. *Journal of Experimental Psychology: Human Learning and Memory*, 3: 608–618.

Hultman, C. M., Öhman, A., Öhlund, L. S., Wieselgren, I., Lindström, L. H., & Öst, L. (1996). Electrodermal activity and social network as predictors of outcome of episodes in schizophrenia. *Journal of Abnormal Psychology*, 105: 626–636.

Iacono, W. G., Ficken, J. W., & Beiser, M. (1993). Electrodermal nonresponding in first-episode psychosis as a function of stimulus significance. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Activity* (pp. 239–256). New York: Plenum Press.

Iacono, W. G., Ficken, J. W., & Beiser, M. (1999). Electrodermal activation in first-episode psychotic patients and their first-degree relatives. *Psychiatry Research*, 88: 25–39.

Isen, J. D., Iacono, W. G., Malone, S. M., & McGue, M. (2012). Examining electrodermal hyporeactivity as a marker of externalizing psychopathology: a twin study. *Psychophysiology*, 49: 1039–1048.

Isen, J. D., Raine, A., Baker, L., Dawson, M., Bezdjian, S., & Lozano, D. I. (2010). Sex-specific association between psychopathic traits and electrodermal reactivity in children. *Journal of Abnormal Psychology*, 119: 216–225.

Ivory, J. D. & Kalyanaraman, S. (2007). The effects of technological advancement and violent content in video games on players' feelings of presence, involvement, physiological arousal, and aggression. *Journal of Communication*, 57: 532–555.

Jennings, J. R. (1986). Bodily changes during attending. In M. G. H. Coles, E. Donchin, & S. W. Porges (eds.), *Psychophysiology: Systems, Processes, and Applications* (pp. 268–289). New York: Guilford Press.

Kappeler-Setz, C., Gravenhorst, F., Schumm, J., Arnrich, B., & Tröster, G. (2013). Towards long term monitoring of electrodermal activity in daily life. *Personal and Ubiquitous Computing*, 17: 261–271.

Katkin, E. S. (1975). Electrodermal lability: a psychophysiological analysis of individual differences in response to stress. In I. G. Sarason & C. D. Spielberger (eds.), *Stress and Anxiety, Volume 2* (pp. 141–176). Washington, DC: Aldine.

Katsanis, J. & Iacono, W. G. (1994). Electrodermal activity and clinical status in chronic schizophrenia. *Journal of Abnormal Psychology*, 103: 777–783.

Kelsey, R. M. (1991). Electrodermal lability and myocardial reactivity to stress. *Psychophysiology*, 28: 619–631.

Kim, D. K., Shin, Y. M., Kim, C. E., Cho, H. S., & Kim, Y. S. (1993). Electrodermal responsiveness, clinical variables, and brain imaging in male chronic schizophrenics. *Biological Psychiatry*, 33: 786–793.

Kivikangas, J. M., Chanel, G., Cowley, B., Ekman, I., Salminen, M., Järvelä, S., & Ravaja, N. (2011). A review of the use of psychophysiological methods in game research. *Journal of Gaming and Virtual Worlds*, 3: 181–199.

Koelega, H. S. (1990). Vigilance performance: a review of electrodermal predictors. *Perceptual and Motor Skills*, 70: 1011–1029.

Kring, A. M. & Elis, O. (2013). Emotion deficits in people with schizophrenia. *Annual Review of Clinical Psychology*, 9: 409–433.

Kring, A. M. & Neale, J. M. (1996). Do schizophrenic patients show a disjunctive relationship among expressive, experiential, and psychophysiological components of emotion? *Journal of Abnormal Psychology*, 105: 249–257.

Krzywicki, A. T., Berntson, G. G., & O'Kane, B. L. (2014). A non-contact technique for measuring eccrine sweat gland activity using passive thermal imaging. *International Journal of Psychophysiology*, 94: 25–34.

LaBar, K. S., Gatenby, J. C., Gore, J. C., LeDoux, J. E., & Phelps, E. A. (1998). Human amygdala activation during conditioned fear acquisition and extinction: a mixed-trial fMRI study. *Neuron*, 20: 937–945.

Lacey, J. I., Kagan, J., Lacey, B. C., & Moss, H. A. (1963). The visceral level: situational determinants and behavioral correlates of autonomic response patterns. In P. H. Knapp (ed.), *Expression of the Emotions in Man* (pp. 161–196). New York: International Universities Press.

Lacey, J. I. & Lacey, B. C. (1958). Verification and extension of the principle of autonomic response-stereotypy. *American Journal of Psychology*, 71: 50–73.

Lader, M. H. & Wing, L. (1966). *Psychological Measures, Sedative Drugs, and Morbid Anxiety*. London: Oxford University Press.

Landis, C. (1930). Psychology of the psychogalvanic reflex. *Psychological Review*, 37: 381–398.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1998). *International Affective Picture System (IAPS): Technical Manual and Affective Ratings*. Center for Research in Psychophysiology, University of Florida, Gainesville.

Lang, P. J., Greenwald, M. K., Bradley, M. M., & Hamm, A. O. (1993). Looking at pictures: affective, facial, visceral, and behavioral reactions. *Psychophysiology*, 30: 261–273.

Levenson, R. W. & Gottman, J. M. (1983). Marital interaction: physiological linkage and affective exchange. *Journal of Personality and Social Psychology*, 45: 587–597.

Levenson, R. W. & Gottman, J. M. (1985). Physiological and affective predictors of change in relationship satisfaction. *Journal of Personality and Social Psychology*, 49: 85–94.

Lim, S. & Reeves, B. (2009). Being in the game: effects of avatar choice and point of view on psychophysiological responses during play. *Media Psychology*, 12: 348–370.

Lim, S. & Reeves, B. (2010). Computer agents versus avatars: responses to interactive game characters controlled by a computer or other player. *International Journal of Human–Computer Studies*, 68: 57–68.

Lim, C. L., Rennie, C., Barry, R. J., Bahramali, H., Lazzaro, I., Manor, B., & Gordon, E. (1997). Decomposing skin conductance into tonic and phasic components. *International Journal of Psychophysiology*, 25: 97–109.

Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., & Pine, D. S. (2005). Classical fear conditioning in the anxiety disorders: a meta-analysis. *Behaviour Research and Therapy*, 43: 1391–1424.

Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: a meta-analysis. *Psychological Bulletin*, 130: 531–552.

Lovibond, P. F. & Shanks, D. R. (2002). The role of awareness in Pavlovian conditioning: empirical evidence and theoretical implications. *Journal of Experimental Psychology: Animal Behavior Processes*, 28: 3–26.

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Social Psychology*, 55: 6–10.

Lykken, D. T. (1959). The GSR in the detection of guilt. *Journal of Applied Psychology*, 43: 383–388.

Lykken, D. T. (1974). Psychology and the lie detector industry. *American Psychologist*, 29: 725–739.

Lykken, D. T. (1995). *The Antisocial Personalities*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Lykken, D. T., Rose, R. J., Luther, B., & Maley, M. (1966). Correcting psychophysiological measures for individual differences in range. *Psychological Bulletin*, 66: 481–484.

Lykken, D. T. & Venables, P. H. (1971). Direct measurement of skin conductance: a proposal for standardization. *Psychophysiology*, 8: 656–672.

Lyytinen, H., Blomberg, A. P., & Näätänen, R. (1992). Event-related potentials and autonomic responses to a change in unattended auditory stimuli. *Psychophysiology*, 29: 523–534.

Mangina, C. A. & Beuzeron-Mangina, J. H. (1996). Direct electrical stimulation of specific human brain structures and bilateral electrodermal activity. *International Journal of Psychophysiology*, 22: 1–8.

Matthews, A., Naran, N., & Kirkby, K. C. (2015). Symbolic online exposure for spider fear: habituation of fear, disgust and physiological arousal and predictors of symptom improvement. *Journal of Behavior Therapy and Experimental Psychiatry*, 47: 129–137.

Meijer, E. H., Selle, N. K., Elber, L., & Ben-Shakhar, G. (2014). Memory detection with the concealed information test: a meta analysis of skin conductance, respiration, heart rate, and P300 data. *Psychophysiology*, 51: 879–904.

Montague, J. D. (1963). Habituation of the psycho-galvanic reflex during serial tests. *Journal of Psychosomatic Research*, 7: 199–214.

Morris, J. S., Buchel, C., & Dolan, R. J. (2001). Parallel neural responses in amygdala subregions and sensory cortex during implicit fear conditioning. *NeuroImage*, 13: 1044–1052.

Moscovitch, D. A., Suvak, M. K., & Hofmann, S. G. (2010). Emotional response patterns during social threat in individuals with generalized social anxiety disorder and non-anxious controls. *Journal of Anxiety Disorders*, 24: 785–791.

Mosig, C., Merz, C. J., Mohr, C., Adolph, D., Wolf, O. T., Schneider, S., & Zlomuzica, A. (2014). Enhanced discriminative fear learning of phobia-irrelevant stimuli in spider-fearful individuals. *Frontiers in Behavioral Neuroscience*, 8: 328. doi:10.3389/fnbeh.2014.00328

Mundy-Castle, A. C. & McKiever, B. L. (1953). The psychophysiological significance of the galvanic skin response. *Journal of Experimental Psychology*, 46: 15–24.

Munro, L. L., Dawson, M. E., Schell, A. M., & Sakai, L. M. (1987). Electrodermal lability and rapid performance decrement in a degraded stimulus continuous performance task. *Journal of Psychophysiology*, 1: 249–257.

Neumann, E. & Blanton, R. (1970). The early history of electrodermal research. *Psychophysiology*, 6: 453–475.

Nuechterlein, K. H. & Dawson, M. E. (1984). A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin*, 10: 300–312.

Öhman, A. (1981). Electrodermal activity and vulnerability to schizophrenia: a review. *Biological Psychology*, 12: 87–145.

Öhman, A. (2009). Of snakes and faces: an evolutionary perspective on the psychology of fear. *Scandinavian Journal of Psychology*, 50: 543–552.

Öhman, A., Dimberg, U., & Esteves, F. (1989a). Preattentive activation of aversive emotions. In T. Archer & L. G. Nilsson (eds.), *Aversion, Avoidance, and Anxiety* (pp. 169–193). Hillsdale, NJ: Lawrence Erlbaum Associates.

Öhman, A. & Mineka, S. (2001). Fears, phobias, and preparedness: toward an evolved module of fear and fear learning. *Psychological Review*, 108: 483–522.

Öhman, A., Öhlund, L. S., Alm, T., Wieselgren, I., Öst, L., & Lindström, L. H. (1989b). Electrodermal nonresponding, premorbid adjustment, and symptomatology as predictors of long-term social functioning in schizophrenics. *Journal of Abnormal Psychology*, 98: 426–435.

Öhman, A. & Soares, J. J. F. (1998). Emotional conditioning to masked stimuli: expectancies for aversive outcomes following nonrecognized fear-relevant stimuli. *Journal of Experimental Psychology: General*, 127: 69–82.

Patterson, J. C., Ungerleider, L. G., & Bandettini, P. A. (2002). Task-independent functional brain activity correlation with skin conductance changes: an fMRI study. *NeuroImage*, 17: 1797–1806.

Payne, A. F. H., Dawson, M. E., Schell, A. M., Singh, K., & Courtney, C. G. (2013). Can you give me a hand? A comparison of hands and feet as optimal anatomical sites for skin conductance recording. *Psychophysiology*, 50: 1065–1069.

Payne, A. F., Schell, A. M., & Dawson, M. E. (2016). Lapses in skin conductance responding across anatomical sites: comparison of fingers, feet, forehead, and wrist. *Psychophysiology*, 53: 1084–1092.

Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans: role of the amygdala and vmPFC. *Neuron*, 43: 897–905.

Picard, R. W., Fedor, S., & Ayzenberg, Y. (2015). Multiple arousal theory and daily-life electrodermal activity asymmetry. *Emotion Review*, 8. doi: 10.1177/1754073914565517

Picard, R. W. & Healey, J. (1997). Affective wearables. *Personal Technologies*, 1: 231–240.

Poh, M., Swenson, N. C., & Picard, R. W. (2010). A wearable sensor for unobtrusive, long-term assessment of electrodermal activity. *IEEE Transactions on Biomedical Engineering*, 57: 1243–1252.

Pole, N. (2007). The psychophysiology of posttraumatic stress disorder: a meta-analysis. *Psychological Bulletin*, 133: 725–746.

Prokasy, W. F. & Kumpfer, K. L. (1973). Classical conditioning. In W. F. Prokasy & D. C. Raskin (eds.), *Electrodermal Activity in Psychological Research* (pp. 157–202). New York: Academic Press.

Prokasy, W. F. & Raskin, D. C. (eds.) (1973). *Electrodermal Activity in Psychological Research*. New York: Academic.

Quay, H. C. (1965). Psychopathic personality as pathological stimulation-seeking. *American Journal of Psychiatry*, 122: 180–183.

Raby, K. L., Roisman, G. I., Simpson, J. A., Collins, W. A., & Steele, R. D. (2015). Greater maternal insensitivity in childhood predicts greater electrodermal reactivity during conflict discussions with romantic partners in adulthood. *Psychological Science*, 26: 348–353.

Raine, A., Venables, P. H., & Williams, M. (1990). Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. *Archives of General Psychiatry*, 47: 1003–1007.

Roisman, G. I. (2007). The psychophysiology of adult attachment relationships: autonomic reactivity in marital and premarital interactions. *Developmental Psychology*, 43: 39–53.

Rothemund, Y., Ziegler, S., Hermann, C., Gruesser, S. M., Foell, J., Patrick, C. J., & Flor, H. (2012). Fear conditioning in psychopaths: event-related potentials and peripheral measures. *Biological Psychology*, 90: 50–59.

Roy, J. C., Sequeira, H., & Delerm, B. (1993). Neural control of electrodermal activity: spinal and reticular mechanisms. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Research* (pp. 73–92). New York: Plenum Press.

Sakai, L. M., Baker, L. A., & Dawson, M. E. (1992). Electrodermal lability: individual differences affecting perceptual speed and vigilance performance in 9 to 16 year-old children. *Psychophysiology*, 29: 207–217.

Sano, A., Picard, R. W., & Stickgold, R. (2014). Quantitative analysis of wrist electrodermal activity during sleep. *International Journal of Psychophysiology*, 94: 382–389.

Scerbo, A. S., Freedman, L. W., Raine, A., Dawson, M. E., & Venables, P. H. (1992). A major effect of recording site on measurement of electrodermal activity. *Psychophysiology*, 29: 241–246.

Schell, A. M., Dawson, M. E., & Filion, D. L. (1988). Psychophysiological correlates of electrodermal lability. *Psychophysiology*, 25: 619–632.

Schell, A. M., Dawson, M. E., & Marinkovic, K. (1991). Effects of potentially phobic conditioned stimuli on retention, reconditioning, and extinction of the conditioned skin conductance response. *Psychophysiology*, 28: 140–153.

Schell, A. M., Dawson, M. E., Nuechterlein, K. H., Subotnik, K. L., & Ventura, J. (2002). The temporal stability of electrodermal variables over a one-year period in patients with recent-onset schizophrenia and in normal subjects. *Psychophysiology*, 39: 124–132.

Schell, A. M., Dawson, M. E., Rissling, A., Ventura, J., Subotnik, K. L., Gitlin, M. J., & Nuechterlein, K. H. (2005). Electrodermal predictors of functional outcome and negative symptoms in schizophrenia. *Psychophysiology*, 42: 483–492.

Schumm, J., Bachlin, M., Setz, C., Arnrich, B., Roggen, D., & Troster, G. (2008). Effect of movements on the electrodermal response after a startle event. *Methods of Information in Medicine*, 47: 186–191.

Seligman, M. E. (1970). On the generality of the laws of learning. *Psychological Review*, 77: 406–418.

Sequeira, H. & Roy, J.-C. (1993). Cortical and hypothalamo-limbic control of electrodermal responses. In J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier (eds.), *Progress in Electrodermal Research* (pp. 93–114). New York: Plenum Press.

Shiban, Y., Pauli, P., & Mühlberger, A. (2013). Effect of multiple context exposure on renewal in spider phobia. *Behaviour Research and Therapy*, 51: 68–74.

Shields, S. A., MacDowell, K. A., Fairchild, S. B., & Campbell, M. L. (1987). Is mediation of sweating cholinergic, adrenergic, or both? A comment on the literature. *Psychophysiology*, 24: 312–319.

Siddle, D., Stephenson, D., & Spinks, J. A. (1983). Elicitation and habituation of the orienting response. In D. Siddle (ed.), *Orienting and Habituation: Perspectives in Human Research* (pp. 109–182). Chichester: John Wiley.

Sokolov, E. N. (1963). *Perception and the Conditioned Reflex*. New York: Macmillan.

Spohn, H. E., Coyne, L., Wilson, J. K., & Hayes, K. (1989). Skin-conductance orienting response in chronic schizophrenics: the role of neuroleptics. *Journal of Abnormal Psychology*, 98: 478–486.

Stern, R. M., Ray, W. J., & Quigley, K. S. (2001). *Psychophysiological Recording*. Oxford University Press.

Straube, E. R. (1979). On the meaning of electrodermal nonresponding in schizophrenia. *Journal of Nervous and Mental Disease*, 167: 601–611.

Subotnik, K. I., Schell, A. M., Chilingar, M. S., Dawson, M. E., Ventura, J., Kelly, K. A., … & Nuechterlein, K. H. (2012). The interaction of electrodermal activity and expressed emotion in predicting symptoms in recent-onset schizophrenia. *Psychophysiology*, 49: 1035–1038.

Tarchanoff, J. (1890). Galvanic phenomena in the human skin during stimulation of the sensory organs and during various forms of mental activity. *Pflugers Archive für die Gesamte Physiologie des Menschen und der Tiere*, 46: 46–55.

Tarrier, N. & Barrowclough, C. (1989). Electrodermal activity as a predictor of schizophrenic relapse. *Psychopathology*, 22: 320–324.

Tarrier, N., Vaughn, C., Lader, M. H., & Leff, J. P. (1979). Bodily reactions to people and events in schizophrenics. *Archives of General Psychiatry*, 36: 311–315.

Tartz, R., Vartak, A., King, J., & Fowles, D. (2015). Effects of grip force on skin conductance measured from a handheld device. *Psychophysiology*, 52: 8–19.

Tranel, D. (2000). Electrodermal activity in cognitive neuroscience: neuroanatomical and neurophysiological correlates. In R. D. Lane & L. Nadel (eds.), *Cognitive Neuroscience of Emotion* (pp. 192–224). Oxford University Press.

Vaidyanathan, U., Isen, J. D., Malone, S. M., Miller, M. B., McGue, M., & Iacono, W. G. (2014). Heritability and molecular genetic basis of electrodermal activity: a genome-wide association study. *Psychophysiology*, 51: 1259–1271.

Van Bockstaele, B., Verschuere, B., Koster, E. H. W., Tibboel, H., De Houwer, J., & Crombez, G. (2011). Effects of attention training on self-reported, implicit, physiological and behavioural measures of spider fear. *Journal of Behavior Therapy and Experimental Psychiatry*, 42: 211–218.

van Dooren, M., de Vries, J. J., & Janssen, J. H. (2012). Emotional sweating across the body: comparing 16 different skin conductance measurement locations. *Physiology & Behavior*, 106: 298–304.

Vaughn, C. E. & Leff, J. (1976). The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129: 125–137.

Vaughn, C. E., Snyder, K. S., Jones, S., Freeman, W. B., & Falloon, I. R. H. (1984). Family factors in schizophrenic relapse: a California replication of the British research on expressed emotion. *Archives of General Psychiatry*, 41: 1169–1177.

Veit, R., Konica, L., Klinzing, J. G., Barth, B., Yilmaz, O., & Birbaumer, N. (2013). Deficient fear conditioning in psychopathy as a function of interpersonal and affective disturbances. *Frontiers in Human Neuroscience*, 7: 706. doi:10.3389/fnhum.2013.00706

Venables, P. H. & Christie, M. J. (1973). Mechanisms, instrumentation, recording techniques, and quantification of responses. In W. F. Prokasy & D. C. Raskin (eds.), *Electrodermal Activity in Psychological Research* (pp. 1–124). New York: Academic Press.

Venables, P. H. & Christie, M. J. (1980). Electrodermal activity. In I. Martin & P. H. Venables (eds.), *Techniques in Psychophysiology* (pp. 3–67). Chichester: John Wiley.

Venables, P. H. & Mitchell, D. A. (1996). The effects of age, sex and time of testing on skin conductance activity. *Biological Psychology*, 43: 87–101.

Verona, E., Patrick, C. J., Curtin, J., Bradley, M. M., & Lang, P. J. (2004). Psychopathy and physiological responses to emotionally evocative sounds. *Journal of Abnormal Psychology*, 113: 99–108.

Verschuere, B. & Ben-Shakhar, G. (2011). Theory of the concealed information test. In B. Verschuere, G. Ben-Shakhar, & E. H. Meijer (eds.), *Theory and Application of the Concealed Information Test* (pp. 128–148). Cambridge University Press.

Verschuere, B., Ben-Shakhar, G., & Meijer, E. H. (eds.). (2011). *Memory Detection: Theory and Application of the Concealed Information Test*. Cambridge University Press.

Vigouroux, R. (1879). Sur le rôle de la résistance électrique des tissues dans l'electro-diagnostic. *Comptes Rendus Société de Biologie*, 31: 336–339.

Vigouroux, R. (1888). The electrical resistance considered as a clinical sign. *Progrès Medicale*, 3: 87–89.

Vossel, G. & Rossman, R. (1984). Electrodermal habituation speed and visual monitoring performance. *Psychophysiology*, 21: 97–100.

Wallin, B. G. (1981). Sympathetic nerve activity underlying electrodermal and cardiovascular reactions in man. *Psychophysiology*, 18: 470–476.

Wang, G. H. (1964). *The Neural Control of Sweating*. Madison, WI: University of Wisconsin Press.

Wieselgren, I. M., Öhlund, L. S., Lindstrom, L. H., & Öhman, A. (1994). Electrodermal activity as a predictor of social functioning in female schizophrenics. *Journal of Abnormal Psychology*, 103: 570–573.

Wilhelm, F. H., Pfaltz, M. C., Grossman, P., & Roth, W. T. (2006). Distinguishing emotional from physical activation in ambulatory psychophysiological monitoring. *Behavioral Sciences Instrumentation*, 42: 458–463.

Wilhelm, F. H. & Roth, W. T. (1998). Taking the laboratory to the skies: ambulatory assessment of self-report, autonomic, and respiratory responses in flying phobia. *Psychophysiology*, 35: 596–606.

Woodworth, R. S. & Schlosberg, H. (1954). *Experimental Psychology*, rev. edn. New York: Holt & Co.

Zahn, T. P., Carpenter, W. T., & McGlashan, T. H. (1981). Autonomic nervous system activity in acute schizophrenia: II. Relationships to short-term prognosis and clinical state. *Archives of General Psychiatry*, 38: 260–266.

Zahn, T. P., Grafman, J., & Tranel, D. (1999). Frontal lobe lesions and electrodermal activity: effects of significance. *Neuropsychologia*, 37: 1227–1241.

Zhang, S., Hu, S., Chao, H. H., Luo, X., Farr, O. M., & Li, C. S. (2012). Cerebral correlates of skin conductance responses in a cognitive task. *NeuroImage*, 62: 1489–1498.