The Terrible Twos—Anger and Anxiety

Hazardous to Your Health

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Anxiety and anger are hazardous to health. This article offers a selective review of research that illustrates how anxiety and anger increase vulnerability to illnesses, compromise the immune system, increase lipid levels, exacerbate pain, and increase the risk of death from cardiovascular disease and from all sources of death. Possible mechanisms for such effects are identified, including the role of cardiovascular reactivity. Finally, data are provided on Anxiety Management Training, a brief, structured psychological intervention that has proven effective in anxiety as well as anger management.

A recent study of 46,000 workers at six major U.S. companies confirmed that employees with high psychosocial risk factors incur higher medical costs (Goetzl et al., 1998). Those employees showing both high psychosocial and physical risk factors for heart disease had medical bills that were three times higher than those of employees who were free of risk. Among the major risk factors identified was stress. This finding is yet another documenting the consequences of stress on physical health. The purpose of this article is to review the evidence supporting the premise that both anxiety and anger are emotional conditions that are hazardous to health.

This article is divided into two parts. The first part summarizes illustrative research findings showing the impact of anxiety and anger—the “terrible twos”—on physical illness, the immune system, cholesterol levels, pain, and mortality.1 The second part presents a psychological intervention, developed at Colorado State University, called Anxiety Management Training (AMT), which has proven effective for both anxiety and anger management.

Part 1. Anxiety and Anger Are Hazardous to Your Health

Physical Illness

Stress and anger are hazardous to health by increasing a person’s susceptibility to illness. The influence of stress on physical illness is readily illustrated by a prospective study of adolescent girls. Siegel and Brown (1988) surveyed 364 girls in Grades 7 to 11. Testing was conducted at two time periods: in September and again in May of the following year. Positive and negative life events were recorded for the previous year (Time 1) and since the fall semester began (Time 2). Correlations between negative life events and illnesses were significant for both time periods ($r_1 = .26$ and $r_2 = .32$) but did not reach significance for positive life events ($r_1 = .02$ and $r_2 = .09$). Illnesses tended to be upper respiratory infections such as colds, sore throats, or headaches. The authors also found a significant interaction effect whereby a high number of negative life experiences was especially predictive of illness when the number of positive life experiences was also low.

Although other researchers have concluded that stress is especially associated with an increase in upper respiratory infections (Kiecolt-Glaser, Dura, Speicher, Trask, & Glaser, 1991), one very interesting study found a possible association with cancer. Stone, Mezzacappa, Donatone, and Gonder (1999) examined results from a community prostate cancer screening program involving 318 men, of whom 90% were Caucasian, 3% were African American, and 3% were Asian. These were generally older men, with their mean age being 60 years. Prostate-specific antigen (PSA) levels suggestive of prostate cancer were analyzed along with stress and social support levels. Stress was assessed with the Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983), and social support was measured with the Satisfaction With Social Contacts questionnaire (Schulz, Williamson, Morycz, & Biegel, 1992).

Both stress and social support levels were related to PSA levels but in opposite directions. Raw PSA scores were 2.46 for men with high stress, 1.53 for men with low stress, 1.58 for men with high social support, and 2.46 for men with low social support. A chi-square analysis found

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1 Readers who are interested in seeing additional relevant research articles can obtain a bibliography from the author.
that stress and social support were significantly associated with abnormal PSA values: Specifically, 16.0% of high-stress men versus 4.8% of low-stress men had elevated PSA values, whereas 2.8% of men with high social support versus 12.9% of men with low social support had elevated PSA values. Correlations between stress scores and PSA scores and between social contact and PSA values were 0.22 and −0.21, respectively, both significant, indicating that high stress was associated with high PSA values whereas high social support was associated with low PSA values.

If stress increases vulnerability to illness, there are some data suggesting that anger–hostility may also be associated with physical illness. Ranchor, Sanderman, Bouma, Buunk, and van den Heuvel (1997) identified patients diagnosed with various physical illnesses from a random sample of 2,663 men in the Netherlands. Illness categories included respiratory illnesses such as asthma, internal disease such as disease of the liver, and rheumatic arthritis. Scores on the Buss Durkee Hostility Inventory (Buss & Durkee, 1957) were used to measure three components of hostility: resentment, suspiciousness, and aggression. In contrast with the sample of healthy men, the hostility scores of men with various physical illnesses were significantly higher on all three components. For example, aggression scores were as follows: 16.46 for healthy persons, 17.40 for patients with respiratory illness, 18.09 for patients with internal disease, and 17.95 for patients with rheumatic arthritis.

Immune System
Stress and anger are hazardous to health because they impair the immune system. A number of studies using different methodologies and samples have demonstrated the impairment of cellular immunity resulting from stress. Kiecolt-Glaser et al. (1991) compared changes in cellular immunity in a sample of 69 spousal caregivers of patients with Alzheimer’s disease and a control sample matched for age, income, and education. Caregivers provided care over an average of five years, for more than eight hours a day, leading to chronic stress. Over 13 months, caregivers showed compromised cellular immunity in three functional measures: antibody titers to latent Epstein–Barr virus and blastogenesis with two mitogens, concanavalin A and phytohemagglutinin.

Of special interest were individuals considered “at risk” on the basis of consistent decrements across all three functional immune assays. No differences existed between this at-risk group and other caregivers in years or hours per day of caregiving. However, the at-risk group did report higher levels of distress associated with experiencing dementia behaviors of their spouses, such as wandering, being unable to recognize familiar persons, or hiding objects. This result on the at-risk caregivers offered further support for the relationship between stress and immune function.

The caregivers’ data also demonstrated a relationship between stress and vulnerability to illness. Although health-related behaviors such as smoking, alcohol use, caffeine intake, and medication did not differ compared with the control group, the caregiver individuals reported more days of being unable to perform normal duties because of illnesses, longer illness episodes, and more physician visits for illnesses. The primary type of illness was upper respiratory tract infections.

Relying on a natural environmental stressor, Kiecolt-Glaser et al. (1986) followed 34 medical students before and during course examinations. Reductions indicative of immune downregulation were observed in helper/inducer T-lymphocytes and in the helper/inducer-suppression/cytotoxic cell ratio. For further analysis, half of the participants were randomly assigned to relaxation training. In this group, frequency of relaxation practice to reduce stress turned out to be a significant predictor of the percentage of helper/inducer cells during the examination period.

As a final illustration, Matthews et al. (1995) induced stress through experimental conditions. A speech stress required women to defend themselves against an accusation of committing a crime. On the basis of their cardiovascular response to this speaking stress task, women were classified as high or low reactors. They also faced two other stressful tasks, mirror tracing and the Stroop Color Word Test. Results showed declines in cell immunity levels (CD4+) for the high reactors as a result of the experimentally induced stress experiences.

Anger also has been found to negatively affect cellular immunity. Perhaps the most intriguing study is by Kiecolt-Glaser et al. (1993) on newlyweds. Ninety couples who obtained their marriage licenses 4–6 months previously were admitted for 24 hours to the Ohio State University Clinical Research Center to control for diet, caffeine intake, and physical activity. They were asked to resolve two or three marital issues involving conflict for 30 minutes while their interactions were videotaped. These behavioral com-
munications were then coded as high or low anger. The couples were selected on stringent mental and physical health criteria, including high marital satisfaction.

When the high-anger couples were compared with the low-anger couples, decreases were discovered in four functional immunological assays—natural killer cell lysis, blastogenic response to two mitogens, and the proliferative response to a monoclonal antibody to the T3 receptor. Except for the natural killer cell result, the general findings were consistent with the research demonstrating the effects of stress in reducing cellular immunity levels. As specific examples, high-anger couples showed greater reductions in percentage of macrophages after the high-stress conflict than low-anger couples declined from 12.41% to 11.48%. Macrophage cells are considered to be important in initiating immune activity. Neutrophil activity is considered relevant to fighting bacteria. High-anger couples showed declines in neutrophils (from 9.74% to 9.31%), whereas low-anger couples showed increases (from 6.77% to 8.42%). Finally, similar to findings about stress by Kiecolt-Glaser et al. (1991), antibody titers to latent virus were compromised for the high-anger couples compared with the low-anger couples.

**Stress, Immunity, Infections, and Recovery**

Whereas the previously cited studies directly measured cellular immunity changes, two studies tested the hypothesis that if the immune system is impaired from stress, then consequences should be observable in vulnerability to illness or rate of recovery. Regarding stress, Cohen et al. (1998; Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997; Cohen, Tyrrell, & Smith, 1991, 1993) studied how stress affects vulnerability to viral infections. Volunteers were 125 men and 151 women judged to be in good health. Participants were exposed by means of nasal drops to low doses of one of two different respiratory viruses. They were quarantined and then evaluated for cold symptoms through nasal secretion and behavioral samples. Life stresses were assessed by the Life Events and Difficulties Schedule (Harris, 1991).

Results demonstrated that chronic life stress was clearly associated with vulnerability to developing colds. For the group high in chronic stress, 69% developed colds compared with 27% in the group low in chronic stress. Chronic interpersonal stressors and under- or unemployment were the stressors most commonly predictive of infections. Ingestion of Vitamin C lowered the risk, but zinc intake did not. There was also a linear relationship between duration of stressor and relative risk of a cold, referred to by the authors as a dose–response effect—that is, the risk of developing a cold increased as a direct function of the duration of life stress.

Another daring study on stress created biopsy scalpel wounds in the hard palate of 11 volunteer dental students during summer vacation and again during their first major examination (Marucha, Kiecolt-Glaser, & Favaghehi, 1998). The researchers measured the size of the wounds from first being established to final healing to determine rate of healing. They found that the rate of healing was 40% slower during the high-stress examination period than during the more relaxing vacation period: It took 10.91 days for healing during the high-stress period versus 7.82 days for healing during vacation. This slower pattern of healing during stress was uniformly characteristic of every participant.

To determine the relationship between stress level and immune cell functioning, Marucha et al. (1998) also took measures of interleukin-I beta messenger RNA cells, cells responsible for helper immune cell growth. Results confirmed a 71% decline in immune cells from the low-stress assay period to the high-stress assay period.

**Pain**

Stress and anger are hazardous to health because they can influence pain perception. With lower tolerance for pain or continuation of pain, recovery or adjustment to illness can be affected. A variety of research approaches have studied the way in which stress or anger influences pain perception. In an early report with a sample of 56 Japanese students, Shiomi (1978) used a cold pressor test to study the association between anxiety and pain experiences. Cold pressor tests involved repeated trials at each of two temperature levels, 3 °C and 4 °C. Correlations between pain tolerance and Taylor Manifest Anxiety scores (Taylor, 1953) at 3 °C were –.49 for the first trial and –.50 for the second trial. For 4 °C, the correlations were –.43 and –.49 for the first and second trials, respectively. Thus, high anxiety was associated with low pain tolerance.

Two studies actually experimentally induced anxiety and then studied the relation between anxiety and participants’ pain perception or pain tolerance. Rhudy and Meagher (2000) induced anxiety by leading participants to anticipate the delivery of a painful electric shock. Pain was induced by exposing the participant’s finger to a radiant heat bulb focused through a condenser lens. Pain threshold was measured by latency to finger withdrawal. Results showed that anxiety diminished pain threshold compared with baseline levels and when anxious participants were compared with control participants. Cornwall and Dondori (1988) also induced anxiety to study its effect on perception of pressure-induced pain. Anxiety was induced through preinstructions that prolonged exposure to the pressure could be harmful or that a stressful interview regarding the participants’ intelligence or personality would immediately follow the pain task. Both types of induced anxiety increased these participants’ perception of pain intensity compared with participants exposed to the painful pressure but without anxiety instructions.

More recently, Keogh and Birkby (1999) confirmed significant correlations between anxiety sensitivity and pain threshold as well as pain intensity, with correlations of –.22 and .23, respectively. Furthermore, there was a linear relationship between anxiety sensitivity scores and pain measures. Linton (2000) focused on research studies of patients suffering from back or neck pain. From a database of 913 studies, he reviewed 37 between 1991 and 1999 that met the criterion of involving prospective longitudinal de-
signs. Sample sizes ranged from 45 to more than 4,500, with 16 samples numbering more than 400 participants. On the basis of his own grading system, Linton concluded that “stress, distress, or anxiety as well as mood and emotions . . . were found to be significant factors” influencing pain (p. 1148).

There have also been a number of studies confirming a relationship between anger and pain levels. Using the cold pressor laboratory test and using the State-Trait Anger Expression Inventory (Spielberger et al., 1985), Gelkopf (1997) found anger-in correlated .60 with pain assessment and −.43 with pain tolerance. Outside of the laboratory, several studies have proven an association between anger and pain perception and daily functioning in patients experiencing pain. Burns, Johnson, Mahoney, Devine, and Pawl (1996; Burns, Johnson, Devine, Mahoney, & Pawl, 1998) examined 101 chronic pain patients before and after a multidisciplinary pain treatment program. Anger-out was found to be significantly negatively correlated with improvement assessed by a lifting test (r = −.20). High anger-out among male patients was also significantly correlated with lower lifting (r = −.32).

Chronic pain patients were also studied by Korns, Rosenberg, and Jacob (1994). These patients from a Department of Veterans Affairs Medical Center had experienced pain for an average of 11.9 years, generally due to musculoskeletal low-back pain. Again, anger was significantly correlated with pain perception as well as interference with daily functioning. Anger-in correlated .41 with pain intensity, .36 with pain behaviors, and .29 with interference with daily functioning. Anger intensity was also significantly correlated with pain intensity (r = .30), pain behaviors (r = .26), and interference with daily functioning (r = .38).

Cholesterol

A proven threat to health is cholesterol level. Documentation also demonstrates that stress and anger are hazardous to health through increasing cholesterol levels. Stoney, Bausserman, Ninura, Marcus, and Flynn (1999a) measured cholesterol levels of 127 male and female pilots under acute and chronic stress. Of these pilots, 68.5% were flying for commercial airlines. The chronic stress involved the proficiency examination required of pilots every 6–12 months, consisting of aircraft or flight simulation maneuvers and procedures. Failure can lead to demotion, suspension, mandatory retraining, or revocation of the pilot’s commercial license. The acute stress condition involved a serial subtraction task and a five-minute speech stress test, which involved defending oneself against a shoplifting accusation before a judge. In both stress conditions, cholesterol levels increased over baseline. When the pilots were faced with the chronic stressor, their cholesterol levels increased from 196.52 to 212.68; in the acute stress conditions, cholesterol levels increased from 195.93 to 199.16 with the serial subtraction task and to 202.70 with the speech stress test.

McCann and colleagues (McCann et al., 1999; McCann, Warlick, & Knopp, 1990) operationally defined major work deadline periods as indicative of high stress among employees. Participants were 173 attorneys studied during a high workload period involving imminent tax deadlines versus normal workload periods. Plasma lipid levels increased during the stressful workload period even though dietary intake and exercise remained constant.

Trait anger has been found to be correlated with cholesterol among both Caucasian and African American samples. Waldstein, Manuck, Bachen, Muldoon, and Bricker (1990) used the State-Trait Anger Expression Inventory (Spielberger, 1988) and reported correlations between cholesterol level and trait anger to be .44 and between cholesterol level and anger-out to be .46. The sample involved 29 healthy young Caucasian men. In a similar manner, Johnson, Collier, Nazzarro, and Gilbert (1992) obtained lipid data on 38 male African American volunteers who were free of heart disease, hypertension, and medication. No significant correlations between anger and cholesterol levels were discovered; however, trait anger was significantly correlated with the LDL–HDL ratio (r = −.41).

Cardiovascular Disease and Death

Of course the most serious consequence of hazards to health is death. Some convincing research has shown the relationship of anxiety and anger to cardiovascular disease; to deaths from cardiovascular disease; and, surprisingly, to total deaths from all causes. In an early review of more than 50 epidemiologic, clinical, and experimental studies of behavioral influences on atherosclerosis and cardiovascular disease, Manuck, Kaplan, and Matthews (1986) concluded that high levels of life stress were predictive of coronary heart disease. Miller, Smith, Turner, Gujjarro, and Hallet (1996), from a meta-analysis of 45 studies, also concluded “that hostility is an independent risk factor for coronary heart disease” (p. 322).

To study cardiovascular disease, Kaplan and colleagues (Kaplan et al., 1996; Kaplan, Manuck, Adams, Weingand, & Clarkson, 1987; Manuck, Marsland, Kaplan, & Williams, 1995) worked with nonhuman primates. Cynomolgus monkeys were assigned to either a high- or low-stress living condition and were fed a fat diet “designed to mimic that often eaten by North Americans” (Kaplan, Botchin, & Manuck, 1994, p. 133). After almost two years, the monkeys’ coronary arteries were examined for extent of atherosclerosis. Results showed that dominant monkeys living in the stress condition had more than twice the amount of atherosclerosis than dominant monkeys living in the nonstress condition.

Matthews, Owens, Kuller, Sutton-Tyrrell, and Jansen-McWilliams (1998), in a prospective study, followed 200 healthy women for whom measures of hostility and anxiety were obtained. Ten years later, measures of atherosclerosis were obtained by using ultrasound of carotid arteries to measure intima-media thickness, a marker of atherosclerosis. After smoking, triglycerides, and pulse pressure were statistically controlled, results showed a significant relationship between the earlier measures of anxiety and
hostility and current levels of atherosclerotic disease symptoms.

A rare longitudinal study of African Americans was conducted on hostility and cardiovascular disease symptoms by Irizarry et al. (1999). As known as the Coronary Artery Risk Development in Young Adult Study, participants were 374 African American and Caucasian men and women from Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California. After 10 years, amount of coronary artery calcification was assessed using electron-beam computed-tomographic instrumentation. Results using the Cook-Medley Hostility scale showed a nearly linear relationship between hostility scores and amount of coronary artery blockage. For instance, for hostility scores less than 14, 8% of the participants were positive for calcification; for hostility values between 14 and 19, blockage increased to 9% of the group; for hostility scores between 20 and 25, blockage level involved 17% of participants; and for hostility scores greater than 25, blockage was found for 18% of participants.

Numerous research studies have also found that level of anger-hostility is associated with death from cardiovascular disease as well as total deaths. In a series of prospective studies, Barefoot and colleagues (Barefoot, 1989; Barefoot, Dahlstrom, & Williams, 1983; Barefoot, Larsen, von der Lieth, & Schroll, 1995; Barefoot et al., 1987) demonstrated a significant association between Cook-Medley Hostility scale scores and cardiovascular disease incidence and deaths. Samples included 118 law students followed for almost 30 years, 255 medical school students followed for 25 years, 500 older adults followed for 15 years, and 830 Danish older adults followed for 27 years. With the law students, a linear relationship was found between hostility scores and deaths. For the medical students, hostility level was associated with both cardiovascular disease deaths and death from all causes. For instance, the mortality rate for cardiovascular disease for low hostility was 0.9 per 1,000 versus 4.5 per 1,000 for high hostility. For total mortality, the figures were 0.9 per 1,000 for low hostility versus 5.8 per 1,000 for high hostility. The research with older adults also showed similar results, even when age, health status, and traditional risk factors such as smoking, lipid levels, and blood pressure were controlled.

An intriguing speculation regarding the mechanism that ties anxiety-anger to mortality is offered by two studies on mortality and the immune system. Murasko, Weinger, and Kaye (1988) studied 403 elderly adults and identified 18% who had impaired immune systems, confirmed by assays showing lower lymphocyte proliferation in response to three mitogens. These individuals had twice the mortality rate of participants with normal immune functions. It is interesting that the primary cause of death was cardiovascular-related disease or sudden death.

Wayne, Rhine, Garry, and Goodwin (1990) reported similar findings in a 20-year prospective study of 273 healthy adults over age 60. Once again, poorer cellular immunity was predictive of subsequent morbidity and mortality. Given the documentation that anxiety and anger compromise the immune system, this possibly is the mechanism explaining the additional association between anxiety-anger and mortality.

**Mechanisms**

With the incontrovertible evidence that anxiety and anger are hazardous to health, the question arises regarding the mechanisms involved. Several possibilities have been suggested:

1. Anxiety-anger leads to vigilance and scanning, which, along with the emotional arousal itself, involves activation of the sympathetic nervous system (Smith, Ruiz, & Uchino, 2000; Williams, Barefoot, & Shekelle, 1985). The prior research regarding anxiety-anger and cardiovascular reactivity is relevant to this hypothesis.

In addition, the premise is that arousal of the sympathetic nervous system mediates other bodily functions such as the cardiovascular system, the immune system, and lipid levels (Dinsdale, Herd, & Hartley, 1983; Niaura, Stoney, & Herbert, 1992; Stoney & West, 1997). For instance, Sgoutas-Einich et al. (1994) demonstrated significant interactions among the autonomic nervous system, the immune system, and the endocrine system. Also, Stoney, Baussman, Niaura, Marcus, and Flynn (1999) reported that high cardiovascular reactors showed greater cholesterol changes when under chronic stress than did low cardiovascular reactors.

2. Anxiety-anger leads to poor health behaviors (Leiker & Hailey, 1988). For example, a number of studies have reported on the association between anxiety or anger and unhealthy lifestyles, such as alcohol consumption, unhealthy diet, use of tobacco, disturbed sleep patterns, poor compliance with medical regimes, and distrust of physicians (Cohen & Williamson, 1991; Lee et al., 1992; Scherwitz et al., 1992; Siegler, Peterson, Barefoot, & Williams, 1992; Smith & Christensen, 1992b).

3. Anxiety-anger is associated with psychosocial characteristics affecting vulnerability. For instance, correlates of anger have been found to include low social support and high interpersonal conflicts, both factors influencing health (Krantz, Conrado, Hill, & Friedler, 1988; Smith & Pope, 1990; Suarez & Williams, 1989).

4. Anxiety-anger as well as health levels are an expression of underlying constitutional or biological factors (Krantz & Durel, 1983; Plomin, Chipuer, & Loehlin, 1990). The hypothesized basic biological factor might be a hyperresponsive nervous system or even serotonin. Williams (1994) summarized information in support of the premise that brain serotonin differences not only can account for increasing health risk behaviors such as poor eating habits, alcoholic intake, and smoking but also can precipitate hostile behaviors.

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2 For additional research on stress and risk among African Americans, see Anderson, McNeilly, and Myers (1991); Barnes et al. (2000); Calderon et al. (1999); Light et al. (1995); and Pickering (1999).
Cardiovascular Reactivity

Among these various explanations, the role of cardiovascular reactivity has received increased attention. For example, one study compared African Americans and Caucasians as well as men and women. Durel et al. (1989) challenged 135 African American and Caucasian men and women with the stresses of a cold pressor test, the Type A Structured Interview (Rosenman, 1978), and a video game. Results indicated that African Americans showed greater systolic and diastolic blood pressure reactivity during the cold pressor test than Caucasians. In contrast, Caucasian participants showed higher blood pressure reactivity than African Americans during the Structured Interview. Finally, men had greater diastolic blood pressure reactivity than women during the video game. These findings not only demonstrated the influence of stress on cardiovascular reactivity and the different effects of different stressors but also pointed to some potential ethnic and gender differences.

Fleming, Baum, Davidson, Rectanus, and McArule (1987) operationally defined chronic stress as high-density living. Seventeen residents in crowded city blocks and 24 residents in low-density areas completed an embedded-figures acute stressor while cardiovascular reactivity measures were taken. Increases in systolic and diastolic blood pressures and heart rate from baseline to poststress were used as indicators of reactivity. Results showed that persons living in higher density housing had significantly greater increases in cardiovascular reactivity as well as slower cardiovascular recovery under the acute stressor.

Regarding anger, Suarez, Kuhn, Schanberg, Williams, and Zimmermann (1998) preselected men from the top and bottom quartiles on the Cook–Medley Hostility scale. Compared with low-hostility men, high-hostility men who were harassed during a solvable anagram task showed higher blood pressure, heart rate, and forearm blood flow. In addition, norepinephrine, testosterone, and cortisol responses were also higher. These researchers also found similar results in that high-hostility women under harassment had greater cardiovascular responses than low-hostility women (Suarez, Harlan, Peoples, & Williams, 1993).

Some studies have found that stress and anger interact in their influences on cardiovascular reactivity (Benotsch, Christensen, & McKelvey, 1997; Davis, Matthews, & McGrath, 2000; Harralson, Suarez, & Lawler, 1997; Houston, Smith, & Cates, 1989; Smith & Brown, 1991; Smith & Christensen, 1992a; Suarez & Williams, 1989, 1990). For instance, Christensen and Smith (1993) randomly assigned high-hostility and low-hostility men to either a stressful self-disclosure condition or a nonstressful non-self-disclosure condition. They found that high-hostility men showed greater cardiovascular response during the stressful condition but did not differ from the low-hostility group during the nonstressful interaction.

Powch and Houston (1996) involved 109 women in a high-stress or a low-stress condition. The high-stress condition had each participant discuss a current social issue that was rated of high importance by the participant. Involved in this discussion was a confederate who was unpleasant and condescending in taking a strongly opposite position. The low-stress condition involved discussion of an issue of low importance and the confederate amicably expressing an agreeing view. Hostility was positively associated with change in systolic blood pressure in the high-stress condition but negatively related to change in systolic blood pressure in the low-stress condition.

As further proof of the importance of cardiovascular reactivity to health, some researchers have demonstrated a relationship between cardiovascular reactivity and cholesterol levels and immune function. Stoney et al. (1999b) found moderate associations between cardiovascular reactivity (blood pressure and heart rate) and total cholesterol when participants were under speech stress. With regard to immune function, Kiecolt-Glaser, Cacioppo, Malarkey, and Glaser (1992), Kiecolt-Glaser & Glaser (1994), and Herbert et al. (1994) reported that higher cardiovascular reactivity was related to higher immune system change. In addition, Matthews et al. (1995) discovered that high cardiovascular reactors to an experimentally induced public-speaking stress showed declines in cell immunity levels.

Summary

This selective review of the research literature provides clear confirmatory evidence that anxiety and anger are indeed hazardous to health. Over the years, anxiety and hostility have been consistently demonstrated to increase vulnerability to illness, impair the immune system, increase levels of cholesterol, prevent adjustment to chronic pain, increase risk of atherosclerosis, and even increase mortality from both cardiovascular disease and all causes. A number of hypotheses have been offered to explain this negative impact of anxiety and anger on health, with some attention to interaction between these emotional states and cardiovascular reactivity.

Various cognitive–behavioral interventions have been developed either for control of anxiety or for control of anger (Barlow & Lehman, 1996; Barrett, Dadds, & Rapee, 1996; Holten & Beck, 1994; Meichenbaum, 1985; Moon & Eisler, 1983; Novaco, 1975). Part 2 of this article offers brief information on one psychological intervention that has proven effective for both anxiety and anger control: AMT.

Part 2. AMT for Anxiety and Anger Control

AMT

AMT was the term originally given to the cognitive–behavioral intervention for anxiety management developed in 1971 by Suinn and Richardson. The fundamental principle is the importance of exposing individuals to anxiety arousal and then deactivating the emotional arousal through relaxation skills (Suinn, 1990, 1998; Suinn & Deffenbacher, 1988, in press). Although other interventions have some similar components, AMT is unique in that it is highly structured, is brief (six to eight sessions), and relies on exposure through visualization but is followed by relax-
ation, homework practice for generalization, self-monitoring, and gradual fading in of self-control skills.

A number of controlled studies have proven the value of AMT for anxiety-related disorders such as generalized anxiety disorder, phobic disorder, medical conditions associated with high anxiety, and performance anxiety (Cragan & Deffenbacher, 1984; Durham et al., 1994; Jorgensen, Houston, & Zurawski, 1981; Nakano, 1990; Quilen & Denney, 1982; Rose, Firestone, Heick, & Faught, 1983; Stanley, Beck, & Glassco, 1996; Vance & Watson, 1994; Van Hassel, Bloom, & Gonzales, 1982).

AMT and Anxiety

Two studies illustrate the effectiveness of AMT for generalized anxiety disorder and for posttraumatic stress disorder. Butler, Fennell, Robson, and Gelder (1991) provided treatment based on the AMT approach for 57 patients experiencing general anxiety for a duration of three years. Of interest are the results on the Beck Anxiety Inventory (Beck, Brown, Epstein, & Steer, 1988), the State-Trait Anxiety Inventory ( Spielberg, Gorsuch, & Lushene, 1970), the Cognition Checklist (for anxiety cognitions; Beck, Brown, Eidelson, Steer, & Riskind, 1987), the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), and the Dysfunctional Attitude Scale (for depressive thoughts; Rush, Weissburger, & Eaves, 1986).

Results for the various anxiety measures were as follows: For the Beck Anxiety Inventory, the AMT group baseline was 25.0; at three months posttreatment, their score was 18.0; and at six-month follow-up, their score was 15.0. For a wait-list control group, the baseline score was 26.1, and at three-month follow-up, their score was 26.4 (because the wait-list group was provided with treatment after three months, there were no data for six-month follow-up). For the State-Trait Anxiety Inventory, the corresponding scores for the AMT group were 57.2, 52.8, and 51.9, respectively, compared with 55.5 and 56.8, respectively, for the wait-list group. For the Cognition Checklist, the corresponding scores for the AMT group were 85.7, 71.9, and 75.1, respectively, compared with 98.1 and 99.2, respectively, for the wait-list group. Changes from baseline to posttreatment and from posttreatment to follow-up were significant for the AMT group on the Beck Anxiety Inventory and on the Cognition Checklist measures. Changes on the Cognition Checklist between the AMT group and the wait-list group were statistically significant.

Results for the depression measures also showed reductions in depressive symptoms as follows: For the Beck Depression Inventory, corresponding scores for the AMT group were 18.6, 14.2, and 12.7, respectively; for the wait-list group, scores were 17.7 and 19.6, respectively. For the Dysfunctional Attitude Scale, scores for the AMT group were 155.0, 135.3, and 136.5, respectively; scores were 160.4 and 163.5 for the wait-list group. Beck Depression Inventory and Dysfunctional Attitude Scale values for the AMT group were significantly different from those for the wait-list group. The reduction in depression was consistent with other similar findings using AMT to treat general anxiety but in which depression was not directly addressed (Cragan & Deffenbacher, 1984; Jannoun, Oppenheimer, & Gelder, 1982).

As a possible explanation for the decrease in depression, Salvatore and Deffenbacher (1996) determined that one side effect of AMT was an increase in self-efficacy, possibly due to the self-control aspect of AMT. They also found a decrease in negative cognitions along with an increase in positive cognitions following AMT. Perhaps these side effects help to explain the changes in depression among AMT-treated persons.

For posttraumatic stress disorder, Pantalon and Motta (1998), in a single-participant design, treated six Vietnam veterans who had been in heavy combat. Posttraumatic stress disorder was diagnosed using Diagnostic and Statistical Manual of Mental Disorders (3rd ed., rev.; American Psychiatric Association, 1987) criteria applied to a posttraumatic stress disorder interview schedule. Treatment was for six sessions for two hours each and included AMT and then cognitive therapy. The Clinician Administered Posttraumatic Stress Disorder Interview scale was used to measure intrusions and avoidance symptoms (Blake et al., 1988). Significant reductions were found for both intrusion and avoidance scores. For instance, the baseline intrusion frequency score went from 12.6 to 6.0, whereas the avoidance intensity score declined from 22.0 to 8.3.

AMT and Anger

Because both anxiety and anger are arousal states, I hypothesized that the deactivation approach of AMT should be effective with both. The first studies confirming the application of AMT to anger appeared in 1986 (Deffenbacher, Demm, & Brandon, 1986; Hazaleus & Deffenbacher, 1986). A number of controlled studies have subsequently supported the efficacy of AMT for anger control (Deffenbacher, Huff, Lynch, Oetting, & Salvatore, 2000; Deffenbacher, Lynch, Oetting, & Kemper, 1996; Deffenbacher & Stark, 1992). Thus, "Anxiety Management Training" seemed more properly called "Anxiety-Anger Management Training."

As an example, Deffenbacher et al. (1986) treated 29 participants selected on the basis of trait anger scores in the upper quartile on the State-Trait Anger Scale. Six AMT sessions were conducted with assessments at pretreatment, posttreatment, five-week follow-up, and one-year follow-up. Results for the AMT group and wait-list control group on trait anxiety were as follows: For the AMT group, scores were 28.00, 20.01, 18.78, and 17.89 at pretreatment, posttreatment, five-week follow-up, and one-year follow-up, respectively; the corresponding scores for the control group were 25.00, 24.09, 23.63, and 24.27, respectively. Thus, not only was AMT effective for anger control, but improvements were maintained over a one-year assessment period after treatment ended.

In a recent study using AMT with a cognitive component added, Deffenbacher, Filetti, Lynch, and Dahlen (2000) treated 55 persons who scored in the upper quartile on a measure of road rage (DAS; Deffenbacher, Oetting, & Lynch, 1994). In earlier research, high road rage was
associated with greater lifetime incidence of accidents, more loss of vehicular control, and higher frequency of damage to one’s car (Deffenbacher, Huff, et al., 2000). Hence, control of road rage would be an important objective for drivers. Results found reductions in anger levels at posttreatment and at four-week follow-up. Specifically, the DAS road rage scores for the AMT group were 128.06 at pretest, 111.84 at posttreatment, and 103.81 at four-week follow-up; for the wait-list control group, DAS road rage scores were 129.05 at pretest, 128.55 at posttest, and 126.61 at four-week follow-up.

Concluding Statement

Anxiety and anger have been proven to be hazardous to health in this selective review. These “terrible twos” increase vulnerability to illnesses, especially upper respiratory illness; compromise the immune system; increase lipid levels; exacerbate pain; and increase the risk of death from cardiovascular disease and from all sources of death. At least one psychological intervention, AMT, has proven equally effective for the control of both anxiety and anger. Although future research is needed to determine if anxiety–anger reductions through AMT also lead to direct health benefits, it is encouraging to note that the terrible twos can be managed.

REFERENCES


