

Anger: definition, health consequences, and treatment approaches.

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Abstract:

In this review, four main aspects of anger are discussed. First, the definition and measurement of anger is examined; second, the neurobiological basis for anger and its expression is outlined; third, health consequences are summarized; and fourth treatment approaches are outlined. Based upon this review, it is evident that anger is a very important emotion and is different from aggression, although many confuse one with the other. Anger, with or without aggression, has been shown to have devastating health consequences, but may be amenable to several treatment modalities, including medication approaches. Well-controlled prospective studies will be necessary to determine the best treatment approaches and their effects on health consequences in the future.

Anger definition

Emotions play an organizing role in an individual's experience of reality, sense of self, and orientation toward others (107). Anger is a complex emotion and occurs as a result of an interaction between one or more eliciting events, the individual's pre-anger state, appraisals of the eliciting events, and available coping resources (33). In the broader sense, anger is composed of interrelated elements of cynical beliefs and attributions, angry emotional states, and aggressive or antagonistic behaviors (90). In a narrower sense, however, anger describes the affective experience, which can range from mild annoyance to fury and outrage, and can be differentiated from hostility, which refers to a person's tendency to view the world in a negative, cynical fashion, or aggression, which is used to describe destructive and violent behavior. This triad of anger, hostility, and aggression has been referred to as the "AHA" (anger, hostility, aggression) structure (137) and is also summarized as the "ABC" structure of trait anger, i.e. "Angry Affect", "Behavioral Aggression", and "Cynical Cognition" (90).

Anger can be elicited by specific events, behaviors of others, and one's own behavior, or a combination of external events and anger-related memories. Anger can also be elicited by thoughts and feelings that are associated with memories or anticipation of anger. A pre-anger state is often critical for the expression of anger and consists of the immediate feeling preceding the anger situation. This pre-anger state frequently emerges when a person is in an aversive emotional or physical situation, his/her identity is challenged, or if expectations or desires are not met. The basis for the pre-anger state as well as the basis of an angry response is often entrenched in personal identity, frequently represents consequences of traumatic life history, and can be associated with both Axis I and Axis II disorders (118)

Factors affecting anger prevalence and presentation

The precise prevalence of anger related problems is unknown, however, in a large epidemiological study, about 15% of individuals reported extremely high hostility scores (119). Some investigators have shown that problems associated with anger do not seem to vary significantly with age (14) but are inversely related to education (125). Higher levels of trait anger have been reported in younger individuals, African Americans, and in males (125). A range of factors ranging from social to genetic has been found to significantly affect the prevalence of anger. There is a strong connection between observed aggressive parenting and subsequent aggressive parenting in the offspring generation, which seems to suggest that anger/aggression is passed on via parenting styles from one generation to the next (24). This finding is consistent with the observation that hostility aggregates in families (91). Both negative life events and lack of social support were associated with higher hostility scores (125). Some have raised the issue of gender-specific expression of anger. Specifically, physical and verbal antagonism was found more frequently in men while more women reported passive consent (12). More recent evidence points to a specific genetic influence on anger prevalence, e.g. relative to the L allele presence of any U allele in the tryptophan hydroxylase gene was associated with a higher likelihood of outward expression of anger (87), which has been replicated in a different sample (121). Thus, the modulation of anger is multifactorial and involves gender-specific effects, genetic background, and family environment.

Among the most frequently reported anger situations is road rage. Anger expressions during road rage have been categorized into verbal or physical aggressive expression (34). In a recent survey, about 50% of driving individuals reported being shouted at, cursed at or had rude gestures directed at them in the past year. About 7% of respondents were threatened with damage to their vehicle or personal injury by others. Moreover, nearly a third of respondents admitted to shouting or cursing at someone (130). Relative to other common daily activities, anger was more likely to occur while driving (108). After controlling for gender, age, driving frequency, and annual miles driven, verbal

expression, an angry/threatening driving subscale of road rage was significantly associated with hazardous driving behaviors including frequency of driving over the legal blood alcohol limit, receipt of tickets in the past year, and habitually exceeding the speed limit as well as being involved in a motor vehicle accident (MVA) (155). This is consistent with reports that in driving simulations, high anger drivers were twice as likely to become involved in a MVA while under a high stress situations (32).

Tools for the assessment of anger

There are many scales and assessment tools currently used in anger studies; however, only three of the more widely used measures will be discussed here. The most widely used anger scale, with extensive normative data and predictive validity, is a paper-and-pencil measure devised by Spielberger and colleagues: the State-Trait Anger Expression Inventory (STAXI) (47;137). The STAXI is a 44-item scale that measures anger experience, expression, and control (92). The STAXI has several factors: State Anger (S-Anger); Trait Anger Temperament and Reaction; and Anger-In, Anger-Out, and Anger-Control. More recently, a “feeling like expressing anger” factor has been added to augment the original model (47). The most frequently used distinction, Anger-Out versus Anger-In, refers to the tendency to focus angry expression outward on other people or objects versus directed inward towards oneself, respectively. Further analyses using this scale revealed seven additional forms of anger expression: Noisy Arguing, Verbal Assault, Physical Assault-People, Physical Assault-Objects, Reciprocal Communication, Time Out, and Direct Expression (35). Interestingly, the STAXI was not effective in accurately classifying subjects as high and low in self-report of aggressive behavior (152). Thus, measuring angry feelings may not appropriately predict aggressive behavior.

Alternatively, the Buss Durkee Hostility Inventory (BDHI)(18) has been used widely, and its reliability and factor structure have been validated repeatedly (11). While the BDHI has better reliability than other anger scales and possesses some ability to predict the experience of anger, most of the BDHI subscales do not measure specific states or behavior (13).

Finally, the Cook and Medley Hostility (Ho)(25) scale has been widely used in studies with cardiac patients (see below) and shows good convergent and discriminant validity. This scale primarily assesses suspiciousness, resentment, frequent anger, and cynical distrust of others rather than overtly aggressive behavior or general emotional distress. Moreover, subjects with high scores high exhibited a particularly unhealthy psychosocial risk profile (134).

Psychiatric comorbidity and anger

Drugs and Alcohol: High trait anger individuals report more drug and alcohol use (143). In women, hostility was associated with increased tobacco smoking, caffeine use, and the number of alcoholic beverages consumed (19). Moreover, individuals who report low levels of trait anger may be more resistant to the potentiating effects of alcohol on aggression (109). In youth, elevated levels of anger and irritability have been shown to predispose to subsequent use of alcohol and drugs (144;147).

Axis I: Anger and aggression are prominent in psychiatric outpatients (113). Relative to patients with anxiety disorders, depressed individuals had twice the prevalence of anger attacks, which was associated with more depressed symptoms (53). Others have found that increased levels of anger suppression have been associated with less improvement in depression (17). Anger problems have frequently been reported in individuals with PTSD (52). Higher prevalence of anger has also been reported for individuals with Attention-Deficit/Hyperactivity Disorder (116)

Axis II and Personality: Cluster B personality disorders contribute most prominently to the presence of both anger and aggression in psychiatric populations (113). For example, subjects with high levels of narcissism reported greater verbally expressed anger and males high in narcissism were more likely to express anger physically (93). In addition, anger has been linked to various personality traits and coping styles. For example, a higher level of anger as measured by the Ho scale was associated with increased levels of neuroticism, attentional overload, and interpersonal alienation (21). In contrast, self-control skills were inversely related to hostility, anger, and aggression (58). High-anger-arousal subjects were found to score lower on socialization, self-control, tolerance, psychological-mindedness, and flexibility (10). Moreover, in interpersonal conflict situations men with high levels of hostility responded with significant increases in self-reported anger and anxiety or overt hostile behavior and saw the disagreement-engendering behavior as more intentional (135).

High impulsivity has also been related to high expression of anger feelings (43). Assertiveness and fear play an important role in the expression of anger. For example, individuals who express a high level of assertiveness expressed overt forms of more anger and aggression, whereas those with low level of assertiveness experienced more covert anger (38). Whereas fearful people expressed pessimistic risk estimates and risk-averse choices, angry people expressed optimistic risk estimates and risk-seeking choices (83). High Ho individuals report low covert self-esteem, avoid seeking or accepting social support, experience anger that is excessive and that occurs in a wide variety of situations, and suppress expression of anger, these individuals tend to drink more alcohol and drive a car more frequently after drinking and to have greater relative weight (60). Finally, very low ratings for spirituality was often associated with anger, pointlessness, selfishness, abandonment, and loneliness (54). In combination, although not a single personality factor predisposes to a high expression of anger, converging evidence suggests that neuroticism (a trait

measure of fear), narcissism, impulsivity, low spiritualism is associated with higher levels of anger.

Neurobiology of anger:

The somatic marker hypothesis proposed by Damasio and colleagues (26) is a useful conceptual approach to understand the neurobiology of emotions in general, and anger specifically. The key idea is that “marker” signals, which are brain representations of “body states”, have critical influence on how individual respond to external stimuli. This influence takes place at multiple levels of operation, some of which occur overtly or consciously and some of which occur covertly or non-consciously. The marker signals arise as the result of bioregulatory processes, i.e. resolving the differences between information about the “body state” from the peripheral nervous system and the brain generated expectation of the current state. The markers are called somatic because they relate to body-state structure and regulation even when they do not arise in the body proper but rather in the brain's representation of the body (27). This view of multi-level bioregulatory processes underlying the modulation of anger and other emotions is consistent with some of the special, attentional features of emotional processing, i.e. the automaticity of fear and anger reactions, hyper-reactivity to minimal threat-cues, and the evidence that the physiological responses in anger may be independent of slower, language-based appraisal processes (78). Some investigators have posited that impulsive anger and aggression arise as a consequence of faulty emotion regulation (30). Particular emphasis has been placed on the prefrontal cortex, anterior cingulate, parietal cortex, and the amygdala as critical components of the circuitry that may be dysfunctional in people with anger-related problems (29).

In a recent review of the functional neuroimaging literature, the medial prefrontal cortex was found to have a general role in emotional processing. Whereas fear specifically engaged the amygdala, and sadness was associated with activity in

the subcallosal cingulate. The emotional induction by visual stimuli activated the occipital cortex and the amygdala. In comparison, induction by emotional recall/imagery recruited the anterior cingulate and insula. Finally, emotional tasks with cognitive demand also involved the anterior cingulate and insula (112). Thus, a circuitry comprising the amygdala, anterior cingulate, and insula may be the key structures for the assessment and expression of anger.

The study of the brain systems underlying the evaluation and expression of anger has been based primarily on how individuals view and process faces. The majority of the individuals when asked to look at a human face with standard emotional expressions choose to describe feeling states as the message, such as disgust, fear, sadness, happiness, and surprise. In comparison, anger display tends to elicit the expression of behavioral intention or action request, which is consistent with the view that facial expressions communicate feelings, intentions, and wishes (59). In experimental situations, facial displays of anger are often used as communicative tools even though there is no audience and the individual is not aware of it (67). Other studies have looked at how angry behavior is acquired. For example, anger responses can be acquired via associative learning even when the individuals are unaware of the conditioning process (41). The recognition of anger appears to depend on the intact right inferior parietal cortex and on the right mesial anterior infracalcarine cortex (2).

The amygdala plays an important role in triggering knowledge related to threat and danger signaled by facial expressions (3). Bilateral amygdala activation is found during processing of threat words (62). Among the basic emotions, amygdala damage disrupts effective processing of anger and fear, in particular, the amygdala is critical for multi-modal appraisal of these emotions (126). Others have emphasized that the amygdala, in conjunction with other brain areas, supports the normal appraisal of auditory signals of danger (4). Apart from the amygdala, other cortical areas provide important top-down modulatory functioning for the expression of anger. For example, both anxiety and anger

conditions were found to increase cerebral blood flow in left inferior frontal and left temporal pole regions (75). Some investigators have proposed a specific role of the posterior part of the right gyrus cinguli, and the medial temporal gyrus of the left hemisphere has been ascribed to the processing of angry faces (138). However others have found anger to be associated with activation of the left orbitofrontal cortex, right anterior cingulate cortex affective division, and bilateral anterior temporal poles (37). This finding is consistent with brain activation patterns associated with transgressions of social norms involving systems previously found to be related to angry responses, e.g. the lateral orbitofrontal cortex and medial prefrontal cortex (9). In comparison, guilt, an emotion often associated with anger, blood flow increases occurred in anterior paralimbic regions of the brain: bilateral anterior temporal poles, anterior cingulate gyrus, and left anterior insular cortex/inferior frontal gyrus (127). To summarize, the appraisal and expression of anger involves a distributed neural system, which involves limbic, para-limbic, and cortical areas. Although several studies have been conducted using facial processing to assess anger-related neural circuitry, there is a need to extend these findings to other situations such as cue-induced anger. Moreover, it is unknown whether this distributed neural system is altered in individuals with anger problems.

Health consequences associated with anger

Early investigations of health consequences associated with high trait anger were focused only on the cardiovascular system, but new evidence shows adverse health consequences on other important areas of functioning, which include almost every major organ system.

Coronary artery disease and myocardial infarction (MI):

Initially, increased MI prevalence had been linked to Type A personality, however, subsequent analyses supported the hypothesis that only anger and

hostility may be the critical aspects of the Type A pattern in predisposing individuals to risk of coronary artery disease (36). Those individuals with high scores on the MMPI Ho scale had greater mortality risk, which remained significant even after controlling for age, sex, physician's ratings of functional health, smoking, cholesterol, and alcohol intake (7).

Other investigators have proposed that poor health habits of high hostility individuals may be another pathway from high anger to subsequent disease (82). For example, in both men and women, measures of hostility were positively associated with increased cholesterol intake and less vigorous physical activity. Among women, hostility measures were positively related to animal fat intake and negatively related to fiber intake (97). Moreover, a multivariate analysis in women revealed that 25% of the variance of improper health maintenance behavior was accounted for by hostility, low education, and the combination of high anger experience and outward expression of anger (70).

There is now robust empirical evidence linking emotional disturbances such as anxiety, depression and anger to coronary heart disease (146). Hostile affect and depression predict long-term cardiac-related mortality following an MI independently of each other and independent of cardiac disease severity (48). In addition, persuasive clinical evidence indicates that anger evokes physiological responses that are potentially life threatening in individuals with coronary artery disease. In combination, emotional stress, anger, and worry have a profound influence on the severity, frequency, and treatment responsiveness in coronary artery disease (50). For example, relative to individuals with low levels of anger, subjects with the highest level of anger in a coronary high-risk group were associated with a 3 times increased risk of premature coronary vascular disease, including 3.5 times higher risk of premature coronary heart disease and 6.4 times higher risk of premature MI (22). Other risk estimates, after controlling for age, education, body mass index, exercise, smoking history, and alcohol consumption, indicate that hostility was associated with a 1.3 to 2.2 increased

risk for a history of coronary bypass surgery or coronary angioplasty and a history of MI in high-risk women (76;157). In an attempt to quantify the acute effects of anger on coronary artery disease, investigators monitored subjects continuously and found that during a 1-hour period after an episode of anger, with an intensity of at least "very angry," the relative risk of having an MI was 9.0 times higher. When restricting the analysis to subjects who were identifying an anger episode within the hour, the trigger risk was found to be 16 times higher (96). In evocative situations, harassment produced increases in self-rated anger, irritation, and tension, but it was only among those subjects with high Ho scores that increased anger and irritation were associated with enhanced cardiovascular arousal (140). Others have suggested that situations, which evoke suspiciousness and mistrust, rather than anger in general, may be critical for the increased blood pressure reactivity among angry individuals (154).

However, some studies have been unable to replicate the association between hostility and coronary artery disease mortality, morbidity, or total mortality either before or after adjustment for baseline risk factors (57). For example, responses of MI patients and controls to questions about stair climbing, irritability and mood were virtually identical (8). Some have proposed that social isolation, a poor self-rated health status and ventricular irritability are equally strong predictors of mortality in a group of patients with ischemic heart disease (106). Reviewing the literature, Smith et al. (132) emphasized that there are important inter-individual differences among angry persons and that the social context plays an important role in the effects of hostility on health. Finally, aside from coronary artery disease, anger can affect the heart in other ways. For example, anger can trigger ventricular arrhythmias (77) and can lead to greater heart rate reactivity to stress (114).

Hypertension:

Several studies in the late 1970s related levels of anger to hypertension (85;86;88;89). Both, hostility and anxiety may precede the diagnosis of high blood pressure (136) and correlate with concurrent blood pressure levels (69). The effects of anger may be particularly pronounced in individuals with more labile blood pressure (64;128). Compared to low hostility subjects, individuals with high levels of anger displayed larger systolic and diastolic blood pressure responses (133). Conversely, subjects, who show an association between heart rate and mood variations were more anxious, reported more anger, and had higher systolic blood pressures (71). In addition, perceived stress affects both systolic and diastolic blood pressure as well as heart rate (79). Even in children, multivariate analyses revealed that expressive hostility was positively associated with blood pressure (151).

However, some studies have not confirmed the relationship between hostility and increased blood pressure (123;131). This may be due to individual differences such as physiological reactivity, family history, race and gender (40), specific anger styles, or how subjects cope with anger. For example, African Americans relative to Caucasians show longer-lasting blood pressure reactivity to anger (49). Moreover in subjects with a parental history of hypertension, systolic blood pressure reactivity was associated with low irritability, while for persons without a parental history of hypertension, systolic blood pressure reactivity was associated with high scores for irritability (72). In another study, high levels of antagonistic hostility but not neurotic hostility were significantly associated with greater systolic blood pressure and poorer recovery to harassment (141). Similarly, individuals who show a high outward expression of anger showed exaggerated diastolic blood pressure response in contrast to those participants who “held their anger in” (142). Also, when monitoring ambulatory indices of cardiac function, subjects who were high in both defensiveness and hostility showed heart rate responses approximately 10 beats per minute higher and increases in diastolic blood pressure when compared to subjects who were high in hostility but low in defensiveness (65). Defensiveness during anger episodes

was positively related to diastolic blood pressure reactivity (110). Finally, relative to individuals who do not talk about their anger, those who do have less problems with systolic blood pressure, diastolic blood pressure, body mass index, exhibit higher levels of aerobic exercise, perceive health as more important and show a better health status (148). In summary, there is moderate support for psychological factors as predictors for the development of hypertension and anger appears to be the strongest predictor (122).

Stroke:

Several recent studies have shown a highly significant association between anger and risk for stroke. For example, men who reported the highest level of expressed anger relative to those with low levels of anger were at twice the risk of stroke even after adjustments for age, resting blood pressure, smoking, alcohol consumption, body mass index, low-density and high-density lipoprotein cholesterol, fibrinogen, socioeconomic status, history of diabetes, and use of antihypertensives. Additional analyses showed that these associations were particularly evident in men with a history of ischemic heart disease. In these individuals, outwardly expressed anger predicted greater than 6-fold increased risk of stroke after risk factor adjustment (42). In another study, individuals younger than 60 years of age with high trait anger were at 3-fold higher risk for hemorrhagic and ischemic strokes relative to those who reported having low trait anger (156). Finally, anger can also be a consequence of a stroke. Specifically, inability to control anger or aggression has been closely related to lesions affecting frontal-lenticulocapsular-pontine base areas (74).

Metabolic processes:

Anger levels also affect metabolic processes. For example, individuals with a general tendency to either always express or always inhibit the expression of anger show higher triglyceride reactivity, relative to those with a more flexible

style of anger expression (46). In women, high hostility scores are associated with greater body mass index, higher levels of serum triglycerides, fewer high-density lipoprotein cholesterol, poorer self-rated general health, and less education (23). Moreover, individuals with impulsive anger-out had heightened glucose levels and a more negative lipid profile of high total serum cholesterol, low density lipoproteins, and triglyceride levels, as well as heightened glucose levels (129). In a multivariate analysis, only hostility levels predicted low high-density lipoprotein cholesterol levels (98). Finally, the disposition to experience and express anger when frustrated, criticized, or treated unfairly has been related to increased total serum cholesterol and to low-density lipoprotein levels (115).

Pain:

Anger stands out as one of the most salient emotional correlates of pain, even though past research has been largely confined to the study of depression and anxiety (45). Multiple regression analyses revealed that a style of inhibiting the expression of angry feelings was the strongest predictor of reports of pain intensity and pain behavior among a group of variables including demographics, pain history, depression, anger intensity, and other styles of anger expression. Moreover, anger intensity contributes to predictions of perceived pain interference and pain-related reduction in activity level (73).

Some have argued that pain adversely impacts mood rather than considering negative mood as a predisposing factor in the development of chronic pain (51). Others have shown that pain threshold and tolerance were lower in individuals with increased anger (68). Consistent with this view, anger management style and hostility contribute to the exacerbation of chronic low back pain (16). It has been proposed that anger-in and anger-out affect pain sensitivity through different mechanisms, i.e. only the effects of anger-out may be mediated by endogenous opioid dysfunction (15). Finally, anger toward oneself was

significantly associated with pain and depression, whereas only overall anger was significantly related to perceived disability (105).

Individuals who report problems with headaches relative to those without have higher scores measuring the tendency to “hold anger in” even after controlling for levels of trait anger, depression, and anxiety (99). Others have reported that subjects with migraine reported elevated aggression-hostility (20). These findings suggest that anger-related coping strategies are closely associated with self-report of pain and headaches.

Cancer:

Extremely low anger scores have been noted in several studies of patients with cancer. Such low scores suggest suppression, repression, or restraint of anger. Some investigators have suggested that suppressed anger is a psychological risk factor for the development of cancer, and also a factor in its progression after diagnosis (149). Behavioral oncologists have similarly attempted at conceptualizing a "Type C" or biopsychosocial cancer risk pattern, as they have noted the denial and suppression of emotions, in particular anger. This personality profile has been referred to as "pathological niceness" and is characterized by avoidance of conflicts, exaggerated social desirability, harmonizing behavior, over-compliance, over-patience, as well as high rationality and a rigid control of emotional expression. This pattern, usually concealed behind a facade of pleasantness, appears to be effective as long as environmental and psychological homeostasis is maintained, but collapses in the course of time under the impact of accumulated strains and stressors, especially those evoking feelings of depression and reactions of helplessness and hopelessness (6).

This proposed “Type C personality” is consistent with findings in a longitudinal study of anger among cancer patients and their caregivers, who showed low,

stable anger scores for both patients and their caregivers (145). Women patients with cancer reported lower scores than healthy women on total hostility, but men patients reported higher scores than healthy men. Introverted hostility was increased in both male and female patients, but due to different patterns: in women, the increase was due to lower scores on extroverted hostility subscales, especially acting-out hostility, whereas in men, the increase was due to higher scores on introverted hostility subscales, especially guilt (5). Thus, one of the main characteristics of a personality prone to cancer is either the repression or the inability to express or feel anger (84). However, others have found that in a large epidemiological study, overall emotional control and emotional control of anger and of anxiety were not convincingly related to cancer risk (150), or cancer outcome (104). In summary, these findings indicate that both low and high anger may have adverse health effects. However, future studies are necessary to better delineate the effects of anger or the lack of expression of anger on cancer.

Other Health Effects:

Anger levels have been related to several biochemical and molecular markers, which are relevant for key disease processes. For example, high levels of anger have been related directly to elevated cortisol secretion during normal daily activities (153). On a cellular level, hostility and aggression are associated with an increased expression of cytokines implicated in coronary vascular disease (139). Similarly, natural killer cell activity was significantly higher in the CHD group than the normal control and was significantly elevated by the suppression of anger and negative emotions (63).

Treatment and anger

Clinicians and researchers agree that anger represents one of the most challenging emotions encounter in psychotherapy and treatment (100). An

important impediment to the future success of anger management is the failure to fully address the issue of treatment readiness (61). Systematic Desensitization has been used in various studies to address problems of anger dyscontrol (103;117). Its efficacy, however, has been questioned (1).

Several studies have been published that utilize cognitive behavioral strategies (56;94). Using this treatment approach, investigators found that subjects in a negative-thought- reduction and meditation group versus placebo showed improvement in trait anger, anger aroused through high-anger situations, anger scores across a wide variety of situations, unconstructive coping, and anger measured through physiological symptoms (39). Moreover, the gains made through intervention were maintained at a 6-week follow-up (39). Anger management treatment subjects exhibited significantly more assertiveness skills and lesser diastolic blood pressure reactivity at post treatment than their control counterparts. This was demonstrated during a confrontive interaction but not during the neutral role-play interaction (80).

Others have proposed relaxation techniques as a main focus in anger management (102;111). Relaxation treatment combined with education about good psychological health maintenance behavior lowered blood pressure more than did hygiene alone. Neither treatment favorably affected a paper-and-pencil measure of anger but relaxation did lower anger-hostility on a new cognitive assessment procedure (31). In comparison, the main intervention principle in emotionally focused therapy is attention to the subject's present subjective experience. This type of therapy first establishes an alliance with the patient, evokes core emotions and explores bad feelings, and aids in the restructuring of maladaptive emotion and integration of new information (107). Finally, from a buddhist perspective, anger management has been divided into several steps which include taking responsibility, increasing awareness, understanding anger as well as the processes of reflection, decision-making, and relaxation (81).

Aside from traditional psychotherapeutic approaches, investigators have examined other health-maintenance behaviors as they relate to anger. For example, exercise has been found to be useful in the treatment of anger. In a cross-sectional study, individuals who exercised at least two to three times a week experienced less depression, anger, cynical distrust, and stress than those exercising less frequently or not at all (55). In another study, participation in the martial arts is associated, over time, with decreased feelings of assaultive and verbal hostility (28).

Several medications have been used to treat anger symptoms. In individuals with borderline personality disorder, some investigators have reported that the most striking finding was a clinically decrease in anger after treatment with fluoxetine (124). In another study, two thirds of depressed patients with anger attacks at baseline did not report anger attacks following fluoxetine treatment (44). Others have summarized various treatment studies with fluoxetine and have concluded that this medication may be useful in moderating anger as it occurs as part of the symptomatology of a number of Axis I and Axis II disorders (120). More recently, nefazodone's effect on anger attacks in depression was related to widespread changes in 5HT₂ receptor binding potential (95). Finally, administration of the nicotine patch, compared with the placebo patch, has been shown to reduce anger in diary reports in high-hostile participants (66).

Future directions

Anger is an important emotion that has profound health consequences and is based on a complexly organized neural circuitry. Although anger plays an increasingly important role in our daily experience, surprisingly few studies have been carried out to systematically determine which treatment approaches are most effective. A key element in the search for an "effective" treatment for anger is the establishment of the motivation for change in the patient. This may offer a unique opportunity for medication approaches because individuals may not want

to exert the psychological effort to participate in individual or group anger therapies but may be ready to take medications in light of the obvious and well-documented health consequences of extreme anger. Nevertheless, future studies will need to examine which types of psychotherapy are most effective in bringing about lasting change. Also, it would be useful to define the interactional benefit of both medication and psychotherapy in the reduction of anger episodes and intensity. Thus, although compared to a review by Novaco (101) there are many more studies that support the adverse health consequences of anger, one could still support his view that many aspects of anger are still poorly understood.

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