

Contagion: A Theoretical and Empirical Review and Reconceptualization

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Abstract	235
A Theoretical and Empirical Review	236
Proposed Redefinitions of Contagion	266
Summary Implications and Conclusions	274
Appendix.....	277
References.....	279

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ABSTRACT. We have reviewed theories and research in the area of contagion with an emphasis on definitions of contagion. The review shows that a great deal of the confusion surrounding the term is due to the fact that the phenomena involved in contagion are extremely heterogeneous, yet they typically have been placed under homogeneous rubrics. Accordingly, we propose herein that contagion should be reconceptualized as a general type, *social contagion*, and three subtypes: *disinhibitory*, *echo*, and *hysterical*. In this article, we have distinguished social contagion and its subtypes from other types of social influence phenomena and reclassified theoretical and research articles under the newly proposed definitions. The proposed reconceptualization shows how contradictions in the literature can be resolved by distinguishing the type of contagion in question and provides the foundation for a more comprehensive and useful psychology of contagion.

SOCIAL INFLUENCE is one of the most historically central and widely studied areas in modern social psychology. In fact, social psychology can almost be defined as the study of social influence (Jones, 1985). During the last 50 years, topics such as conformity, obedience, persuasion, compliance, deindividuation, social norms, contagion, interdependence, leadership, reactance, social facilitation, social inhibition, social loafing, and vicarious learning have dominated both theoretical and empirical foci. A number of taxonomies of social influence also have been proposed, the most noteworthy of which are Kelman's (1958, 1961) compli-

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ance-identification-internalization trichotomy, and French and Raven's six bases of social power: coercive, reward, legitimate, referent, expert, and informational (French, 1956; French & Raven, 1959; Raven 1965; Raven & Rubin, 1983b).

Within the area of social influence, however, there exists a distressing lack of consensus regarding the use of some its most fundamental, key, and central concepts. Definitions have been vague, inconsistent, and often times contradictory (see Levy & Collins, 1989a, 1989b, 1990; Nail, 1986, for discussion). A thorough review and reformulation of the social influence literature therefore is called for. Although such an undertaking is beyond the scope of this monograph, an initial step would be to isolate and examine one area of social influence that is particularly muddled.

Perhaps the most striking example of such confusion is the phenomenon of *contagion*. Although considered as important conceptually as other types of social influence, historically it has not enjoyed a great deal of theoretical or empirical attention. Moreover, the term has been subject to imprecise, inconsistent, and incongruous usage. It is thus a ripe starting point for a larger examination of the social influence literature.

Our purpose in this article is five-fold: (a) to present a historical review of theories and research related to contagion; (b) to propose a new definition of contagion, along with definitions of three specific subclassifications of the term; (c) to distinguish contagion and its subcategories from other types of social influence; (d) to reclassify selected research studies in psychology under the newly proposed definitions of contagion; and (e) to show how the new definitions can resolve apparent contradictions in the literature.

A Theoretical and Empirical Review

Historically, contagion has been approached from a number of diverse disciplines, including sociology, social psychology, social learning, and psychoanalysis. What follows is a chronological review of the contagion literature, drawn from these schools of thought. To achieve our goal of a comprehensive review, we have chosen to include both theoretical and empirical studies (as well as definitions found in social science dictionaries and textbooks, which appear in the Appendix of this monograph). Although a chronological (rather than thematic or conceptual) presentation format has some inherent drawbacks (e.g., sometimes abrupt transitions), it serves to illustrate the lack of cohesion and integration that has plagued the literature. In fact, it is this confusion that serves as the impetus for our proposed reconceptualizations of contagion.

The review is limited to publications in which the term contagion (or direct derivations thereof) have appeared. In the interest of historical accuracy, the authors' exact usage of the terms are presented (Table 1), and the author's exact usage of the terms are italicized in the text. Theories and research studies that

TABLE 1
Chronological Listing of Authors, Year of Publication, and Original Term Usage

Author(s)	Year	Term usage
Le Bon	1895	contagion
Baldwin	1897	contagion of feeling contagion of crime
McDougall	1920	contagion of emotion
Freud	1922	contagion
Blumer	1939	social contagion
Redl	1949	contagion
Polansky, Lippitt, & Redl	1950	behavioral contagion echo contagion
Grosser, Polansky, & Lippitt	1951	behavioral contagion
Lippitt, Polansky, & Rosen	1952	behavioral contagion
Brown	1954	mass contagion
R. H. Turner & Killian	1957	social contagion
English & English*	1958	social contagion mass contagion group contagion
R. H. Turner	1964	contagion (theories)
Wheeler, Smith, & Murphy	1964	contagion
Smith, Murphy, & Wheeler	1964	behavioral contagion
Wheeler	1966	behavioral contagion
Wheeler & Caggiula	1966	behavioral contagion
Wheeler & Levine	1967	contagion of aggression
Wheeler & Smith	1967	behavioral contagion
Kerckhoff & Back	1968	contagion hysterical contagion
Chaplin*	1968	contagion
Ritter & Holmes	1969	behavioral contagion
Milgram, Bickman, & Berkowitz	1969	contagion
Stephenson & Fielding	1971	behavioral contagion
Eysenck, Arnold, & Meili*	1972	psychic contagion
Bandura	1973	contagion contagion of aggression
Wolman*	1973	contagion mass contagion
Russell, Wilson, & Jenkins	1976	behavioral contagion
Milgram	1977	contagion
Cohen, Colligan, Wester, & Smith	1978	contagious occupational psychogenic illness
Goethals & Perlstein	1978	contagion of aggression
Freedman & Perlick	1979	contagion
Freedman, Birsky, & Cavoukian	1980	contagion
Stang & Wrightsman*	1981	contagion

(table continues)

TABLE 1 (continued)

Author(s)	Year	Term usage
Colligan & Murphy	1982	contagion contagious psychogenic illness
Freedman	1982	contagion
Raven & Rubin*	1983a	social contagion hysterical contagion
Worchel & Cooper*	1983	behavioral contagion
Goldenson*	1984	contagion group contagion
Reicher	1984	contagion
Reber*	1985	behavioral contagion emotional contagion social contagion
R. H. Turner & Killian	1987	contagion
Crandall	1988	social contagion
Sears, Peplau, Freedman, & Taylor*	1988	contagious violence
Sullins	1991	emotional contagion
Levy	1992	social contagion disinhibitory contagion
Levy & Nail	(this monograph)	social contagion disinhibitory contagion echo contagion hysterical contagion

*Cited in the Appendix

address genotypically similar phenomena but do not explicitly use the term contagion are cited in a subsequent section of this article.

The Early 1900s

In his seminal book, *The Crowd* (1895/1903), Le Bon asserted that a crowd is governed by an emergent collective mind, which is characterized by inhibition of intellect, intensification and exaggeration of emotion, submission to authority, and extreme suggestibility. Le Bon maintained that a crowd is dominated by unconscious processes, at the expense of individuality. He postulated that despite the variety of peoples' conscious minds, their unconscious minds are very similar.¹

¹Freud (1922/1959) differentiated Le Bon's conception of the unconscious from his own. Although he agreed with Le Bon that the unconscious holds deeply buried features of the inherited "racial mind," Freud further identified and distinguished the "unconscious repressed" (p. 7 n), which Le Bon did not address.

According to Le Bon (1895/1903), individuals in a crowd display new psychological characteristics, which they had not previously possessed.² Among the causes of this phenomenon, he cited contagion and suggestibility, stating that suggestibility is an antecedent to *contagion*, and imitation is an effect of contagion. However, Le Bon did not offer a clear definition of contagion, nor did he present a coherent explanation for its occurrence. He wrote that, "contagion is a phenomenon of which it is easy to establish the presence, but that it is not easy to explain. It must be classed among those phenomena of a hypnotic order" (p. 33). Le Bon maintained that in a crowd, emotions and behaviors are contagious to such a degree that the individual readily sacrifices his personal interest to the collective interest. He believed that the power of contagion in a crowd is analogous to the spreading of microbes; he cited, for example, the suddenness of panics.

Baldwin (1897) discussed the *contagion of feeling* in terms of social suggestion and imitation. He wrote: "An emotion may sweep through a gathering of people with a strength altogether out of proportion to the occasion of it in the individual's ordinary thought or life" (p. 234). As an example of *contagion of crime*, Baldwin noted that newspaper reports of a peculiar form of suicide stimulates others not only to kill themselves, but also to adopt the identical peculiar form of self-destruction.

In *The Group Mind* (1920), McDougall described the manner in which individuals are carried away by a common impulse by means of the "principle of direct induction of emotion by way of the primitive sympathetic response" (p. 37), that is, *contagion of emotion*. This principle states that each instinct is capable of being excited in one individual by the expressions of the same emotion in another. McDougall used panic as a typical instance of emotion by contagion, stating that, "the essence of panic is the collective intensification of the instinctive excitement, with its emotion of fear and its impulse to flight" (p. 36). Thus, fear in a group is heightened to enormous proportions through contagion.

From a sociological perspective, Blumer (1939/1951) viewed social contagion as an elementary and spontaneous form of collective behavior. He defined *social contagion* as "the relatively rapid, unwitting, and nonrational dissemination of a mood, impulse, or form of conduct" (p. 176). As examples, he cited the spread of crazes, manias, fads, financial panic, and patriotic hysteria. He stated further that in its more extreme forms, it takes on the character of a social epidemic, such as the dancing mania of the Middle Ages (see Hecker, 1837/1970; Major, 1955). Blumer maintained that social contagion is likely to take place when collective excitement is intense and widespread, and that it is able to attract and infect individuals who began as detached and indifferent spectators and by-

²In his critique of Le Bon, Freud (1922/1959) maintained that individuals in a group cast off the repressions of their unconscious instinctual impulses. Thus, the characteristics to which Le Bon referred are not "new"; rather, they are manifestations of pre-existing forces in the unconscious.

standers. Blumer explained social contagion in terms of a "circular reaction," namely, interpersonal stimulation that assumes a circular form, in which individuals reflect each other's feeling-states.³

The 1940s and 1950s

Redl (1949) described *contagion* as "the 'spread' of behavior from one person to another or to a whole group" (p. 315). According to Redl, such behavior is picked up nearly automatically by other group members. He asserted that conscious or unconscious intent is not necessary on the part of the individual who initiates contagion.

Redl (1949) proposed several hypotheses regarding group psychological factors that determine contagion. First, he hypothesized that the probability of contagion is positively correlated with the status of the initiator (i.e., the person whose behavior is being imitated). Second, the probability of contagion is greater when the behavior is related to high value ratings in the group code. Third, the most sweeping contagion is likely to occur under circumstances in which the behavior is liable to give vent to the suppressed needs of the largest number of group members with high group status. Fourth, although group size may not be related directly to contagion, both the formation of subgroups and democratic organizations tend to decrease the probability of contagion. Fifth, behaviors that have a high affinity to the group mood have a greater probability of contagion. Redl noted that these social factors "determine more the 'when' and 'how far' of a contagious effect than the 'why'" (p. 319).

According to Redl (1949), four personality factors are involved in the production of contagious events. First, the imitator is experiencing an approach-avoidance conflict; there is an impulse toward fulfilling a need, yet also suppression from the ego or superego. Second, there is a high degree of lability in the "personality balance"; the controls are only just strong enough to prevent release. If the controls were any stronger, there would be no contagion; if they were weaker, there would be no need for an initiator. Third, a similar type of strong urge toward impulse expression exists in the initiator. Fourth, the initiator openly acts out in the direction of impulse satisfaction, with an equally open display of a lack of fear or guilt. For imitators, the visualization of "fearless and guiltless enjoyment of what they really wanted to do sways their labile balance" (p. 321).

Redl also described an anti-contagion concept, the "shock effect," in which the observer's fear of loss of self-control results in increased reaction formation; that is, the contagious action is so guilt-producing that actively rejective behaviors

³Milgram (1977) later described circular reaction as a process involving an individual "who stimulates another in a crowd, sees or hears the intensified response which his behavior has produced in the other; he is in turn restimulated to a higher level of activity by the sight of his neighbor, and so on until ever high peaks of excitement are achieved" (p. 239).

are evoked. In contrast to contagion, in the shock effect, the recipient's intrapsychic balance favors superego success. Moreover, the initiator's expressed freedom from fear and guilt threatens the recipient by threatening his or her superego control. The process of the shock effect involves a desire for expression of a behavior, but a stronger reaction formation against it. The individual observes others openly and freely expressing the behavior; the observer then experiences a discrepancy between his or her range of self-acceptance and that of others, resulting in anxiety and an increase in reaction formation. Redl concluded his paper by calling for an integration of psychoanalysis and sociology in the study of contagion.

Polansky, Lippitt, and Redl (1950) identified and contrasted two major types of social influence, *behavioral contagion* and "direct attempts at influencing." They defined a direct influence attempt as "an event where the actor deliberately and openly tries to evoke a particular response in the recipient" (p. 321). By contrast, in contagion, the initiator does not openly communicate an intention to influence others. In addition, contagious influence always involves similarity in behavior between the actor and recipient; such is not the case with direct influence attempts.

Polansky et al. (1950) operationally defined behavioral contagion as

an event in which a recipient's behavior has changed to become "more like" that of the actor or initiator. This change has occurred in a social interaction in which the actor has not communicated intent to evoke such a change. (p. 322)

Polansky et al. also delineated the related phenomenon of *echo contagion* as a situation in which an individual "... [does] not imitate spontaneously but only after the number of [individuals] who have already been affected by contagion seems to him adequate to show that the particular behavior is clearly group-accepted" (p. 338). Thus, the process cannot occur unless there is at least the possibility of communication between initiator and recipient.

Polansky et al. (1950) maintained that contagion occurs both in disorganized, mass group situations and also in more structured organizations, such as therapy groups; in fact, their interest in contagion grew out of their clinical experience with face to face groups. They stressed an interactional approach, asserting that a purely individual psychological approach is inadequate for understanding contagion. As a consequence, they pointed to the necessity for understanding the group psychological factors (i.e., conditions) under which contagion takes place.

Polansky et al. (1950) conducted one of the earliest field studies of contagion. Their primary research focus was on the relationship between contagion and status in the group, with the major hypothesis that influence in groups is a function of prestige. The researchers collected observational data on direct attempts at influencing, incidents of contagion, and status indicators. Their sample was comprised of "disturbed children" of lower socioeconomic backgrounds at a summer camp setting.

Results showed that children higher in prestige were more frequently the initiators of behavioral contagion and direct influence attempts. Further, although the higher prestige children were more susceptible to influence through contagion, they were better able to resist direct influence. Although a significant relationship between contagion initiation and attributed power was found throughout the observation period, the authors reported that the relationship between these two factors was not significant in an "experimental" situation.⁴ On the other hand, whereas no relationship was found between contagion initiation and impulsiveness during the observation period, this relationship was significant in the experimental setting (i.e., under stress). Here, impulsive children both induced contagion and were more susceptible to contagion.

No relationship was found throughout the camp observation period between the children's level of impulsiveness and contagion susceptibility. The researchers also found no relationship between frequency of attempts at influencing and the percentage of these that were successful. They noted that the freedom to behave spontaneously may be a determinant of one's ability to initiate, or be susceptible to, behavioral contagion. They concluded that the likelihood of behavioral contagion is a function of four factors: (a) the security to act spontaneously; (b) attributed group position; (c) the possibility of communicating with the group; (d) the degree to which individual reactions represent a common need present in the group.

Two of the earliest experimental laboratory studies of contagion were conducted by Grosser, Polansky, and Lippitt (1951). Their definition of *behavioral contagion* paralleled closely that of Polansky et al. (1950), namely:

a social interaction in which a "recipient's" behavior changes to become "more like" that of another person, and where this change has occurred in a social interaction in which the "initiator" (other person) has not communicated intent to influence the behavior of the recipient. (p. 115)

The authors acknowledged that although the interpersonal relationship between the initiator and the recipient is an important variable in the process of contagion, their focus was on the function of the initiatory act itself as a stimulus for the recipient. They analyzed contagion from a Lewinian orientation (see Lewin, 1938; 1951).

According to Grosser et al. (1951), the initiatory act can serve two primary functions. First, in a situation where the recipient has an explicit goal but is not certain how to achieve this goal, the initiatory act may provide information regarding a course of action. In a second scenario, the recipient can possess a clearly

⁴The "experimental" situation described by Polansky et al. (1950), in fact, consisted of a single condition ("group frustration") with two blocking factors, namely, attributed power and impulsiveness. Since this procedure lacked a manipulated independent variable, it would be more accurately classified as a nonexperimental design.

perceived goal and means, but may be experiencing a conflict due to restraints against action toward the goal. In this case, the initiatory act may serve either: (a) to increase the attractiveness of the goal by demonstrating the need satisfaction present in it (i.e., raising the approach gradient); or (b) to reduce the strength of the restraints (i.e., lowering the avoidance gradient).

Grosser et al. (1951) noted that Redl (1942) originally conceptualized the process by which restraints are reduced as one in which responsibility for the act is "projected" onto the initiator after the recipient has acted. Grosser et al. expanded upon this idea, postulating that "projection" in this second scenario involves four stages. First, the recipient assumes that others define the situation in the same way that he or she does. Second, as a consequence, the recipient interprets changes in the behavior of others as signaling changes in the definition of the situation. Third, in a conflict situation, action by the other is perceived as representing the other's solution to the problem situation. Fourth, where the conflict is characterized by ambiguity as to the consequences of acting, the initiatory act of the other may be perceived by the recipient as signaling an absence of serious repercussions.

The purpose of the first Grosser et al. (1951) experiment was to investigate the hypothesis that where two individuals possess the same need but also experience restraints against the expression of that need, if one individual breaks the restraints, the other will follow suit. Subjects in this experiment were 39 boys, ranging in age from 12 to 14 years. Results supported the researchers' hypothesis.

Experiment 2 was designed to test the hypothesis that behavioral contagion may be related to changes in perception of consequences of acting. In this experiment, four factors were manipulated. Factor 1 (active vs. passive) consisted of the confederate's willingness to engage in the activity, namely, playing with a toy. Factor 2 (attractive vs. unattractive) was the valence of the activity. Factor 3 (high friendly vs. low friendly) was the friendliness of the confederate toward the subject. Factor 4 (high safety vs. low safety) was the likelihood of unpleasant consequences for engaging in the activity.

Results showed that the active condition produced significantly greater contagious behaviors than did the passive condition. Further, the high friendly condition yielded more exact contagions than the low friendly condition. No significant differences were found in the attractive versus unattractive and in the high versus low safety conditions.

An additional condition was examined to explain further the causes of the contagion effect produced by Factor 1. The authors concluded from this condition that the contagion effect was the result of both a decrease in the restraining force in the active condition and an increase in restraining force in the passive condition.

Lippitt, Polansky, and Rosen (1952) compared the findings of two investigations on behavioral contagion and direct influence processes in groups of children (for similar summaries, see Lippitt, Polansky, Redl, & Rosen, 1953, 1958). The first study was the previously cited study by Polansky et al. (1950); the second

study was a replication of that study, with the addition of a matched comparison group of middle-class, nondisturbed boys. Following their prior research, the authors defined *behavioral contagion* as:

An event in which a person's behavior is changed to resemble that of another person. This change occurs in a social-interaction situation in which the person acting as the "initiator" has not communicated intent to evoke such a change in the other. (p. 41)

Lippitt et al. (1952) contrasted behavioral contagion with "direct-influence attempts," which they defined as a social interaction in which one person consciously and deliberately tries to get another person to do something "... in such a way that the research observer is aware of the intent" (p. 41). Thus, the intentionality of the initiator's influence attempt is assessed from the perspective of an observer rather than of the recipient.

All of the major findings of the first study were confirmed in the replication. Specifically with regard to contagion, children tended to imitate the behavior of those to whom they had attributed high power; that is, high-prestige children were the most effective models for contagion. In the lower-class sample, children high in prestige were found to be more susceptible to influence through contagion than those with low prestige; however, this relationship was not found in the middle-class sample. The authors also reported that activity level is not an independent determinant of frequency of contagion initiation or of successful induction.

In his paper on mass phenomena, Brown (1954) discussed *mass contagion* in terms of social "fads" and "crazes" engaged in by large collectivities, such as classes and nationalities. According to Brown, mass contagion occurs under conditions in which large numbers of people, who are not gathered in one place, are neither attending to any common stimulus nor identified with one another. Brown considered both fads and crazes to be types of "mass folly." He noted that whereas fads are concerned with relatively trivial cultural phenomena (e.g., clothing fashions, linguistic colloquialisms), crazes may have serious personal and social consequences. As examples of crazes, Brown cited the dancing mania of the Middle Ages, the Salem witch trials, and Johnson's (1945) study of mass hysteria in "The 'phantom anesthetist' of Mattoon."

Sociologists R. H. Turner and Killian (1957) proposed that "milling" is the basis of *social contagion* and is the fundamental process through which a common mood is developed in a collectivity. They defined milling as "a search for socially sanctioned meaning in a relatively unstructured situation" (p. 59). That is, when an individual is placed in an ambiguous situation (i.e., one that is lacking in cognitive clarity), he or she seeks social cues in the reactions of others to help define the situation. Turner and Killian pointed out that, although milling can take many forms and comprise a number of different behaviors, it is essentially a communication process. They identified three specific consequences of milling: sensitization of individuals to each other, the development of a common mood, and the development of a collective definition of the situation.

Turner and Killian (1957) maintained that much can be learned about the principles underlying social contagion from studies of rumor. They noted that rumor, which is a form of milling in its primarily verbal aspect, has been the mechanism of social contagion most subjected to empirical investigation. They cited, for example, the work of Festinger et al. (1948).

Turner and Killian stated that immunity to social contagion is, to a large degree, a function of the extent to which a situation is subjectively well defined for an individual. In such a scenario, the person is not responsive to the tentative definitions advanced by others. Immunity to social contagion may also be related to the rigidity of attitudes and to the intensity of motivation in those individuals who do not succumb.

The 1960s

R. H. Turner (1964) discussed and evaluated three competing sociological theories of collective behavior: contagion, convergence, and emergent norm. According to Turner, *contagion theories* "explain collective behavior on the basis of some process whereby moods, attitudes, and behavior are communicated rapidly and accepted uncritically" (p. 384). These theories maintain that when individuals feel anonymous in a crowd, emotions are easily transferred, and self-control is relinquished to the crowd.

Turner cited five criticisms of contagion theories. First, these theories rely excessively on rare and extreme episodes of collective behavior; moreover, accounts that support instances of contagion tend to be reported by "untrained and horrified observers." Second, contagion theories make the dubious assumption that socialization is merely a thin veneer for a permanently primitive human nature. Third, mechanisms (e.g., suggestion) that are cited to explain contagion appear to be uninvestigatable empirically. Fourth, contagion is ill-equipped to explain shifts (e.g., cessation) in a collectivity's behavior. Fifth, contagion does not provide a description of group organizational structure.

In contrast to contagion theories, convergence theories "explain collective behavior on the basis of the simultaneous presence of people who share the same predispositions and preoccupations" (Turner, 1964, p. 384). The underlying assumption of these theories is that members of a collectivity share latent tendencies that predispose them to act in a similar fashion; in responding to a common stimulus, they merely reveal their true selves. Convergence theories, like contagion theories, view crowd behavior as unanimous, uniform, spontaneous, and irrational. Turner believed that these characteristics restrict greatly one's ability to account for certain crowd phenomena; moreover, he maintained that both theoretical approaches provide little predictive utility.

Turner's (1964) theory of emergent norms attempts to explain collective behavior in terms of intragroup processes. This perspective views collective behavior as "regulated by a social norm which arises in a special situation" (p. 384).

When individuals are exposed to an ambiguous situation, they turn to others around them for cues as to the appropriate course of action. This information is relayed through the process of "milling" (see R. H. Turner & Killian, 1957). Unfortunately, individuals' assessment of reality is biased by the salient characteristics of a few crowd members, which results in a collective illusion of behavioral unanimity. As examples of emergent norm theory, Turner cited the studies of Asch (1951) and Sherif (1935).

Turner (1964) outlined several important distinctions between contagion and emergent norm theories. First, whereas contagion sees crowd participant behavior as unanimous, emergent norm theories assert that there is differential member expression. Second, contagion views crowd emotion as arising spontaneously; emergent norm asserts that it is a consequence of "the imposition of conformity under the impact of a norm" (p. 390). Third, contagion is limited to arousing situations; emergent norm is equally applicable to subdued and to excited states. Fourth, contagion theories focus primarily on the transmission of the dominant emotion in a crowd, whereas emergent norm emphasizes the transmission of norms. Fifth, contagion does not offer an adequate explanation for the upper limits on crowd behavior; emergent norm provides such a reason. Sixth, contagion stresses the importance of member anonymity as an antecedent to crowd behavior; emergent norm maintains that crowd behavior is a function of members' recognizable identity. Turner concluded that despite some limitations to his approach, episodes of collective behavior are best characterized by the emergence of norms.

Wheeler, Smith, and Murphy (1964) conducted four experiments on the contagion of game-playing behavior, focusing on nonverbal imitation. The authors cited prior definitions of *contagion* (i.e., Polansky et al., 1950), but they did not offer a new definition of the term.

In a rather lengthy experimental procedure, subjects (groups of eight new Army recruits) were given a "general briefing" (actually, a cover story). An experimental confederate (posing as a new Army recruit) then escorted one subject at a time to the laboratory room, which contained various games (e.g., darts, shuffleboard) and magazines. The experimenter entered the room, stated that there would be a delay, and then departed. At that point, the confederate began a predetermined activity (e.g., playing darts), depending upon the particular experimental condition.

In a control condition, the confederate did not remain in the room with the subject; instead, he left with the experimenter. During the next 20-minute "waiting period," the subject's behavior was measured unobtrusively by means of a concealed microphone and a hidden closed-circuit television camera. The subject was then taken to a small room to fill out various personality tests. Finally, the experimenter took each subject to a soundproof room for a test in "auditory acuity" (actually, a modified Asch-type conformity experiment).

During the entire procedure, subjects were led to believe that the key test was the auditory acuity task, and that the waiting period in the lab room was not

a part of the experiment. For the first two experiments, the experimenter attempted to make the subject experience some restraints against game playing by never stating directly that it was permissible to use the game equipment; however, he did not state that game playing was prohibited. The four primary dependent variables were: (a) activities engaged in by the subject; (b) latency for the onset of each activity; (c) total time spent on each activity; (d) sequence of the subject's activities.

Results of Experiment 1 showed that contagion occurred whether the confederate engaged in a game that was of high valence (i.e., darts) or low valence (i.e., shuffleboard) to the subject. Other results indicated that mere activity (i.e., walking around the room) on the part of the confederate did not lead to game playing, and that contagion tended toward specificity of behavior.

To examine the role of competition as a possible explanation for the contagion effect observed in Experiment 1, in Experiment 2, equipment that permitted him to play the same game as the confederate was never available to the subject. Results indicated that contagion was not entirely due to a desire to compete in game playing, and that specificity of contagion was not necessary.

In Experiment 3, the experimenter expressly told all subjects that it was permissible to play with the games. With no restraints against game playing, contagion of a low-valence game (shuffleboard) was not produced. Experiment 4 failed to produce contagion of a high-valence game (darts) with no restraints against game playing.

Wheeler et al. (1964) concluded that the observed contagion was mediated by restraint reduction; specifically, the violation of the experimenter's implied prohibition was due to a reduction of restraints following the confederate's initiatory act. Contagion did not appear to be a function of increased needs to play with the games.

The authors discussed several ways in which an initiating act may reduce restraints. First, if one violates a prohibition, it is less noticeable as the number of violators increases. Second, the initiating act provides information that the prohibition does not actually exist or that it is not enforced. Third, once the prohibition has been violated by the initiator, the recipient views further violations as unimportant. Fourth, shared punishment is less frightening than individual punishment.

Wheeler et al. (1964) rejected the first three explanations as untenable for these experiments, but left the fourth open to question. Other results showed that subjects who responded to the confederate's game playing did not tend to be conformers in the Asch-type situation (i.e., there was no relationship between contagion and Asch-type conformity). Last, the authors concluded that the recipient's engaging in the specific behavior of the imitator is not necessary for the occurrence of contagion; rather, contagion may be said to occur if the behaviors belong to the same behavioral class (e.g., playing darts or playing shuffleboard).

Smith, Murphy, and Wheeler (1964) reported on the relationship of intelligence and authoritarianism to *behavioral contagion* and conformity, using data

obtained from the experiments conducted by Wheeler et al. (1964). In addition to measures of behavioral contagion and Asch-type conformity, subjects completed the 30-item California F scale (authoritarianism; Adorno, Frenkel-Brunswick, Levinson, & Sanford, 1950) and the Army GI (general intelligence) test.

Results showed no relationship between behavioral contagion and conformity. Similarly, no meaningful relationship was observed between contagion and either authoritarianism or intelligence. Other results indicated that, with intelligence partialled out, there was a significant positive relationship between authoritarianism and conformity; in contrast, partialling out authoritarianism, the correlation between intelligence and conformity was nonsignificant.

Wheeler (1966) advanced a theory of *behavioral contagion* and contrasted it with other types of social influence. He noted that while many psychologists associate the term contagion with the spread of an emotional climate, his focus was on overt behaviors, accompanied or not by the spread of emotions. His central theoretical tenet was that contagion is due to the lowering of the observer's avoidance gradient in an approach-avoidance conflict; that is, contagion is mediated by restraint reduction.

Wheeler (1966) noted that the definition of behavioral contagion offered by Polansky et al. (1950) does not adequately distinguish it from other types of social influence, such as Asch-type conformity. He defined behavioral contagion through the use of reduction sentences:

If the set of test conditions T_i exists, then contagion has occurred if and only if Person X [the observer] performs Behavior N (B_N) where T_i is specified as follows: (a) A set of operations has been performed on Person X which is known to produce instigation toward B_N in members of the class to which X belongs; (b) B_N exists in the response repertoire of X, and there are no physical restraints or barriers to prevent the performance of B_N ; (c) X is not performing B_N ; (d) X observes the performance of B_N by Person Y [the model]. (p. 180)

The implication is that if Person X is not performing B_N , then X possesses internal restraints against performing B_N . In other words, X is experiencing an approach-avoidance conflict. Further, X's observation of the performance of B_N by Person Y changes the relative strengths of the approach and avoidance gradients.

Wheeler (1966) differentiated behavioral contagion from four other types of social influence: conformity, pressures toward uniformity, social facilitation, and imitation. He stated that although contagion and conformity both involve conflict, they are differentiated with respect to the role that other individuals play in the conflict. In conformity, conflict is produced in the individual by the judgments of others. By contrast, in contagion, the individual experiences conflict prior to the presence of others.

Moreover, in contagion, the presence of others contributes to a resolution in the individual's conflict. Wheeler (1966) argued that there is essentially no difference between conformity and "pressures toward uniformity" (Festinger, 1954). "Social facilitation" is distinguished from conformity, contagion, and social pres-

tures by the absence of any marked conflict. Wheeler considered "imitation" a generic term that subsumes contagion, conformity and social pressures, and social facilitation.⁵

Wheeler (1966) derived a number of theoretical statements based on a review of studies dealing with contagion. First, regarding the specificity of response matching, the observer's behavior may or may not be an exact imitation of the model's behavior, depending upon the initial strength of the approach and avoidance gradients (Grosser et al., 1951; Wheeler et al., 1964). Second, with regard to the balance of approach and avoidance tendencies, the likelihood of contagion is greatest when the avoidance gradient is just slightly above the approach gradient (Freed, Chandler, Mouton, & Blake, 1955; Kimbrell & Blake, 1958). Third, with respect to consequences to the model for his behavior, the observer vicariously performs the model's behavior and vicariously experiences the consequences of the behavior; to the extent that the model is rewarded or not punished, the observer's avoidance gradient is lowered (Bandura, Ross, & Ross, 1963a; 1963b; Walters, Leat, & Mezei, 1963; Walters & Parke, 1964).

Fourth, regarding characteristics of effective models, to the extent that the model is perceived by the observer to be rewarded or not punished for whatever behavior he emits, the observer's avoidance gradient is lowered for whatever behavior the model performs (Lefkowitz, Blake, & Mouton, 1955; Lippitt et al., 1953; Polansky et al., 1950). Fifth, with regard to characteristics of the observer, persons most susceptible to contagion do not appear to be anxious, of low self-esteem, or authoritarian (Smith et al., 1964); beyond that, no general statements can be made.

Wheeler (1966) noted that no systematic research has been done with regard to the locus of restraints. He offered his predictions for three types of loci. First, when the restraints are group-derived and a member of that group performs the prohibited act, the behavior should spread quickly throughout the group. Second, when the locus of restraints is the observer's superego, the observer's fear is reduced to the extent that the model performs the behavior without manifesting guilt or fear. Third, when the locus of restraints is an authority figure, the most effective model is one who is not punished by the authority for performing the behavior (see, for example, Milgram, 1965).

Based on his review of the deindividuation literature, Wheeler (1966) predicted that in a contagion situation where the observer is not punished for his

⁵ It should be noted that "social facilitation," as Wheeler used the term, refers to situations in which "the performance of a more or less instinctive pattern of behavior by one member of a species will tend to act as a releaser for the same behavior in another or in others, and so initiate the same lines of action in the whole group" (Thorpe, 1956, p. 120). Wheeler noted that this is akin to the term "eliciting effect" of a model, from social learning theory (Bandura & Walters, 1963). Thus, Wheeler's conceptualization of social facilitation differs sharply from the one formulated by Zajonc (1965) namely, a process whereby the presence of others enhances the individual's emission of dominant responses.

behavior, the observer will like the model more than previously and will feel more similar to him on a number of dimensions; that is, the observer will be less individuated from the model. In his concluding remarks, Wheeler noted that theoretically, contagion need not be limited to socially undesirable behavior. He postulated, for example, that the brutal murder of Kitty Genovese could probably have been prevented if one individual had made a visible attempt to aid the victim; in such a scenario, fear would likely be reduced in other observers, who then would have acted.

The contagion of aggression was investigated experimentally by Wheeler and Caggiula (1966). Following the theorizing of Redl (1949) and Wheeler (1966), Wheeler and Caggiula defined *behavioral contagion* by the following conditions: "(a) an observer is instigated to perform a behavior, (b) the observer does not perform the behavior, (c) a model performs the behavior in question, and (d) the observer performs the behavior" (p. 1). Implicit in this formulation is the concept of restraint reduction.

The authors discussed the findings of deCharms and Wilkins (1963), who investigated the effects of a model's aggression on an observer's expression of aggression. In that experiment, a subject was insulted in conversation, and then was allowed, under two degrees of restraints against aggression, to communicate to the instigator, or to hear a model aggress against the instigator, or to be complimented by the model, or to neither communicate to the instigator nor to hear the model. The greatest amount of aggression by the subject against the instigator was produced when the model aggressed against the instigator.

Wheeler and Caggiula (1966) designed their study to evaluate three competing explanations (vicarious frustration, conformity pressures, and contagion) for this finding. The design of their experiment was similar to that of deCharms and Wilkins, but with additional conditions. Essentially, the subject was instigated to aggress against a target and was exposed to a model who aggressed against the target. The target's response to the model's aggression was varied in order to test the vicarious frustration hypothesis. The vicarious frustration hypothesis predicts that the degree of aggression by the subject toward the target would be affected by the extent to which the target yields (i.e., retreats) to the aggressive model. Thus, if the target does not yield (e.g., he counteraggresses), then the subject would experience more frustration and therefore exhibit greater aggressiveness toward the target.

Two additional conditions were added to assess the conformity and contagion explanations. The conformity hypothesis predicts that the model's aggressive behavior would be sufficient in itself to account for the subject's degree of aggression toward the target, irrespective of the subject's instigation to aggression. The contagion hypotheses predicts that the subject's aggressive behavior would be a function of both the model's aggressive behavior and the subject's instigation to aggression.

The sample was comprised of enlisted men in the U.S. Navy. Each subject was taken individually to a soundproof room equipped with speakers and a microphone and was informed that he would be exchanging opinions on six topics (e.g., religion, sex, liquor) with two other enlisted men (actually, standardized tape recordings of two confederates). With the exception of one condition, the target expressed socially deviant opinions, which were designed to instigate aggression. In all conditions, the model's opinions were identical and always socially desirable.

Subjects were randomly assigned to one of six experimental conditions: (a) model aggresses against target, target does not comment; (b) model aggresses against target, target counter-aggresses against model; (c) model aggresses against target, target retreats; (d) model disagrees with target, target does not comment; (e) subject cannot hear model, target does not comment; (f) target does not express socially deviant opinion, model aggresses toward target, target does not comment. The dependent variable was verbal aggression expressed by the subject toward the target.

Results showed, first, that when the subject was instigated to aggression and was exposed to an aggressive model (Conditions A, B, and C), the amount of yielding by the target did not affect the subject's aggression. This finding contradicted the vicarious frustration hypothesis. Second, the combination of instigation to aggression and exposure to an aggressive model produced a greater frequency of aggression by the subject than a simple additive model of these two factors would predict. According to the researchers, this finding supported a contagion explanation over that of conformity. Wheeler and Caggiula (1966) concluded that these results provide powerful confirmation of the hypotheses derived from the contagion framework. They noted that "Social psychology has long relied on 'push-pull' explanations of social influence. Perhaps we will find that much social influence is simply releasing the individual from social 'pushes' and 'pulls'" (p. 9).

Wheeler and Levine (1967) investigated the effects of observer-model similarity in the contagion of aggression. They used the term *contagion of aggression* to describe the effect whereby "permitting an angered or frustrated individual (the observer) to watch an expression of aggression by someone else (the model) increases the probability that the observer himself will aggress" (p. 41). Wheeler and Levine discussed two theories' differential predictions regarding the effect of observer-model background similarity on the contagion of aggression. One theoretical position, based on social comparison processes (i.e., Festinger, 1954), predicts that observer-model similarity should heighten contagion of aggression. The other position, based on contagion theory (i.e., Wheeler, 1966), predicts that observer-model similarity is irrelevant in the contagion of aggression.

The sample was comprised of members of the U.S. Navy. The experimental procedure was similar to that used by Wheeler and Caggiula (1966); that is, each subject engaged in a "discussion" with two tape-recorded confederates. In the

experiment, one confederate (the target) expressed socially undesirable opinions designed to anger the subject; the other confederate (the model) then aggressed against the target. Differential observer-model similarity was created through the use of short biographical inventories, which the subject (and supposedly the two other "subjects") completed prior to the experiment.

Before beginning the discussion, each subject read the background inventories of the two confederates and filled out a "personal impression questionnaire," which assessed similarity and liking toward the other two men. After the discussion period, the subject was administered this questionnaire again. The dependent variable was the subject's aggression toward the target, as rated by two naive observers.

Subjects were randomly assigned to one of two experimental conditions. For the *similar condition*, the model's inventory was made to appear quite similar to the subject's inventory (in terms of age, family size, ordinal position, parents' ages, home state, hobbies, favorite sports to watch and to participate in, hometown size, marital status, religion, and race). For the *dissimilar condition*, the model's inventory was made very dissimilar on these characteristics. For both conditions, the biographical inventory for the target was tailored to be fairly similar to the subjects' inventories (i.e., an 18-year-old, single, White, Protestant from a medium-sized town who likes cars and sports) was held constant.

Contrary to their expectations, the researchers found that subjects (observers) who were exposed to a dissimilar model aggressed more toward the target than those who were paired with a similar model. They explained this effect in terms of "unexpected support": When an observer and a model are of similar backgrounds, the model's behavior provides very little new information regarding the situational norms. However, when the model is dissimilar, the observer does not expect his reactions to be the same, so that when the model responds negatively toward the target, this indicates to the subject an expected response. In short, the observation of a dissimilar aggressive model provided greater justification for the subject's own anger.

Other results showed that, whereas subjects' attitudes toward the similar model changed only slightly and inconsistently as a consequence of the discussion, their attitudes toward the dissimilar model changed markedly in the direction of more positive feelings. Wheeler and Levine (1967) concluded by hypothesizing that disagreement by someone similar in background will reduce one's confidence more than disagreement by a dissimilar other; further, agreement by a dissimilar other will increase one's confidence more than agreement by a similar other.

Wheeler and Smith (1967) studied experimentally the contagion of aggression as a function of response consequences to the model. They adopted the same definition of *behavioral contagion* used by Wheeler and Caggiula (1966), again conceptualizing the phenomenon in terms of restraint reduction. The authors noted that according to Wheeler's (1966) theory of behavioral contagion, the oc-

currence of restraint reduction depends in part upon the observable consequences to the model for his behavior. Wheeler and Smith cited several studies (Bandura et al., 1963b; Walters et al., 1963; Walters & Parke, 1964) that demonstrated the importance of response consequences to the model. Specifically, they concluded that consequences to the model affect the observer's performance rather than his or her learning.

Subjects were enlisted men in the U.S. Navy. Each subject participated in a "discussion" with 3 tape-recorded confederates. The six experimental conditions were: (a) target expresses socially desirable opinions, model aggresses against target, peer reacts indifferently; (b) target expresses socially undesirable opinions, model calmly disagrees with target, peer reacts indifferently; (c) target expresses undesirable opinions, model aggresses against target, peer reacts indifferently; (d) target expresses undesirable opinions, model aggresses against target, experimenter censures model; (e) target expresses undesirable opinions, model aggresses against target, peer censures model; (f) target expresses undesirable opinions, model aggresses against target, model censures himself.

Thus, the response consequences to the model were varied (Conditions D, E, and F) with respect to the source of the censure: the experimenter (Condition D), a peer (Condition E), or the model himself (Condition F). In contrast, the model's behavior was not followed by any censure in Conditions A, B, (control groups) and C (*unqualified contagion* condition).

Dependent variables were the subject's overt verbal aggression toward the target, measures of the subject's affect, and subject's perception of other group members. The researchers hypothesized that aversive response consequences to the model would result in less overt verbal aggression by the subject toward the target.

Results showed that compared to the unqualified contagion group (Condition C), subjects in the control groups (Conditions A and B) exhibited relatively little aggression. Of the three censure conditions (Conditions D, E, and F), only experimenter censure (Condition D) yielded significantly less aggression than the no-censure group (Condition C). Further, experimenter censure (Condition D) resulted in significantly less aggression than both peer censure (Condition E) and self-censure (Condition F).

Wheeler and Smith (1967) noted that experimenter censure produced slightly less aggression than the no-aggression condition (Condition B). Other results indicated that subjects in the no-aggression and self-censure groups (Conditions B and F) expressed greater liking toward the model than did subjects in the experimenter-censure and peer-censure groups (Conditions D and E). As the authors noted, these differences are not consistent with Wheeler's (1966) prediction that models who successfully reduce restraints will be liked.

In their book, *The June Bug: A Study in Hysterical Contagion*, Kerckhoff and Back (1968) defined *contagion* as "the spread of behavior within a collectivity in which the spread is rather rapid and in the nature of a geometric progression"

(p. 24). The authors conceptualized contagion as a form of collective behavior in which: (a) individuals have a need to perform an action; (b) a barrier exists that prevents the action; and (c) a means of action is proposed that lowers the barrier. Restraint reduction in such situations is achieved typically by the example of other individuals.

Kerckhoff and Back (1968) distinguished their use of the term contagion from that of previous authors. First, they noted that Redl's (1949) definition implied that performance barriers are solely self-imposed; by contrast, Kerckhoff and Back asserted that such barriers could be either self- or externally imposed. Second, whereas Grosser et al. (1951) described contagion in terms of dyadic relationships, Kerckhoff and Back maintained that the term should apply primarily to collectivities. Third, the authors noted that contagion involves a more rapid and widespread dissemination of behavior than was implied by Grosser et al.

Kerckhoff and Back (1968) defined *hysterical contagion* as "the dissemination of a set of symptoms among a population in which no manifest basis for the symptoms may be established" (p. 12); as such, hysterical contagion was viewed as a subclassification of contagion. They selected the term "hysterical" as a prefix to "contagion" for two reasons. First, they noted that persons who were the recipients of contagion tended to be hysterical in a vernacular sense, that is, they were inclined to exhibit highly emotional behavior. Second, the symptoms they exhibited closely resembled those of clinical hysteria (e.g., anxiety, fainting spells, dizziness, nausea, and weakness).

Kerckhoff and Back (1968) presented their theory of the typical course and development of hysterical contagion. First, a number of individuals become exposed to a common source of social strain (e.g., inadequate social relationships, role conflicts), which results in intrapsychic tension. Poor coping mechanisms (e.g., denial) lead to a manifestation of physiological symptoms as well as a cognitive disassociation between the existing tension and the reason for its presence; the individuals are thus left in a state of unresolved and unidentified arousal. As a consequence, they are prompted to look to others in their environment for clues. This leads to the development of a hysterical belief. According to Kerckhoff and Back, the salient behavior of a few social isolates provides a credible cause and tangible evidence of an external threat:

Belief in a tangible threat makes it possible to explain and justify one's sense of discomfort—instead of anxiety, one experiences fear, and it is then possible to act in some meaningful way with respect to this tangible threat rather than just feeling frustrated and anxious. (pp. 159–160)

Symptoms are spread as more and more individuals become affected by the newly established group norms. Information about the belief is spread primarily through communication channels. As the epidemic progresses, increasing numbers of outside individuals enter the scene with new and conflicting information. As a conse-

quence, the individuals re-evaluate their situation, a new norm becomes established, and the epidemic declines and terminates.

Kerckhoff and Back (1968) presented a case study of what they believed to be a classic example of hysterical contagion: the "June bug" incident. In June, 1962, 62 employees of a textile factory reported a series of mysterious maladies (e.g., nausea, nervousness, hyperventilation, fainting spells, numbness) that they believed were caused by the bite of a poisonous insect. The initiators of the outbreak were a few social isolates, but the symptoms soon spread through several social networks. The course of the epidemic was relatively short-lived (1 week). After a careful investigation, official sources could find no organic reasons for employees' symptoms. As a consequence, the incident was identified as an example of hysterical contagion. Kerckhoff and Back concluded that personal and social characteristics interacted to affect the degree to which an individual would exhibit symptoms. Specifically, the likelihood of becoming affected increased as a function of the degree to which the individual was experiencing strain.

Ritter and Holmes (1969) investigated the occurrence of behavioral contagion as a function of restraint reduction. They adopted Wheeler's (1966) definition of *behavioral contagion*, maintaining that "behavioral restraints and their reduction are central in understanding behavior [*sic*] contagion" (p. 242). The authors distinguished behavioral contagion from other types of social influence (conformity, suggestion, imitation, and social learning). Suggestion, for example, was described as an influence process whereby "action is elicited from a person as a result of having seen a similar action" (p. 243). Ritter and Holmes noted that in this type of influence the individual has minimal (if any) restraints against performing the behavior. Further, prior to the observation the individual is not instigated to perform the behavior.

Thus, suggestion is differentiated from behavioral contagion by an absence of any marked conflict. According to Ritter and Holmes (1969), if "imitation" is seen as a type of social learning, it can be distinguished from contagion in that imitation refers to the learning of new responses, whereas in contagion, the behavior already exists in the observer's behavioral repertoire.

In their experiment, subjects were introductory psychology students who smoked at least five cigarettes daily. A questionnaire was administered to assess students' degree of restraint against smoking in class due to two factors: fear of the instructor's criticism and guilt associated with dropping ashes on the floor. Students who indicated that they were not restrained due to fear were excluded from sample selection. Of the sample, subjects were blocked into high guilt and low guilt groups. Thus, all subjects in this experiment were both instigated to smoke and restrained by fear, but differed with regard to restraint by guilt.

The low guilt subjects were randomly assigned to experimental and control groups, resulting in three conditions: low guilt experimental, low guilt control, and high guilt experimental. In all experimental conditions, the experimenter lighted a cigarette and flicked the ashes onto the floor; in a control condition, the

experimenter did not smoke. The dependent variable (i.e., behavioral contagion) was measured by the subjects' incidence of cigarette smoking.

Ritter and Holmes (1969) hypothesized (a) that a model would be differentially effective in reducing different types of restraints, and (b) that behavioral contagion would only occur when all relevant restraints were reduced; specifically, observing a model smoke would reduce only external restraints (fear), but not internal restraints (guilt). Results of the study supported their two hypotheses, namely, that observing the model smoke increased the incidence of smoking among subjects restrained by fear but not in subjects restrained by fear and guilt. Ritter and Holmes concluded that a model's performance of a behavior may reduce the observer's restraints against performing the behavior if the locus of these restraints is external; however, the model's performance of the behavior probably will not reduce restraints if the locus of such restraints is internal (i.e., the observer's code of conduct).⁶ They emphasized that these findings support the theoretical formulation that behavioral contagion is mediated by restraint reduction.

Milgram, Bickman, and Berkowitz (1969) investigated the relationship between the size of a stimulus crowd and the response of passersby. Their study tested the predictions of two models of crowd formation. First, the Coleman and James (1961) model of free-forming groups maintains that the growth of a group is dependent only upon the number of persons who are available to join it, but is independent of its size. By contrast, under a *contagion* assumption, a person is more likely to join a large group than a small one.

Subjects were 1,424 pedestrians on a busy street in New York City. The independent variable was the size of a stimulus crowd, which was varied from 1 to 15 persons. The stimulus crowd stood on the street pavement and looked up at the sixth-floor window of a nearby building. The dependent variables were the number of passersby who looked up, and number of passersby who stopped.

Results showed that the size of the stimulus crowd significantly affected both the proportion of passersby who looked up and the proportion of passersby who stopped. Specifically, as the size of the stimulus crowd was increased, a greater proportion of passersby adopted the behavior of the crowd. Other results indicated that, regardless of the size of the stimulus crowd, the percentage of those who only looked up was always higher than those who stopped.

Milgram et al. (1969) concluded that their findings contradicted the acquisition assumption of Coleman and James (1961) and supported the contagion assumption, (i.e., the number of persons who join in the observable behavior of a stimulus crowd is directly related to the size of the stimulus crowd). They noted that there is a logical basis for joining larger crowds: "... the larger the crowd the more likely its members are attending to a matter of interest" (pp. 81-82).

⁶This finding appears to run contrary to Wheeler's (1966) prediction, namely, that when the locus of restraints is the observer's superego, the observer's restraints are reduced to the extent that the model performs the behavior without manifesting guilt or fear.

The 1970s

Stephenson and Fielding (1971) studied experimentally the contagion of leaving behavior in small groups. The authors explained the process of restraint reduction in *behavioral contagion* from a perspective of equity theory. Specifically, they cited Homans's (1961) principle of "distributive justice," which states that persons in an exchange relationship should perceive that each has the same net outcomes (i.e., ratio of "profits" to "investments"); when this is not the case, action may be taken to restore equity. Based on this principle, Stephenson and Fielding proposed that once a group member performs a commonly desired action, the payoffs for similar action or nonaction by other group members are materially altered. Specifically, "the initiator, by his action, establishes an inequitable advantage over other members of the gathering which they may proceed to nullify by following his example" (p. 82).

Subjects (female undergraduate, non-psychology students) in their experiments were presented with a boring, impossible word task; specifically, they were instructed to construct as many words as possible using the letters of a given word (e.g., stream and dinosaur). Their obligation to remain in the experimental situation was varied systematically. Equity, deprived, and privileged conditions were created based on whether subjects believed that they were receiving the same, less, or more financial remuneration for taking part in the experiment than previous subjects.

In each of these conditions, a subject was tested either alone (control condition), singly with a confederate who left early (single experimental condition), or in a small group of subjects with a confederate who left early (group experimental condition). Thus, in this 3×3 design, there were nine experimental conditions. The primary dependent variable was the length of time subjects stayed in the situation. Stephenson and Fielding (1971) predicted that compared with the deprived control subjects, the deprived experimental subjects would stay for a much shorter time, and that this difference would be much greater than the equivalent difference in the equity conditions.

Results confirmed the research hypotheses. The differences in time stayed between the equity, deprived, and privileged conditions were substantially greater in the experimental conditions than in the control condition. Further, the experimental confederate had the greatest effect in the deprived condition and the least effect in the privileged condition. When questioned informally after the experiment, nearly all subjects reported that they were unaware of having been influenced by the confederate. Other results from their series of experiments led the authors to conclude that in a group situation, once one member leaves, other group members will follow more quickly and in a more "clustered" fashion than if they had been isolated from others in the group.

Bandura (1973) approached the concept of *contagion* from a social learning perspective, proposing that contagion is a consequence of modeling. Accord-

ing to Bandura, modeling effects are mediated by a number of variables, including the novelty of the behavior to the observer, current social sanctions regarding the performance of the act, the probability of consequences, and the degree of observer provocation. Provided observers are sufficiently instigated to perform a behavior, they are moved to action only if environmental stimuli indicate that the behavior carries with it a probability of nonaversive consequences. Bandura asserted that the judgment of this probability is 'extensively cognitively mediated' (p. 137).

Bandura (1973) discussed several specific modeling effects, namely, inhibition, disinhibition, response facilitation, and the acquisition of new behaviors, attitudes, or values. Response facilitation was defined as a situation in which a "model's behavior simply provides an external inducement for similar actions that can be performed with ease" (p. 127). He proposed that disinhibitory effects occur when the observation of a model leads to a weakening in the restraints of an observer's previously learned behaviors. Specifically, modeling produces disinhibitory effects when the model's conduct is 'legitimized,' or in the absence of negative consequences to the model.

Contagion of aggression was used as an example of a disinhibitory effect. As illustrations of this phenomenon, Bandura cited the Watts riots, and the work of Wheeler (e.g., Wheeler, 1966; Wheeler & Caggiola, 1966; Wheeler & Levine, 1967; Wheeler & Smith, 1967). Bandura suggested that future studies be conducted to investigate differing response consequences to aggression under varying levels of provocation in combination with variation of the characteristics and controlling power of the censoring agent.

Citing Wheeler's (1966) model of *behavioral contagion*, Russell, Wilson, and Jenkins (1976) examined the effects of sex, race, and number of jaywalking models on the jaywalking of pedestrians. No significant differences were found for sex and race; however, all of the models produced increased jaywalking relative to a no-model control group. In addition, two models produced more jaywalking than one, but most of the effect was produced with female models.

Milgram (1977) compared and contrasted contagion, convergence, and emergent norm theories as mechanisms that account for the uniformity of crowd behavior, heightened emotion, and violent antisocial behavior. He defined *contagion* as "the spread of affect or behavior from one crowd participant to another; one person serves as the stimulus for the imitative actions of another" (p. 239). In contrast to the theorizing of McDougall (1920), Allport (1924), and Blumer (1939/1951), Milgram maintained that there are instances in which contagion does not seemingly operate in a purely mechanical fashion. Milgram noted that contagion theorists face several problems. For example, what are the limits of contagion? Under what conditions will resistance develop? He concluded that "contagion is not so much a theory as it is a specific mechanism which may function in the context of other theoretical mechanisms" (p. 240).

Cohen, Colligan, Wester, and Smith (1978) presented Kerckhoff and Back's (1968) case study of the "June bug" incident as one of several examples of what they termed *contagious occupational psychogenic illness*. Cohen et al. defined this term as "a phenomenon characterized by group conversion reaction in which normally functioning employees experience various subjective, nonspecific somatic symptoms at the worksite of sufficient severity to produce an inability to work during the course of the reaction" (p. 10). According to the authors, incidents of contagious occupational psychogenic illness are preceded by some form of environmental stress and structural strain, and they typically are contingent upon factors such as host susceptibility and the mode of rumor transmission.

Cohen et al. (1978) conducted an investigation of such an outbreak that occurred in a Midwestern electronics plant in 1962. Fifty-one "affecteds" (i.e., individuals experiencing three or more symptoms) claimed that they detected a strange odor and complained of symptoms such as headaches, dizziness, light-headedness, weakness, and nausea. After a careful inspection of the plant, no physical or organic agents were found that could produce such symptoms. Results of the researchers' questionnaire indicated that affecteds had poorer interpersonal relations, suffered from more work pressure, had more job-role ambiguity and felt less control in their job situations than nonaffected workers. The authors concluded that the outbreak was a function of working conditions and employee-management relations. These factors resulted in psychological stress for which no outlets were available, causing anxiety and depression. Over a period of time, a socially acceptable means of expression was produced—a mass psychogenic reaction with physical symptomatology.

Goethals and Perlstein (1978) investigated the *contagion of aggression* in an extension of the previously cited study by Wheeler and Levine (1967). Subjects heard their position attacked in either a highly or slightly hostile manner (high vs. low instigation). Then a third person, portrayed as either similar or dissimilar to the subject, verbally attacked the instigator (similar vs. dissimilar model). The dependent variable was the subject's verbal aggression toward the instigator. The results indicated that under high instigation, there was significantly more aggression when the model was dissimilar. Under low instigation, however, the similar model produced more aggression.

Goethals and Perlstein (1978) explained this pattern of results in terms of Kelley's (1967) attribution theory, the so-called "triangulation effect."

If an individual finds that a dissimilar other makes the same judgment about an entity that he does, he can be more confident that his judgment is an accurate reflection of the entity rather than a biased person-caused response. The agreement of a similar other may be caused by the same biasing characteristics and is thus less impressive (1978, p. 116).

Thus, subjects in the high-instigation condition were free to aggress toward the instigator because, having received support from a dissimilar other, they could be confident that the aggression was warranted.

Freedman and Perlick (1979) investigated experimentally the effects of crowding on the contagion of laughter. The authors defined *contagion* as "the spreading through a group of the behavior or mood of one person" (p. 295). They stated that a number of explanations have been offered to explain contagion, including deindividuation (e.g., Festinger, Pepitone, & Newcomb, 1952), restraint reduction (Redl, 1949; Wheeler, 1966), social facilitation (Zajonc, 1965), and by the emergence of social norms (R. H. Turner, 1964). They noted that although there is little in the way of empirical evidence to support any of these explanations, they all probably play some role in contagion.

Freedman and Perlick (1979) suggested that physical density (i.e., crowding) may be another factor that increases the likelihood of contagion. They reasoned that higher density maximizes the salience of the initial act; as a consequence, it is more likely to be noticed. In addition, to the extent that deindividuation, restraint reduction, social facilitation, and normative pressure are causal factors in contagion, high density should increase contagion by magnifying the effect of these mechanisms.

Subjects in their experiment were female students who ranged in age from 17 to 29 years. Groups of three subjects and a female confederate listened to humorous tapes under either high density (small room) or low density (large room) conditions. In half of the groups, the confederate smiled and laughed; in the other half, she was subdued. Subjects were randomly assigned to four experimental conditions: high density/model laughs; high density/model does not laugh; low density/model laughs; low density/model does not laugh. The primary dependent variable was the amount of smiling and laughter in each condition, and was assessed by two independent judges who rated films of the subjects.

Results showed that although the effects of both density and laughter were significant, the only meaningful finding was the interaction of the two factors: Only the high density/model laughs condition differed significantly (i.e., increased laughter) from the other three conditions. Freedman and Perlick (1979) were surprised that the laughing model did not cause an increase in laughter in the low density condition. They noted, however, that the amount of laughter was "considerably higher" (although not statistically significant) in the model laughs condition than in the model does not laugh condition. They concluded (a) that density can be a significant factor affecting contagion, and (b) that the results support an intensification explanation of the effects of crowding as opposed to an arousal explanation.

The 1980s to the Present

Freedman, Birsky, and Cavoukian (1980) conducted a field experiment to investigate the effects of density and number on the contagion of applause. Although they did not offer an exact definition of *contagion*, they used the term in the same way as did Freedman and Perlick (1979). Their study concerned the effects of two

physical factors—density (the amount of space available per person) and number (group size)—on the occurrence of contagion. They attempted to overcome several limitations of the Freedman and Perlick experiment by using larger groups, by conducting the research in a natural setting, and by studying a more voluntary behavior.

Their subjects were visitors to a public science center in Ontario who viewed films in either small (8 to 10 persons) or large (22 to 28 persons) groups and sat either far apart (low density) or close together (high density). In all four experimental conditions, the group watched two humorous films. After the first film, a male confederate seated in the audience remained silent; immediately following the second film, he applauded loudly. The primary dependent variable was the proportion of each group that applauded after each of the films. Thus, the measure of contagion was the proportion of audience members who applauded subsequent to when the confederate applauded. An observer seated in the theater counted the number of persons applauding. All subjects were also administered a questionnaire to assess their enjoyment of the films and to elicit their impressions of the degree to which others liked the films.

Results indicated that there was much more applause in the high density than in the low density conditions; further, there was slightly more applause in the large than in the small groups. The authors concluded that high density and large numbers increase the likelihood of contagion. They cautioned, however, that their study and the one conducted by Freedman and Perlick (1979) involved a limited set of conditions, namely, behaviors that were socially acceptable, or even desirable. The authors pointed out that it remains to be demonstrated whether density and number will have the same effect on antisocial behaviors.

Colligan and Murphy (1982) conceptualized *contagious psychogenic illness* as “the collective occurrence of a set of physical symptoms and related beliefs among two or more individuals in the absence of an identifiable pathogen” (p. 35). According to the authors, contagious psychogenic illness is the consequence of two sequential but non-mutually exclusive processes. The initial stage resembles the process of convergence, that is, “a situation in which group members have, independently of one another, developed common affects or response patterns that are expressed simultaneously” (p. 47).

In this stage, the experience of arousal and accompanying physical and psychological symptomatology appear before the outbreak occurs. The second process is *contagion*, which the authors defined, following Milgram (1977), as a “spread of affect or behavior from group member to member, one person serving as the stimulus for the imitative act of another” (p. 47). This stage is marked by the development and spread of a belief in an external, legitimizing cause of the symptoms.

After reviewing 23 incidents of contagious psychogenic illness, Colligan and Murphy (1982) endorsed Kerckhoff and Back's (1968) theories regarding the phenomenon's underlying mechanisms. They also discussed a number of factors that

might affect contagious psychogenic illness, including gender, boredom, presence of physical stressors, level of perceived job stress, adequacy of interpersonal communications, and labor-management relations.

Freedman (1982) defined *contagion* as "essentially imitation mediated by restraint release due to observing another perform an action that the individual is in conflict about performing himself" (p. 171). He proposed that four criteria must be satisfied to classify a situation as an instance of contagion rather than another type of social influence. First, the behavior in question must be initiated by one or at most, a small number of individuals; by contrast, in conformity, a majority of people initiate the behavior. Second, there is no sufficient reason for an individual to perform the behavior other than the fact that others are doing so; this differs from emergent norms (see R. H. Turner, 1964), in which a norm is operative in inducing individuals to behave similarly. Third, observing others perform a behavior increases the probability of like performance. Fourth, individuals are not merely imitating an individual in their group who is of particularly high social status. Freedman maintained, in addition, that perhaps all instances of contagion involve some degree of conflict.

Freedman (1982) stated that "mass psychogenic illness" is a classic example of contagion. He noted that it meets all four of the above criteria: (a) the illness tends to start in a few persons and then spreads; (b) no known organic cause can be attributed to the manifestations; (c) outbreaks of illness are not directed by particular individuals, nor do sufferers fall victim because they want to follow an individual of influence; (d) evidence derived from case studies suggests that outbreaks are always characterized by the presence of stresses, which tend to produce conflict within the afflicted individuals.

Freedman (1982) reviewed three theories of contagion. First, according to "primitive expression theories" (e.g., Freud, 1922/1959; Le Bon, 1895/1903), contagion results from suppression of an individual's normal restraints and the consequent release of primitive impulses. According to Freedman, although these theories propose fascinating ideas, there is little in the way of research to support them. Further, such theories are inadequate in explaining outbreaks of mass psychogenic illness. In order to accept such theories, one would have to believe that individuals have an unconscious wish to become ill, a notion that Freedman found to be highly implausible.

Second, according to "deindividuation theories" (see Diener, 1976; 1979; Festinger et al., 1952; Zimbardo, 1969), individuals tend to act more freely when in the presence of a group. Specifically, they lose their sense of personal identity and feelings of responsibility for their actions. The emphasis of these theories is not on the emergence of long buried primitive impulses but rather on the lowering of normal restraints due to feelings of perceived anonymity. Freedman concluded that there is no solid evidence that supports the contention that anonymity is involved with contagion.

Third, "imitation theories" (e.g., Redl, 1949; Wheeler, 1966) maintain that contagion is "essentially imitation that is mediated by the reduction of restraint caused by observing someone else's behavior" (p. 179). Support for these theories is provided by researchers such as Bandura et al. (1963b), who found that observing someone else perform an act for which there are no negative consequences increases the likelihood that others will imitate that act. He maintained that imitation theories show promise in their efforts to describe outbreaks of mass psychogenic illness.

Freedman (1982) reported further that "intensification theory" explains when contagion is likely to occur. According to this theory, as density increases, the actions of others become more salient, making it more likely that they will be picked up by observers. Support for this approach was found by Freedman and Perlick (1979) and Freedman et al. (1980).

Freedman (1982) proposed three hypotheses that predict when contagion (and consequently mass psychogenic illness) is most likely to occur: (a) when individuals feel like a part of a group and are anonymous; (b) when large groups are coupled with high density conditions; (c) when the first individual to act suffers no negative consequences for the performing the act, providing that contagion is mediated partly by restraint reduction. Freedman concluded that although the current theories attempt to describe how and when contagion occurs, none explains why it occurs.

Reicher (1984) extended the social identity approach of Tajfel (1978) and J. C. Turner (1982) to account for the social influence or *contagion* that occurs in crowds during riots. According to Reicher, to the degree that individuals in a crowd identify themselves with a common social category, they can be properly regarded as a form of social group. This process of social identification gives rise to a form of social influence referred to as "referent informational influence" (J. C. Turner). According to this view, in deciding how to behave, "group members seek out the stereotypic norms which define category membership and conform their behavior to them. It is, in effect, a process of self-stereotyping" (Reicher, p. 4).

And yet, how are crowd members to conform to stereotypic norms when in novel situations where no established norms exist? "The answer is that crowd members must elaborate an appropriate situational identity which at once provides a guide for action and conforms to their common social identification" (Reicher, 1984, p. 4). This occurs through a process that J. C. Turner (1982) calls the "inductive aspect of categorization." That is, norms will be inferred from the behavior of others provided that (a) they are seen as members of the in-group, and (b) the behavior is consistent with the observer's social identification. This process provides for the quickly evolving yet homogeneous nature of crowd behavior. "Crowd norms may quickly be superseded as new behaviors come to be seen as more appropriate" (Reicher, p. 4).

Following an analysis of the events that occurred in the so-called St. Pauls' Riots in Bristol, England, April 2, 1980, Reicher argued for the superiority of the social identity model over competing theories of crowd behavior. For example, deindividuation theories (Festinger et al., 1952; Zimbardo, 1969) cannot account for the fact that crowd members appeared anonymous only with respect to individuals not from St. Pauls. And emergent norm theory (R. H. Turner, 1964; R. H. Turner & Killian, 1957) cannot account for the rapidity with which norms developed in the novel situations of the riot.

R. H. Turner and Killian (1987) used a symbolic interactionist approach in their analysis of collective behavior. From this perspective, they contrasted their theory of emergent norms with contagion and convergence (see also R. H. Turner, 1964; R. H. Turner & Killian, 1957). Emergent norms maintains that the individual's evaluating and revising process is an active, rational, cognitive event that takes place on a strictly conscious level. By contrast, contagion is marked by the "operation of automatic, uncritical imitation," and convergence involves the "release of compelling, unconscious psychic forces" (R. H. Turner & Killian, 1987, p. 26).

Contagion was defined as a form of crowd behavior "in which a form of highly visible, unusual, bodily behavior spreads rapidly through people assembled in one place" (R. H. Turner & Killian, 1987, p. 21). Suggestibility, defined as "the readiness of the individual to rely on others for psychological patterning when experiencing uncertainty, suspense, or anxiety" (p. 23), is implicated as the condition necessary for the diffusion of emotion. According to Turner and Killian, mechanisms leading to the contagion of behavior represent social influence that is "unnoticed" by the recipient.

Thus, in contrast to emergent norms, contagion is a passive process. As examples of contagion, Turner and Killian (1987) cited the dancing mania of the Middle Ages (Hecker, 1837/1970), Moss and McEvedy's (1966) description of a school fainting epidemic, and the theories of Le Bon (1895/1903), Blumer (1939/1951), and Wheeler (1966). Turner and Killian inferred that Wheeler's conceptualization of contagion involves conscious, cognitive processes—mechanisms not unlike those underlying their conceptualization of emergent norms.

Turner and Killian (1987) noted that, whereas contagion and convergence theories explain collective behavior as arising from the individual and then spreading to the group, emergent norms reflects the imposition of new group norms on the individual. Turner and Killian did not deny the existence of contagion and convergence; however, their central proposition was that the origin of all collective behavior arises from emergent norms.

Crandall (1988) examined the *social contagion* of binge eating in two college sororities based on self-report questionnaires. She found clear evidence for the existence of group norms regarding binge eating, although the norms differed for the two sororities. In one, the more a member binged, the more popular she was. In the other, those binging at the group mean were more popular than those binging too much or too little. Crandall discussed several processes of social influence

that might account for her results. These included: direct teaching, leader modeling, social coercion, and behavioral contagion (Wheeler, 1966). Unfortunately, her data did not allow her to choose between them.

Sullins (1991) investigated the effects of individuals' expressive styles on the contagion of emotions. *Emotional contagion* was defined as "the process by which individuals seem to 'catch' the mood of others around them" (p. 166). Subjects completed the Affective Communication Test (Friedman, Prince, Riggio, & DiMatteo, 1980) of nonverbal expressiveness. Subsequently, they were divided into same sex dyads, two high expressives, two low expressives, or one high and one low. Subjects' moods were measured both before and after a 5-minute waiting period during which talking was not permitted. Some subjects waited with a partner who, like them, was waiting to be in an experiment (relevant partner). Others waited with a partner who, unlike them, was waiting to take a make-up exam (irrelevant partner). Finally, a control group of subjects waited alone. The dependent variable was the amount of mood convergence during the waiting period.

In agreement with social comparison theory (Festinger, 1954), the results indicated greater mood convergence in the relevant partner condition than in the irrelevant partner and control conditions. The latter conditions did not differ from one another. In addition, high-low pairs showed significantly more mood convergence than high-high and low-low pairs; and within the high-lows, lows showed more convergence toward the highs than vice versa.

Most recently, Levy (1992; see also Levy, Brief, & Veranavich, 1990) investigated empirically the dynamics that distinguish conformity from disinhibitory contagion. Levy defined *disinhibitory contagion* as a particular case of *social contagion* in which a recipient, who is in an approach-avoidance conflict, experiences a reduction in restraints as a consequence of observing an initiator perform the desired behavior.

Subjects were recruited for a study purported to investigate attitudes and perceptions. At each experimental session, one subject and a confederate (posing as another subject) completed a series of personality questionnaires and then were requested by the experimenter to volunteer their services, without compensation, for tedious clerical work during the subsequent week. The confederate then either refused (disinhibitory contagion condition) or complied (conformity condition) with the request.

The approach motive thus consisted of the subject's desire to refuse the experimenter's request, and the avoidance motive consisted of restraints against refusing. The primary dependent variables were the amount of time volunteered and the degree of conflict experienced by the subject. Conflict was operationalized by 17 measures, which were factor analyzed to develop a set of two empirically based indices: Emotional Turmoil and Cognitive Effort.

Results showed that subjects in the disinhibitory contagion condition volunteered for less time and experienced less conflict (on both indices) than did sub-

jects in the conformity condition. Levy (1992) concluded that the results provide both theoretical and empirical grounding for the establishment of disinhibitory contagion as a unique and distinct type of interpersonal influence.

Proposed Redefinitions of Contagion

This review of the literature reveals that contagion has been approached, described, and analyzed by authors from numerous and diverse theoretical orientations. Further, there are significant differences with respect both to definitions of contagion and to explanations of its underlying mechanisms. A great deal of the confusion surrounding this term is due to the fact that the phenomena involved in contagion are exceedingly heterogeneous; despite this, they have traditionally been placed under homogeneous rubrics.

A reconceptualization of contagion is therefore proposed. In its broadest sense, the phenomenon is referred to as *social contagion*. In this section, social contagion is defined and differentiated from other types of social influence. Three subtypes of social contagion are then delineated: *disinhibitory contagion*, *echo contagion*, and *hysterical contagion*. Each type possesses unique and distinct characteristics and operates by means of differing dynamics.

Social Contagion

Social contagion is defined here as *the spread of affect, attitude, or behavior from Person A (the "initiator") to Person B (the "recipient"), where the recipient does not perceive an intentional influence attempt on the part of the initiator*. The term "social" was selected as a prefix to "contagion" following the work of Blumer (1939/1951), R. H. Turner and Killian (1957), English and English (1958), Raven and Rubin (1983a), and Reber (1985), and to denote the social context of the phenomenon.

If social contagion is to be viewed as a unique type of social influence from which differential predictions can be made, it first must be distinguished from other types of influence. The most crucial distinction relates to the recipient's perception of intentionality on the part of the initiator. Under the proposed definition, if Person B judges Person A's influence attempt to be intentional, then social contagion has not occurred. Social contagion is thereby distinguished from intentional social influence types, such as obedience and compliance.

As noted by several authors (e.g., Ritter & Holmes, 1969; Wheeler, 1966), the problem with a number of early definitions of contagion (e.g., Grosser et al., 1951; Lippitt et al., 1952; Polansky et al., 1950; Redl, 1949) is that they do not adequately distinguish contagion from other types of social influence, such as social facilitation; vicarious learning, suggestion, imitation, Asch-type conformity, and mass hysteria (or mass psychogenic illness). Ostensibly, all of these situations are marked by an individual who (a) does not perform a given behavior,

(b) observes another person (or persons) perform the behavior, and then (c) performs the behavior him- or herself. Because these situations and the resultant behaviors do not appear to be different phenotypically, motivational dynamics must be employed to distinguish further subtypes of contagion from other types of social influence.

Disinhibitory Contagion

Disinhibitory contagion is defined as a case of social contagion in which a recipient, who is in an approach-avoidance conflict, experiences a reduction in restraints as a consequence of observing an initiator perform the desired act. In disinhibitory contagion, the recipient's behavior is not necessarily an exact replication of the initiator's behavior; rather, the behaviors belong to the same behavioral class. Further, disinhibitory contagion occurs at a relatively high level of cognitive processing.

The term "disinhibitory" was selected as a prefix to "contagion" following Bandura and Walters's (1963) conceptualization of "disinhibitory effects," wherein the "observation of models . . . weaken[s] inhibitory responses" (p. 60). Further, Pavlov (cited in Kaplan, 1966) referred to disinhibition as "the inhibition of an inhibition" (p. 167). Disinhibition refers here to the weakening or removal of a restraint due to an external or internal stimulus.

The following brief formula summarizes the phenomenon of disinhibition: $D: A_p > A_v$, in which D represents disinhibition, A_p represents an approach motive, and A_v represents an avoidance motive. Disinhibition is the outcome when approach is greater than avoidance; or, put another way, disinhibition occurs when the avoidance gradient is lower than the approach gradient in an approach-avoidance conflict.

Thus, the reduction of restraints is central to disinhibitory contagion. As noted by Wheeler (1966), if an individual is instigated to perform a behavior, and is not physically restrained from performing the behavior, there is an implication that the individual possesses psychological restraints against performing the behavior. In other words, the person is experiencing an approach-avoidance conflict. The observation of an initiator's performing the behavior then results in a change of the relative strengths of the person's approach and avoidance tendencies in the direction of the approach motive.

It is the presence of conflict that differentiates disinhibitory contagion from several other forms of social influence, including social facilitation, vicarious learning, suggestion, and imitation. In these other forms of influence, Person B does not necessarily experience any restraints against performing the behavior; thus, conflict may not be present.

In contrast to vicarious learning, which traditionally refers to the learning of new responses, in disinhibitory contagion the behavior already exists in the observer's behavioral repertoire. In suggestion, as compared to disinhibitory conta-

gion, Person B is not instigated to perform the behavior prior to the observation of Person A. Imitation is considered to be a generic term that subsumes a number of different types of social influence, including social contagion, vicarious learning, and conformity (see Wheeler, 1966).

The reduction of the recipient's restraints against performing the behavior differentiates disinhibitory contagion from Asch-type conformity. Although both types of social influence involve conflict, in conformity, the individual's conflict is caused by the observation of other persons. In disinhibitory contagion, the conflict is present prior to the observation of others—in fact, others contribute to a resolution of the conflict.

For example, upon entering a new class a student may be experiencing a conflict between her desire to request information from the instructor and her fear of asking questions. As she observes other students perform the desired behavior, however, she experiences a reduction in her fear, thus freeing her to engage in the behavior.

Of the theories and research studies reviewed thus far, the clearest examples of disinhibitory contagion include Bandura (1973), deCharms and Wilkins (1963), Freedman (1982), Freud (1922/1959), Goethals and Perlstein (1978), Grosser et al. (1951), Kerckhoff and Back (1968), Levy (1992), Redl (1949), Ritter and Holmes (1969), Russell, et al. (1976), Smith et al. (1964), Stephenson and Fielding (1971), Wheeler (1966), Wheeler and Caggiula (1966), Wheeler and Levine (1967), Wheeler and Smith (1967), Wheeler et al. (1964). (See Table 2.)

A number of other theories and empirical investigations in the literature appear to be directly relevant to the present conceptualization of disinhibitory contagion, although they do not employ the term contagion. These include Asch (1951, 1952), Bandura (1977), Bandura and Barab (1973), Bandura, Ross, and Ross (1963a, 1963b), Bandura and Walters (1963), Britt (1940), Dannick (1973), Diener (1979), Diener, Fraser, Beaman, and Kelem (1976), Freed et al. (1955), Kimbrel and Blake (1958), Lefkowitz et al. (1955), Milgram (1965), Osman (1982), Schachter and Hall (1952), Sigelman and Sigelman (1976), Walters et al. (1963), and Walters and Parke (1964) (Table 2). Although the results of these studies do not allow it as an unequivocal conclusion, restraint reduction appears to be the primary mediating factor.

For example, Asch (1951, 1952) carried out a number of experimental variations of his classic conformity paradigm to investigate further the factors that affect conformity and independence. Several of these are relevant to disinhibitory contagion. In one condition, a single confederate was instructed to announce correct responses on all trials, even when the remainder of the confederates did not. Results indicated that the frequency of pro-majority errors dropped to 5.5% (about one fourth of the baseline level). Asch (1951) wrote: "It is clear that the presence in the field of *one other* [original italics] individual who responded correctly was sufficient to deplete the power of the majority, and in some cases to destroy it" (p. 186).

TABLE 2
 Alphabetical Listing of Authors and Years of Publication Under Newly
 Proposed Definition of Disinhibitory Contagion

Author(s)	Year
Asch	1951
Asch	1952
Bandura	1973
Bandura	1977
Bandura & Barab	1973
Bandura, Ross, & Ross	1963a
Bandura, Ross, & Ross	1963b
Bandura & Walters	1963
Britt	1940
Dannick	1973
deCharms and Wilkins	1963
Diener	1979
Diener, Fraser, Beaman, & Kclem	1976
Freed, Chandler, Mouton, & Blake	1955
Freedman	1982
Freud	1922
Goethals & Perlstein	1978
Grosser, Polansky, & Lippitt	1951
Kerckhoff & Back	1968
Kimbrell & Blake	1958
Lefkowitz, Blake, & Mouton	1955
Levy	1992
Milgram	1965
Osman	1982
Redl	1949
Ritter & Holmes	1969
Russell, Wilson, & Jenkins	1976
Schachter & Hall	1952
Sigelman & Sigelman	1976
Smith, Murphy, & Wheeler	1964
Stephenson & Fielding	1971
Walters, Leat, & Mezei	1963
Walters & Parke	1964
Wheeler	1966
Wheeler & Caggiula	1966
Wheeler & Levine	1967
Wheeler & Smith	1967
Wheeler, Smith, & Murphy	1964
Worchel & Cooper*	1983

*Cited in the Appendix

Echo Contagion

Echo contagion is defined as a case of social contagion in which an unconflicted recipient imitates or reflects spontaneously the affect or behavior of an initiator. In contrast to disinhibitory contagion, with echo contagion the responses of the initiator and recipient are relatively exact, and conflict is not necessarily present within the individual. Further the dynamics that underlie echo contagion occur at a lower level of cognitive processing; thus, the recipient's responses are relatively unconscious and involuntary. However, disinhibitory and echo contagion are alike in a fundamental way; in both cases, the initiator behaves in a way that is attractive or desirable to the recipient.

The term "echo" is used as a prefix to "contagion" following English and English's (1958) definition of "echopraxia," namely, "a tendency toward automatic imitation of another's movements" (p. 168). Although "echo contagion" was coined originally by Polansky et al. (1950), their use of the term denoted a more deliberate and voluntary behavioral response than does the present conceptualization.

Under this new definition, of the theories and research studies discussed in this paper, the most distinct examples of echo contagion include Baldwin (1897), Blumer (1939/1951), Brown (1954), Freedman et al. (1980), Freedman and Perlick (1979), Le Bon (1895/1903), McDougall (1920), Milgram et al. (1969), Sullins (1991), Turner (1964), and Turner and Killian (1957, 1987). (See Table 3.)

A number of other theories and empirical investigations in the literature appear to be directly relevant to the present conceptualization of echo contagion, although they do not employ the term contagion. These include Allport (1924), Chaplin (1968), Novakovsky (1924), Pennebaker (1980), Starch (1911), Sullivan (1953), and Tarde (1903).

For example, echo contagion was demonstrated by Starch (1911), who investigated unconscious imitation in handwriting. He defined unconscious imitation as the "unintentional modification in the performance of a specific act due to the presence of a model of that act at the time when it is being executed" (p. 223). Starch focused on the extent to which an individual's normal handwriting style is modified by a model of script. Subjects were instructed to "write out the words and sentences" of several standardized literary passages that had been written previously in longhand. Results showed that despite the fact that they had not been instructed to do so, subjects imitated the longhand style of the stimulus passages on two measures, inclination (slant) and size. Further, greater imitation was seen in women than in men.

As another example, Pennebaker (1980) investigated social and perceptual factors that influence coughing in naturalistic settings. He noted that the traditional medical perspective has failed to consider psychological and perceptual processes that mediate the cough reflex. Pennebaker proposed that coughing and related "reflex actions" (e.g., sneezing, yawning) represent immediate responses

TABLE 3
 Alphabetical Listing of Authors and Years of Publication Under Newly
 Proposed Definition of Echo Contagion

Author(s)	Year
Allport	1924
Baldwin	1897
Blumer	1939
Brown	1954
Chaplin*	1968
English & English*	1958
Eysenck, Arnold, & Meili*	1972
Freedman, Birsky, & Cavoukian	1980
Freedman & Perlick	1979
Le Bon	1895
McDougall	1920
Milgram, Bickman, & Berkowitz	1969
Novakovsky	1924
Pennebaker	1980
Raven & Rubin*	1983a
Sears, Peplau, Freedman, & Taylor*	1988
Starch	1911
Sullins	1991
Sullivan	1953
Tarde	1903
R. H. Turner	1964
R. H. Turner & Killian	1957
R. H. Turner & Killian	1987
Wolman*	1973

*Cited in the Appendix

to perceptions of one's internal state. According to Pennebaker, this process occurs at a very low cognitive level; the individual's orientation to his or her internal sensations is more automatic than conscious. Further, coughing is a social phenomenon: "From a perceptual perspective, hearing another person cough prompts others to monitor quickly their own throat, thus increasing the probability that someone would become aware of throat irritation and emit a cough" (p. 87).

In a series of studies on coughing behavior, Pennebaker measured unobtrusively the behavior of students in intact classrooms. Results showed that students were more likely to cough when they heard others do so and that the number of coughs per person increased as a function both of group size and of physical proximity (closeness) to the cougher. Further, coughing was greatest when competing external stimuli were low or uninteresting. In explaining this last finding, Pennebaker reasoned that since the individual's capacity to process information is

finite, more salient sources will be attended to and processed first. The presence of other, competing stimuli tend to steer the individual away from internal perceptions.

Pennebaker reported that despite the fact that at least 29% of the students coughed at least once during the course of each lecture, when students were asked subsequently if they had heard any coughing, the majority reported that they did not. This finding further supported his contention that the processing of cough-related information occurs at a low level. Pennebaker concluded that coughing is highly amenable to social and environmental influence.

Hysterical Contagion

Hysterical contagion is defined here as a case of social contagion involving the spread of physical symptoms from an initiator to a conflicted recipient in the absence of an identifiable pathogen. In hysterical contagion, the spreading of symptoms may occur with or without the presence of accompanying illusory beliefs. Like disinhibitory contagion, hysterical contagion is characterized by the presence of conflict. On the other hand, like echo contagion, hysterical contagion occurs at a low level of cognitive processing and the contagious behavior is relatively exact.

However, hysterical contagion is unlike both disinhibitory and echo contagion in an important way. With hysterical contagion, the behavior of the initiator is neither attractive nor desirable to the recipient. For example, seeing a co-worker scream that she has been bitten by a bug and faint is not the type of behavior that the recipient wants to engage in (Kerckhoff & Back, 1968). Nevertheless, the behavior becomes contagious because it increases the observer's tension and physical symptoms while, at the same time, providing an explanation for them.

The term "hysterical" was chosen as a prefix to "contagion" following the work of Kerckhoff and Back (1968). The rationale for the selection of this term here is that the underlying explanation for symptom manifestation parallels that of classic *conversion hysteria* (see Breuer & Freud, 1895/1955). Further, in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1987), hysterical neurosis, conversion type was described as

an alteration or loss of physical functioning that suggests physical disorder, but that instead is apparently an expression of a *psychological conflict* [emphasis added] or need . . . The symptoms of the disturbance are not intentionally produced . . . and, after appropriate investigation, cannot be explained by any physical disorder or known pathophysiologic mechanism. (p. 257)

Thus, hysterical contagion is conceptualized here as a group conversion reaction. It is similar to echo contagion in that cognitive processing occurs at a low level in both phenomena. However, hysterical contagion is theorized as being precipitated by the presence of conflict and as involving the spread of pathological symptoms; such is not the case in echo contagion. Of the theories and research presented

in this paper, examples of hysterical contagion include Cohen et al. (1978), Colligan and Murphy (1982), Freedman (1982), and Kerckhoff and Back (1968). (See Table 4.)

Numerous terms have been used throughout the literature to refer to the phenomenon of hysterical contagion. Some of these include (alphabetically) *collective delusion* (Medalia & Larsen, 1958), *collective hysteria* (Goldenson, 1984), *contagious occupational illness* (Cohen et al., 1978), *crowd hysteria* (Goldberg, 1973), *epidemic hysteria* (Goldenson, 1984; Sirois, 1982), *hysterical epidemic* (Moss & McEvedy, 1966), *hysterical imitation* (English & English, 1958), *industrial mass psychogenic illness* (Smith, Colligan, & Hurrell, 1978), *mass hysteria* (Champion, Taylor, Joseph, & Hedden, 1963; Corsini, 1984; Goldenson, 1984;

TABLE 4
Alphabetical Listing of Authors and Years of Publication Under Newly
Proposed Definition of Hysterical Contagion

Author(s)	Year
Champion, Taylor, Joseph, & Hedden	1963
Cohen, Colligan, Wester, & Smith	1978
Colligan & Murphy	1982
Colligan, Pennebaker, & Murphy	1982
Colligan & Stockton	1978
Corsini	1984
English & English*	1958
Freedman	1982
Goldberg	1973
Goldenson*	1984
Hecker	1837
Hefez	1985
Johnson	1945
Kerckhoff	1982
Kerckhoff & Back	1968
Major	1955
Medalia & Larsen	1958
Moss & McEvedy	1966
Phoon	1982
Raven & Rubin*	1983a
Roueche	1978
Singer	1982
Singer, Baum, Baum, & Thew	1982
Sirois	1982
Smith, Colligan, & Hurrell	1978
Stahl	1982
Stahl & Lebedun	1974

*Cited in the Appendix

Johnson, 1945), *mass psychogenic illness* (Cohen et al., 1978; Colligan & Murphy, 1982; Colligan, Pennebaker, & Murphy, 1982; Freedman, 1982; Kerckhoff, 1982; Singer, 1982; Singer, Baum, Baum, & Thew, 1982), and *sociogenic illness* (Stahl, 1982).

Despite the diversity of these terms, they all refer to a condition in which abnormal emotional behavior, beliefs, or physical symptoms are spread from one individual to another. Examples of such physical manifestations include nausea, difficulty breathing, fainting, headaches, dizziness, nervousness, blurred vision, and muscular weakness. Causes have been misattributed variously to insect bites (Champion et al., 1963; Kerckhoff & Back, 1968), mysterious illnesses (Colligan & Stockton, 1978), nuclear fallout (Medalia & Larsen, 1958), evil spirits (Major, 1955; Hecker, 1837/1970; Phoon, 1982), and toxic gases or strange odors (Cohen et al., 1978; Goldberg, 1973; Hefez, 1985; Johnson, 1945; Roueche, 1978; Smith et al., 1978; Stahl & Lebedun, 1974).

Colligan and Stockton (1978) proposed a three-stage process for the development of any psychogenic epidemic. The first stage involves the arousal of negatively associated feelings that are really due to stress but for which the individual fails to make a correct attribution due to perceptual ambiguity. The second stage is the discovery of a salient (but nevertheless normal) environmental stimulus. Based on this discovery, the individual then makes an attribution for his or her negative affect. The third stage in their model consists of two sub-parts: convergence and contagion. Convergence occurs when two or more individuals independently develop the same symptoms but don't openly display them until they are in each other's mutual presence. Contagion, on the other hand, occurs when an individual who is not predisposed to display a symptom before he or she joins a group, subsequently is induced to have similar feelings or to display symptoms simply due to the mere presence of others. (See also Kerckhoff & Back, 1968, discussed earlier, for a theory of hysterical contagion.)

Theoretical And Empirical Studies Too General To Classify

A number of theoretical and empirical studies cited herein are too general to classify by the new definitions provided earlier. These include Crandall (1988), Goldenson (1984), Lippitt et al. (1952), Milgram (1977), Polansky et al. (1950), and Reicher (1984). (See Table 5).

Summary Implications and Conclusions

Historically, contagion has been approached from a number of diverse disciplines, including sociology, social psychology, social learning, and psychoanalysis. Each orientation has offered different and sometimes contradictory descriptions and explanations for the mechanisms that underlie contagion. Moreover, this lack of consensus exists even within each orientation. It appears that much of the confu-

TABLE 5
Alphabetical Listing of Authors and Years of Publication Under Newly Proposed
Definitions of Social Contagion—Too General to Classify

Author(s)	Year
Crandall	1988
Goldenson*	1984
Lippitt, Polansky, & Rosen	1952
Milgram	1977
Polansky, Lippitt, & Redl	1950
Reber*	1985
Reicher	1984
Stang & Wrightsman*	1981

*Cited in the Appendix

sion regarding contagion is due to the manner in which it has been conceptualized. A review of the literature revealed that the phenomena involved in contagion are exceedingly heterogeneous; however, they have traditionally been placed under homogeneous rubrics.

We have proposed that contagion should be reconceptualized. In its broadest sense, social contagion is defined as the spread of affect, attitude, or behavior from Person A (the "initiator") to Person B (the "recipient"), where the recipient does not perceive an intentional influence attempt on the part of the initiator. Three subtypes of social contagion then were identified, each of which possesses unique and distinct characteristics, and which operates under differing dynamics. First, disinhibitory contagion is defined as a case of social contagion in which a recipient, who is in an approach-avoidance conflict, experiences a reduction in restraints as a consequence of observing an initiator perform the desired act. Second, echo contagion is defined as a case of social contagion in which an unconflicted recipient imitates or reflects spontaneously the affect or behavior of an initiator. Third, hysterical contagion is defined as a case of social contagion involving the spread of physical symptoms from an initiator to a recipient in the absence of an identifiable pathogen.

Social contagion and its three subcategories were distinguished from other types of social influence, including obedience, compliance, social facilitation, vicarious learning, suggestion, imitation, conformity, and mass hysteria. Selected theoretical papers and research studies were then reclassified under these newly proposed definitions of contagion. One advantage of distinguishing between the different subtypes of contagion is that it provides for the possible resolution of apparent contradictions in the literature. We offer two tentative proposals.

We reviewed three field studies that examined the social status of initiators. Kerckhoff and Back (1968) found that the initiators of contagion were social iso-

lates; on the other hand, Lippitt et al. (1952) and Polansky et al. (1950) found that initiators were of high social status. This apparent contradiction can be neatly resolved, however, when it is recognized that Kerckhoff and Back were studying hysterical contagion, whereas Lippitt et al. and Polansky et al. were examining disinhibitory and/or echo contagion.

There are at least two factors which, considered together, make the initiators in the case of hysterical contagion most likely to be social isolates. First, given the well documented relationship between the lack of social support and susceptibility to stress (Caplan & Killilea, 1976; Pearlin, Lieberman, Menaghan, & Mulvan, 1981), it follows that social isolates would be the ones most likely to first manifest physical symptoms in a stressful environment. Second, as noted previously, the behaviors, beliefs, and physical symptoms manifested during hysterical contagion are not ones that are generally desired. And yet, it is the social isolates who are most likely to be free from any group standards that might otherwise mitigate the bizarre symptoms of hysterical contagion.

With either disinhibitory or echo contagion, in contrast, it appears that the initiators would be more likely to be group members of high status. Considering disinhibitory contagion, if the behavioral restraints are group imposed, high status members should have greater latitude for violating these restraints than members of lower status (Hollander, 1958); if the restraints are imposed by an authority figure, high status members should be less likely to incur severe penalties for violations than low status members (cf. Lefkowitz et al., 1955; Russell et al., 1976). Considering echo contagion, group members of intermediate or low status may increase their standing in the group by imitating the behavior of high status members (Dittes & Kelley, 1956). Future research should cast light on the validity of these possibilities.

Another apparent contradiction concerns whether similar or dissimilar models produce greater contagion. Sullins (1991) found that similar models produced greater contagion, and Reicher (1984) found greater influence of, presumably similar, in-group members. And yet, Goethals & Perlstein (1978) and Wheeler and Levine (1967) both found greater influence for dissimilar models.

Once more, this apparent contradiction can be resolved by distinguishing the type of contagion in question. The necessary antecedent conditions for the greater influence of dissimilar models appear to be the presence of strong conflict within the observer and the observer's private desire to engage in the model's behavior. These conditions are both present only in the case of disinhibitory contagion. Thus, we propose that similar models will yield greater influence with echo or hysterical contagion and that dissimilar models will yield greater influence with disinhibitory contagion.

It should be noted, however, that the Goethals and Perlstein (1978) and the Wheeler and Levine (1967) studies were both concerned with the contagion of aggression where there was strong instigation for the recipient to behave aggressively. It is thus an open question whether the finding of greater influence by

a dissimilar model generalizes to other types of disinhibited behaviors besides aggression and to other situations with lesser degrees of instigation.

Historically, the term contagion has been closely associated with crowd behavior. Although it is a statement of fact that contagion—that is, the spread of affect, attitude, or behavior—does sometimes occur in crowds, one point this article makes clear is that contagion also refers to three distinct social influence research paradigms (disinhibitory contagion, echo contagion, and hysterical contagion). The problem is that despite the great amount that has been written about contagion, precious little is known about what factors actually control the degree of influence in contagion paradigms, because not enough systematic research has been done. It is thus difficult to draw firm conclusions regarding the effects of different variables on the degree of influence produced.

For example, in all the contagion studies reviewed herein, we located only two that manipulated the relative number of initiators and recipients (Milgram et al., 1969; Russell et al., 1976), only three that manipulated the similarity of initiators and recipients (Goethals & Perlstein, 1978; Sullins, 1991; Wheeler & Levine, 1967), only one that manipulated the friendliness of initiators (Grosser et al., 1951), and only three that manipulated the immediacy or density of initiators and recipients (Freedman et al., 1980; Freedman & Perlick, 1979; Pennebaker, 1980). What is needed is more contagion research that systematically manipulates these and other variables. All the while, this research should carefully distinguish between the three types of contagion identified herein. Only with such research will it be possible to eventually arrive at a more complete psychology of contagion.

Although this article focused on the phenomenon of contagion, it is intended also to be a starting point for a much broader re-examination of the entire social influence literature. Other types of social influence, such as conformity, obedience, persuasion, compliance, social norms, deindividuation, reactance, social facilitation, identification, social loafing, and even attraction and aggression should be thoroughly evaluated and, if need be, reformulated. In this way, knowledge may advance more systematically and expeditiously in the area of social influence, and therefore in social psychology.

APPENDIX

Chronological Listing of Social Science Dictionary and Textbook Definitions of Contagion

English and English (1958) defined *social contagion* as "the spontaneous imitation, by other persons in a group, of a behavior initiated by one member but without overtly shown intention to stimulate such imitation" (p. 117). They contrasted social contagion with direct influence, "in which a person manifestly intends to affect the behavior of another or others" (p. 117). English and English also offered definitions for two other contagion-related terms. *Mass contagion* was defined as "the spread of behaviors among large numbers of people

who are not gathered in one place or not identified with one another" (p. 117); *group contagion* was defined as "the rapid spread of feeling—[especially] fear, anger, amusement, or relief—through an assemblage, caused by perception of the feeling in others" (p. 231).

Chaplin (1968) defined *contagion* as "the spread of behavior patterns through a social group as a consequence of suggestion" (p. 113).

Eysenck, Arnold, and Meili (1972) defined *psychic contagion* as "a supposed process by which certain behavior (e.g., rhythmic movement) is rapidly passed from individual to individual to affect a whole group" (p. 217).

Wolman (1973) defined *contagion* as "the rapid spread of behavior or feelings to other people through suggestion, imitation, or sympathy" (pp. 77-78). He also defined *mass contagion* as "the rapid spread of behaviors among groups of unrelated people who are not necessarily in the same area" (p. 78).

Stang and Wrightsman (1981) defined *contagion* as "the spread of affect or behavior through a crowd" (p. 19).

Raven and Rubin (1983a, 1983c) defined *social contagion* as "the spread of behavior, attitude, or emotional state among the members of a group or social organization in a manner resembling the spread of a contagious disease" (1983c, p. G-21). They maintained that major factors leading to contagion include deindividuation, loosened restraint, satisfaction, and the group's tendency toward extreme behavior. As a dramatic illustration of this phenomenon, the authors cite Clark's (1940) story, "The Ox-Bow Incident."

Raven and Rubin defined *hysterical contagion* as "a form of social contagion in which a number of members of a group or other social unit experience physical symptoms (such as nausea, breathing difficulty, muscular weakness) which appear to result from social influence rather than from an initial physiological malfunction" (1983c, p. G-11). Examples of this psychogenic phenomenon include a fainting epidemic among English schoolgirls (Moss & McEvedy, 1966), the "June bug" incident (Kerckhoff & Back, 1968), and the dancing mania of the Middle Ages (Hecker, 1837/1970; Major, 1955). The authors noted that "just as opinions, beliefs, and behaviors may spread through a group in a systematic fashion, so may the hysterical symptoms of severe physical illness" (p. 550).

Worchel and Cooper (1983) defined *behavioral contagion* as "reducing the restraints to perform a particular act by observing a model performing that act" (p. G). The authors distinguished contagion from conformity by noting that in the former, the conflict about performing a behavior is present before the model acts, whereas in the latter, the model's behavior creates the conflict.

Goldenson (1984) defined *contagion* as "the transmission of ideas, feelings, or mental disorders from person to person or group to group by psychological forces such as suggestion, propaganda, rumor, imitation, or sympathy" (p. 176). He referred to *group contagion* as "an outmoded term denoting the communication or transmission of emotion through a group or crowd, e.g., the rapid spread of fear" (p. 326).

Reber (1985) defined *behavioral* (or *emotional*) *contagion* as the "spread of an activity or a mood through a group" (p. 152), and *social contagion* as "the rapid spread of attitudes, ideas, or moods through a group or society" (p. 706).

Sears, Peplau, Freedman, and Taylor (1988) defined *contagious violence* as the "spreading of the aggressive feelings or behavior of a small number of people throughout the entire group" (p. 572). The authors viewed contagious violence as a form of imitative aggression and maintained that it is an important factor in crime, crowd behavior, and mob violence. They noted that although there is substantial controversy regarding the reliability of research in this area, some studies (see Phillips, 1986) indicate that the publicity given to suicides, prize fights, and executions induces contagious-like imitative acts.

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