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by Jacqueline Schor

Predisposing Factors for Post Traumatic Stress Disorder: A Comparison of Environmental and Cognitive Factors

Post Traumatic Stress Disorder (PTSD) is an anxiety disorder involving stress reactions subsequent to exposure to intense trauma. According to the Diagnostic and Statistical Manual of Mental Disorders (fourth edition) (DSM IV), the trauma may either be experienced personally or witnessed, and must involve "actual or threatened death or serious injury, or threat to the physical integrity of self or others" (p. 427). Reactions to the trauma include recurring distressing thoughts, images and dreams regarding the incident, as well as psychological distress or physiological response to a stimulus which functions as an actual or symbolic representation of an aspect of the traumatic event. The patient may also act or feel as if the traumatic experience is actually recurring; this may take the form of hallucination, illusions, dissociative flashback episodes, etc., and may occur in a wakened state or while intoxicated.

A patient experiencing PTSD will try to avoid stimuli which may be associated with the trauma, which may include avoidance of certain people, feelings, or weather conditions, as well as impairment of memory regarding information pertaining to the trauma. The patient will be generally unresponsive, with an affect which is restricted (relative to the state of the affect prior to the trauma). He or she may exhibit diminished interest in activities as well as general detachment. Additionally, PTSD involves increased arousal, and the patient may experience hypervigilance, difficulty falling asleep, and irritability. The totality of symptoms must be such that general functioning is significantly impaired.

Stressors which may induce PTSD are varied. While war and combat-related traumas compose a large number of the events which trigger PTSD, any significant trauma may serve the same function. Common examples are rape (and other forms of assault), child abuse, natural disasters, life-threatening illnesses, automobile accidents, and kidnapping/hostage situations (DSM IV, 1994).[2]

While trauma acts as a necessary trigger for PTSD, it is clearly not a sufficient condition, since not all people involved in traumatic events develop PTSD. Severity of trauma is widely accepted as a direct cause of the incidence and the severity of PTSD (Berg, Watson, Nugent, Gearhart & Juba 1994; Brendt, Perper, Moritz, Liotus, Richardson, Canobbio, Schweers & Roth, 1995; Marmar, Weiss, Metzler & Delucchi, 1996; McNally & Shin, 1995; Penk, Robinowitz, Black, Dola, Bell, Dorsett, Ames & Noriega, 1989; Shalev, Peri, Canetti & Schreiber, 1996; Sutker, Davis, Uddo & Ditta, 1995; Watson, Anderson & Gearhart, 1996). However, several people may witness or be participants in the same event, and one person may develop PTSD while the others does not (Brendt et al. 1995). Therefore, in keeping with the theory of diasthesis stress (Sutker et al. 1995), it must be concluded that in order for a case of

[1]It should be noted that DSM III R includes in its description of criteria for assessing an event as traumatic that the event is "outside the range of usual human experience and that [it] would be markedly distressful to almost anyone..." (DSM III R pp 251-2). This is noted because the majority of the research discussed in this article makes use of DSM III R as a source of diagnostic criteria for PTSD. PTSD diagnostic criteria in DSM III R is otherwise identicle to PTSD diagnostic criteria in DSM IV.

PTSD to develop there must be present both a trigger event and a preexisting condition which is affected by the trigger. This preexisting condition results from one or multiple factors which pertain to the patient and predisposed him or her to PTSD. The exact nature of these factors is a matter of dispute. In this article I will discuss environmental and cognitive characteristics which act as predisposing factors to the development of PTSD. It is my contention that the cognitive factors are a more convincing primary cause. It is possible for these cognitive factors to be created or encouraged by environmental factors.

Environmental Factors

The emphasis on social factors stems from the concept that a person's reaction to severe trauma is dependent on his or her social network. There are two main categories within the social network. First, the family is examined; a social approach is used, to the specific exclusion of biological influences. Second, the effects of large social grouping are studied, from racial and religious points of view.

Family

Watson, Anderson and Gearhart designed a study to investigate the effects of psychosocial maladjustment within Vietnam veterans' families on the veterans' development of PTSD (1996). Using the psychiatric and chemical dependency wards of a veterans hospital as their subject pool, they selected a group of veterans with PTSD and a group of veterans who had never developed PTSD. They also selected a group of subjects who were workers in the hospital. The experimenters collected data on each subject's father, mother, oldest brother and oldest sister. Each family member was assessed for history of incarceration, history of institutionalization, and history of inpatient or outpatient psychiatric treatment. This information was then compared with the subject's group status (PTSD, psychiatric control, employee control).

Statistical tests revealed the only significant condition within the study to be that the older brothers of the psychiatric controls were more likely to have been incarcerated than were the older brothers of the employee controls. Therefore, no correlation was found between psychosocial maladjustment within one's family and the development of PTSD. However, even if there had been significant results, it would be difficult to assess the realm of their validity. The family members selected were restricted to only full-blooded first degree relatives, but the researchers' aims were clearly not biological, in that they only studied relatives who were well known to the subjects, who had lived with the subject for at least five years since the subject's ninth birthday. In a sociological context, it is difficult to understand the exclusion of step relatives and adoptees who fell within these parameters.

Certain familial factors have been shown to predict resiliency to life stressors within young children (Wyman, Cowen, Work, Raoff, Gribble, Parker & Wannon, 1992). Children in grades four through six whose lives contained a significant number of stressful events (e.g. a close family member with an alcohol or drug problem) were screened for general adjustment, using Global Rating scales administered to both the child's parents and teacher. The researchers then observed whether the children's adjustment levels were related to various variables including the nature of their relationships with their care-givers, stability of the family environment, and the inductiveness and consistency of family discipline. The researchers found that resilient children, those with high adjustment scores, reported a closer relationship with primary caregivers, in a more stable family environment, with more inductive, age-appropriate, and consistent discipline practices.

While the research of Wyman et al. studied children's dealings with stress and did not specifically study individuals with PTSD, a different study of adolescent peers of suicide victims who exhibit PTSD revealed

similar results (Brendt et al., 1995). Statistics revealed that both mother-child relationships and interparental relationships were more discordant among subjects with PTSD (father-child relationships showed a similar tendency but were not statistically significant).

Large Social Grouping

Of all of the possible categorizations by which to classify subjects, perhaps the most tempting is by race. Penk, Robinowitz, Black, Dola, Bell, Dorsett, Ames and Noriega (1989) researched the differences in incidence of PTSD between black, white and Hispanic veterans under differing levels of combat in Vietnam. The study was based on earlier research by Penk, Robinowitz, Dorsett, Bell and Black (1988, as quoted by Penk et al. 1989), which revealed that black combat veterans who sought treatment for substance abuse are more disturbed than white Vietnam combat veterans (Penk et al. 1989, p 730). Penk et al. (1988) cite findings that black heavy combat veterans exhibited less effective coping and problem solving than did white heavy combat veterans (Penk, Peck, Robinowitz, Bell & Littl, 1988, as quoted by Penk et al. 1989). Penk et al. (1989) also noted speculations by Laufer, Yager, Frey-Wouters, Donnellan, Gallops and Steinback (1981, as quoted by Pank et al. 1989) and by Parsons (1985, as quoted by Penk et al. 1989) that minorities as a whole would exhibit greater PTSD symptomotology. Their reasoning behind the theory was that minorities had less to gain from being in the war than whites had to gain, because minorities themselves were branded as the enemy.

Based on these studies, Peck et al. (1989) compared black, white and Hispanic veterans in order to determine whether the difference between blacks and whites is a result of blacks being a minority (under the assumption that if this was the case, the effects would generalize to the Hispanic population as well). Subjects were obtained from among Vietnam veterans being treated for addiction disorders. Under each of the ethnic headings, subjects were divided into categories of either no combat, light combat or heavy combat. This division served two purposes: first, it

controlled for level of combat within subjects (level of combat being a significant cause of PTSD incidence and severity, as stated above), and second, it allowed the experimenters to examine the differences between the subjects on three different levels of combat. All subjects were administered the Minnesota Multiphasic Personality Inventory (MMPI) and results were compared. Blacks were found to differ significantly from Whites and Hispanics, revealing more PTSD symptomotology and incidence, especially in the category of heavy combat. Whites and Hispanics exhibited similar results.

Based on these results, the researchers formed a new theory to explain Black differentiation. Quoting a 1945 study by Grinker and Spiegel, the researchers explain that maladjustment associated with combat is related to loss of faith- a loss of faith in leadership, a loss of faith in the legitimacy for participating in combat, and a loss of faith in personal and social support from one's friends, relatives and fellow citizens (Penk et al. 1989 p 734). The nineteen-sixties and nineteen-seventies were a time of great insecurity for the blacks, based on the assassination of Martin Luther King Jr. and other related factors. They were therefore predisposed to enter into war under nonconducive conditions. However, this conclusion is tenuous. Grinker and Spiegel's study is based on World War II. Penk et al. is based on the Vietnam war, in which circumstances differed radically in terms of cause of the war, outcome, United States involvement, etc.

A second example of a large social grouping is that of a religious network. Anson, Carmel, Bonneh, Levenson and Maoz (1990) studied the effects of membership in an organized religious group on physical and mental health. The researchers selected two kibbutzim, communal living-places in Israel, one of which was religious and one of which was not. They then assessed the members of each kibbutz regarding the number of difficult recent life events (RLE) present in the previous year of each of their lives. Examples included job loss and a family member joining the army. The results of this assessment established categories

into which the kibbutz members were placed based on the number of RLE's experienced. The researchers also assessed religiosity based on five measures, three of which were based on outward, contextual evidences of religiosity (e.g. practice of specific religious practices, the practice of which places the respondent within a definite, widely recognized religious community) and two of which were based on more private manifestations which reflected spirituality more than identification with a specific sect (e.g. private praying). Finally, the researchers assessed the kibbutz members for physical health and an increase in psychological distress.

The researchers found that an increase in RLE's was generally positively correlated with a decrease in physical health and an increase in psychological distress. Regarding members of the religious kibbutz, however, detriments caused by RLE's were markedly less present than were determents caused by RLE's in members of the non-religious kibbutz. Anson et al. theorize that RLE's and religiosity are not intrinsically interrelated. Instead, RLE's exert a negative force on physical and mental health while religiosity exerts a positive force, reducing the negative effects of the RLE's.

In order to determine whether the difference they had measured was societal or personal, the researchers studied the correlation of RLE's health and distress, and religiosity as determined by only the answers to the last two questions (private praying, and theodicy: the extent to which one finds comfort in religion during distressful times). While common sense would dictate high correlations between high personal religiosity and good health and less distress (in that it would appear that a person who finds comfort in religion during distressful times would have a less intense reaction to intense stressors because of his or her interpretation of that stressor as less potentially harmful), Anson et al. found a much lower correlation using the personal level of religiosity than they found using the communal level.

Unfortunately, Anson et al. provide no possible reasons

behind these results. The results remain perplexing from a cognitive point of view. It therefore becomes necessary to examine cognitive theories regarding predisposing factors to PTSD.

Cognitive Factors

Cognitive factors related to the development of PTSD involve a variety of thought patterns which may foster development of this anxiety disorder. Pertinent factors involve intelligence of the subject (as measured by IQ, as opposed to level of education), moral development, perception of social support, coping styles, and overall hardiness, a term which describes the characteristic manner by which individuals interpret and approach experiences (Sutker et al., 1995).

Intelligence

Several studies have attempted to establish level of education received as a predisposing factor to PTSD (Shalev et al. 1996; Sutker et al. 1995). This factor is difficult to isolate, as it usually comes as a part of the social package of socioeconomic status, which may be related to numerous factors such as race (Penk et al. 1989) and poverty level (Shalev et al. 1996). Intelligence, on the other hand, is objective and isolatable. McNally and Shin (1995) assessed intelligence in male Vietnam combat veterans using the Shipley Institute for Living Scale, which measures general intelligence (the test has a reliable correlation with full-scale IQ as measured by WAIS-R). The researchers found that, controlling for socioeconomic status, race, age and preservice health, IQ predicted ten percent more variance in PTSD symptoms than did combat exposure, and three percent more than combat exposure and years of education combined (both percentages were significant).

There are several necessary comments regarding this study. First, the researchers note that their results may be slightly inaccurate, owing to

their controlling for years of education. This is because years of education are often a function of IQ, and therefore controlling for them may alter the effect of intelligence on PTSD symptoms. Second, while it is tempting to conclude that the isolation of IQ, an internal versus environmental factor, ensures a cognitive element in predisposition for PTSD, this conclusion cannot be validly reached through use of this study. McNally and Chin's IQ assessments took place subsequent to the development of PTSD symptoms within PTSD subjects. Therefore, it is impossible to determine causality; lower IQ may be a result of PTSD.

As opposed to intellectual development, Berg, Watson, Nugent, Gearhart and Juba (1994) studied the effects of moral development on PTSD symptomotology. The researchers evaluated moral development in their subjects using the Revised Defining Issues Test, an assessment tool designed to operationally define Kohlberg's scheme of moral development. (It thereby measures moral reasoning as opposed to behavior, in that it presents Kohlberg's scenarios and asks the subjects what they would do, versus what they have done.) Like Penk et al. (1989), they established groupings based on severity of combat experience. Differentiating between high and low combat groups, the researchers found that regarding low moral development subjects, combat severity was extremely significant in determining PTSD severity. Contrarily, regarding subjects with high moral development, the effects of combat severity on PTSD severity were not significant.

The researchers speculate as to what may cause this inverse correlation between moral development and PTSD symptoms. They cite research by Hendin and Hoss (1984), who hypothesize that higher moral development may lower the probability of the subject's having participated in atrocities. Berg et al. also speculate further:

> ... moral immaturity ... may impede the development of cognitive strategies needed to deal with extreme stress, resulting in the large

correlation between combat severity and PTSD in the low moral development sample. It also seems possible that lack of religious faith, and the perceived emotional support that accompanies it, might be prominent in men with low moral development scores... (Berg et al., 1994 p 675).

Perception of Social Support

Within the above statement, the researchers have touched on many of the possible cognitive factors discussed in this article which may appear to be environmental in nature (cognitive strategies will be discussed subsequently). For instance, religious faith is an individual variable, which is purposefully excluded by Anson et al. (1990) in their study of the effects of organized religion. The fact that religious faith is a part of a social framework does not necessitate that its own nature or its source of impact is social.

Additionally, perceived emotional support is a cognitive perspective on environmental factors; it regards the subject's perception of his or her environmental factors, as opposed to an objective evaluation of the specific facts pertaining to the subject's environment.

Wyman et al. found that, beyond life events and parental education level, there were four main factors which determined stress resilience in children. All of these were related to the child's perception, three concerning family support (perception of more consistent family discipline, perception of a stable, positive family environment, and perception of reasoned, inductive discipline approaches). While the researchers made predictive statements about mother-child relationships and stable family environments, their descriptions of the situations within each child's home were elicited from the child (the study did make use of reports from parents, but only in order to classify the children on a scale of adjustment). Therefore, the study measured the effects of the children's perceptions, not

their actual environments.

Sutker, Davis, Uddo, and Ditta (1995) were cognizant of the nature of the social variable. Using a general social support questionnaire along with a family support index, both subject self reports, they determined that less perceived family cohesion and expressiveness and less satisfaction with social supports served as predisposing factors to PTSD. The Social Support Questionnaire was specifically designed to measure perceptions.

Coping Styles

Marmar, Weiss, Metzler, and Derlucchi (1996) studied the effects of various variables on peritraumatic dissociation within emergency service personnel. Peritraumatic dissociation is a dissociation which takes place during the trauma experience itself and is thought to be a major precursor to and predictor of PTSD (Shalev, Peri, Cenetti, & Schreiber, 1996; Spiegel, Hunt, & Dondershire 1988 as quoted in Shalev et al., 1996). Among other variables, Marmar et al. studied coping styles using the Ways of Coping Questionnaire. The researchers studied three types of coping mechanisms: escape-avoidance, which is "wishful thinking and behavioral efforts to escape or avoid dealing with the critical incident" (Marmar et al. 1996, p 97), self control, which is "effort to regulate one's feelings and actions by keeping them to oneself and trying to keep feelings from interfering with other things too much" (Marmar et al. 1996 p 97), and planful problem solving, which is "deliberate problem-focused efforts to alter the situation, coupled with an analytic approach to solving the problem" (Marmar et al. 1996 p 97).

The researchers found that escape-avoidance and self control were strongly positively associated with peritraumatic dissociation. Planful problem solving was also positively associated, although to a lesser degree. Wishful thinking and self-blame coping strategies have also been found to

be positively associated with PTSD (Sutker et al. 1995). This study, however, was conducted after the onset of PTSD, and therefore cannot indicate causation. The results, though, have been replicated numerous times (Wolfe, Kalouppek, Mora, & Wine 1993, Fairbanks, Hansen, & Fitterling 1991 and Solomon, Mikulincer, & Avitzur 1988, as quoted in Stuyker et al. 1995).

Hardy Personality Type

Coping style, however, may not be a factor on its own but may be a component of personality style, a more widely defined category which includes many factors which may predispose a person to PTSD (Marmar et al 1996; Sutker et al. 1995). Effective coping skills (defined as those coping styles not predisposing PTSD) are only one trait associated with a hardy personality, a personality style strongly negatively correlated with PTSD (Marmar et al. 1996; Sutker et al. 1995). In fact, Maddi and Kobasa (1984) (as quoted in Sutker et al. 1995) theorized that "the personality disposition of hardiness, with its link to coping strategies, is pivotal to the relationship between social support phenomena and stress symptoms" (Sutker et al 1995). Simply stated, a person's perceptions, which have been previously stated to act as predisposing factors to PTSD, result from his or her personality style, which is thereby the ultimate predisposing factor.

Hardiness is made up of several components, all of which may influence development of PTSD and therefore each of which must be mentioned. Marmar et al. (1995) includes the factors of strength of identity development and of locus of control, finding that non-PTSD subjects were characterized by stronger identities and by internal loci of control. The Dispositional Resilience Scale, used by Sutker et al. (1995), measures three main components of PTSD: "commitment or sense of meaning, purpose, and perseverance attributed to one's existence; control, or sense of autonomy and ability to influence one's destiny and manage experiences; and challenge, or perceptions of change as exciting growth opportunities" (Sutker et al. 1995) (the commitment variable might have been the variable

which Berg et al. 1994 posited might have been added by a subject's being religious (Anson et al. 1990)). All three of these variables were found to be significant indicators of PTSD, and combined, they were found to be the most influential factors regarding whether or not the subject had PTSD, accounting for 26% of variance within the data. (It should be noted that there are claims that the hardiness scales accidentally measure neuroticism, an acknowledged predisposing factor to PTSD (Breslau, Davis, Andreski, & Peterson, 1991, as quoted by Sutker et al. 1995). This would invalidate any results obtained using these scales (Funk 1992, as quoted by Sutker et al. 1995).

This data supports the conclusion of Kobasa, Maddi, and Kahn (1982, as quoted in Sutker et al. 1995). Kobasa et al. (1982) explained their data by positing that people high in commitment have a sense of purpose, which lets them identify meaning in life events. Therefore, they interpret events as less stressful and can thereby better cope with them; their interpretations may also influence their social support and their perceptions of their social support.

While this conclusion makes sense and elucidates the effects of a variety of factors discussed earlier in this article, it may nevertheless be difficult to assign validity to the study which fostered the conclusion. This is because, like most studies regarding factors which predispose the development of PTSD, the study was conducted after PTSD development. This study, then, cannot demonstrate causation, and can only show that people who are afflicted with PTSD do not possess hardy personalities.

There is a study, however, which has the ability to salvage the theory of hardiness. In 1963 and 1964, the Dartmouth College classes of 1967 and 1968 took the MMPI as freshmen. Between 1985 and 1987, Schnurr, Friedman, and Rosenberg (1993) distributed military history questionnaires to the class members, and compared the results to the information obtained by the pre-military MMPI administrations.

The researchers analyzed each scale of the MMPI to ascertain which scales' results were most predictive of later development of PTSD. Combat veterans with any lifetime PTSD symptoms had higher scores (and often high scores in general) on the sales for hypochondriasis, psychopathic deviate, masculinity-femininity and paranoia. The scales found to be most positively associated with later PTSD symptom development were masculinity-femininity and psychopathic deviate. Schnurr et al. interpreted their findings as follows:

> Normal-range scores on the psychopathic-deviate scale are positively correlated with self-reports of gloominess, dissatisfaction, impulsivity, and irritability, and normal-range scores on the masculinity-femininity scale are positively correlated with inhibition, shyness, withdrawal, and consicientiousness. This suggests that men who are less happy and more withdrawn and inhibited than their peers may be at greater risk of developing lifetime PTSD symptoms if exposed to combat (Schurr et al. 483).

This description stands as direct opposition to the optimistic, positive, empowered description of the hardy personality, and therefore may well corroborate the conclusions regarding the predispositional power of hardiness. The study does only establish that these characteristics are associated with the above-mentioned scales, and therefore there may not be causation. However, these factors will then at least function as positive indicators that the disorder will develop.

Conclusion

There exists great difficulty distinguishing between the environmental and cognitive influences. Any of the cognitive factors discussed in this paper may be environmental in origin. Membership in a religious group may aid the development of identity and morality, and a positive family environment exercising consistent discipline may foster the development of an internal locus of control. All of the factors are

intertwined, making the dichotomy between environmental influence and internal cognition a difficult one to solve.

Based on the research discussed in this paper, a strong conclusion may be reached that the cognitive factors are the more significant factors, outstripping social/environmental factors. This conclusion is supported by several studies in which the researchers controlled for factors such as ethnicity, education, and socioeconomic status. While environmental factors may have been the cause of the cognitive factors which predispose PTSD development, it is the cognitions which act toward PTSD development. The cognitive thinking patterns are necessary factors, which may be caused by a variety of other factors including both internal variables and a variety of possibilities concerning environment. It is necessary to determine not what factors may in some way predispose a person to the disorder, but which factors are the actual pivot-points on which development of the disorder rests. It is another study to determine what factors may shape those points in that specific fashion.

The largest impediment to forming a solid conclusion based on the research presented in this article is the fact that only one study involves reliable data describing pre-trauma characteristics. While the data do not contain results from an actual hardiness assessment , or even a PTSD assessment, they represent the most clinically-valid pre-trauma data included among the research discussed. In fact, studies based on pre-trauma evaluation are a rarity in PTSD research in general. The studies regarding lifetime resilience (Anson et al. 1990; Wyman et al. 1992) are also difficult to interpret within the context of predisposing factors. It is difficult to distinguish whether the nature of the data is pre, peri, or post-traumatic. The fact that it is a combination of the three, that the phenomenon being studied is a consistent one as opposed to relating to one specific event, creates difficulty in generalizing the results of those studies to form a conclusion regarding PTSD. In order to properly determine

predisposing factors to post-traumatic stress disorder, it is necessary to do more research involving pre-trauma data. This being a practical improbability, however, the data present reveals important cognitive factors which seem to be important and constant in the development of PTSD. Environmental factors, while also present, are more peripheral in nature.

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by Gila Liska

Etiology of Alzheimer's Disease: The Genetic Theory

Alzheimer's Disease (AD) was first identified and described in November 1906 by the German physician Alois Alzheimer. He had observed a rapidly developing dementia in a fifty-one-year-old woman which differed clinically and pathologically from all other disease processes at the time. The symptoms he had found were memory loss, disorientation, depression, hallucination, and eventually severe dementia and death. An autopsy of the patient's brain showed severe atrophy, a decay or decrease in size of an organ or tissue, of the cerebral cortex. It also revealed a clumping and distortion in the nerve cells of the brain. These neuro-fibrillary tangles, along with amyloid plaques, have become the identifying factor of AD.

When similar cases were identified, a controversy arose as to whether Alzheimer's disease should be described as an entity separate from senile dementia or should be considered an advanced form of senile dementia. As of yet, this controversy has not been resolved.

Being that it is an age-related disease, Alzheimer's is becoming an increasingly important and deadly syndrome due to increasing lifespan. Approximately four million Americans have AD, and unless a cure or prevention is found, fourteen million may have it by the middle of the next century. The disease is the fourth leading cause of death among adults.

The onset of AD is a slow and nebulous process. It is manifested by mental changes usually occurring between the ages of forty and sixty. The duration of the illness varies, but can be set at an average of two to

five years, with death usually due to an incurrent infection. However, patients have lived with the disease for over twenty years.

A distinction must be made between two types of Alzheimer's: pre-senile, early onset and senile, late onset. Early onset begins before the age of sixty and shows relatively rapid deterioration, specifically involving language impairment. It is also marked by many disorders of the high cortical functions. Aphasia, agraphia, alexia and apraxia occur relatively early in the course of the dementia, whereas senile Alzheimer's, which is more common than the latter one, is marked through a slow progression with memory impairment commonly found as its principle feature.

Symptoms of AD involve many different areas of human functioning and pathology. Examples include cognitive changes and specifically memory deficits, temporal and spatial orientation, attention deficits, verbal deterioration (aphasia), visiospatial incompetence, emotional disorders, personality changes, destructive behavior, hallucination and delusion.

There are several theories of etiology which offer possible explanations of a causal relationship. However, one must be careful to distinguish between real causes and mere risk factors that might be correlationally but not causally connected to Alzheimer's. The four dominant theories are:

(1) *The Toxic-Exposure theory.* This theory explains the connection between trace metals such as aluminum and the development of AD.
(2) *The Cholinergic theory.* This hypothesis accounts for the loss and degeneration of neuronal population which are caused by a 60-90% loss of choline acetyltransferase in the cerebral cortex and the hypocampus.
(3) *The Infectious Agent theory.* This explanation is derived from other kinds of dementia like Kreuzfel-Jacobs

or Kuru that are viral infections.
(4) *The Genetic theory.* This theory will be discussed subsequently.

In order for any of these theories to be fully adequate they must fulfill three criteria. First, it must account for the development of plaques and neurofibrillary tangles. Second, it must include the five confirmed risk factors, which consist of age, family history of AD, head trauma, Down's syndrome, and a family history of Down's syndrome. Third, it must enable scientists to make valid and testable predictions about the disease. Considering the contemporary stand of today's knowledge, it is difficult to fully achieve all three criteria. Thus it is understandable that there is still no unified satisfactory theory offered on the causes of AD. Nevertheless, researchers have been encouraged through recent findings and research continues to advance.

Due to the vast amount of research involved, this article will focus upon only the genetic theory. This theory has been the subject of heated debates and of yet has not been completely validated. However, this article will attempt to show that there is strong evidence to support the belief that the genetic theory shows not only a correlational effect regarding AD but also a causal effect .

Aside from the opinions that completely reject the veracity of the genetic theory, there are scientists who argue that the genetic factors alone cannot account for the occurance of AD. Rather, when the genetic factors are combined with other factors i.e. toxic exposure or an infectious agent, they play an important role in the etiology of AD. Although this idea is beyond the scope of this article, since it would involve explaining the accompanying factors, it is a strong argument and makes the genetic cause more plausible. It will therefore be assumed that the reader is aware of this interplay and will understand that when discussing hereditary or familial Alzheimer's disease as a cause, it is intended not as being the sole cause

but part of a conglomeration of effects.

General Concepts

Familial Alzheimer's Disease (FAD)

Substantial evidence points to the fact that there is a strong hereditary component to the Alzheimer's disease, especially when onset occurs before the age of seventy (Shalet et al., 1987). Heyman et al. discovered that up to 50% of AD patients had a family history of that same disease, meaning that AD is significantly associated with dementia in first and second degree relatives (1983). Therefore, people that have family members with AD, especially first degree relatives, are 4.3 times more likely to contract the disease. Some other researchers estimate the incidence to be around 35-40% (Amaducci et al., 1985). When it appears in succeeding generations of a family it will be apt to occur as an autosomal dominant trait, meaning that half of all offspring of the affected parent will develop the disease (Fitch et al., 1988; U.S. Congress, 1987). The highest association is with the presence of dementia in siblings (Amaducci et al., 1985) Researchers have found an increased incidence of AD among parents and siblings of victims. Some show a slight increase, while others show a considerable difference from the occurrence of AD in the general population.

A near to 100% agreement regarding identical twins would be expected if AD followed a straight genetic pattern. One study done, however, failed to extract these results. This has led to much questioning of the genetic theory (Schellenberg et al., 1988; Tanzi et al., 1987). However, this is no reason to discard the genetic theory completely. Instead, the other interacting factors mentioned above must be included.

There are, however, general problems that arise in researching FAD for several reasons. Firstly, patients with more than one relative affected by AD are more likely to be included in case-control studies

because they are usually issued by hospitals which have their records on file. This is a selection bias and excludes complete random sampling. Therefore, generalization to the rest of the population is difficult. Secondly, the report of AD in the family is usually done by family members. These members will be more aware of the occurrence of the disease than subjects of the control group, since there already is such a case in the family; this would increase the likelihood of reporting AD in patients from families containing other AD patients, again damaging the random sample.

In addition to this bias, another problem arises, since the AD might be the result of some environmental influence to which the whole family was exposed. Further problems in researching the genetic factors arise since some possible gene-carriers die before they get old enough for the disease to set in. This explains why evidence for the genetic theory is derived mainly from individual families with extreme cases of early onset AD. Thus one might assume that hereditary Alzheimer's is only of the presenile type. This, however, is an invalid conclusion, since, as explained above, conclusive studies regarding late onset dementia could not be performed due to mere technical difficulties. These are all serious challenges to the genetic theory which must be solved before the theory can be validated. However, these challenges are not sufficient to completely falsify and reject the theory.

Age and Genetics

Aging is a genetically preprogrammed process brought about by specific genes that shut down certain processes. These processes are formally activated by the genes during a child's development. Every bodily function has certain genes responsible for terminating its specific function, including the brain, specific memory, and intelligence. Because every person has individual biological differences, gene-material timing will vary from person to person. When the brain process terminates earlier than normal, it is called Alzheimer's disease (Wright & Whalley, 1984).

This view explains the relationship between age and AD. In other words, it accounts for the correlation between the increase of the two variables at hand.

Although successful in explaining the age factor, this view still has its limitations. It offers a solution only to late-onset AD, and not for early-onset. Furthermore, the individuality of the aging process isn't as flexible as stated above. AD is not a mere diversion of the normal aging process. It is a clear malfunction which is not explained by this theory but merely replaced by different terminology.

Down's Syndrome and Alzheimer's

Another interesting association has been found between AD and Down's syndrome (DS). DS is a form of mental retardation which is accompanied by skeletal and other developmental anomalies. Virtually all people with DS develop the brain changes of Alzheimer's in middle age (Lai & Williams, 1989); 15-50% of the subjects also develop clinical evidence of dementia. Heston and associates (1981) found a prevalence of DS among relatives of AD patients that was higher than that of the general population. (Similar results were found by Heyman, Wilkinson et al., 1983).

DS patients have an extra copy of chromosome 21. This leads to an extra supply of genes on that chromosome. As a result, there is an overabundance of certain essential chemicals and substance produced by these genes. A certain product of these genes is probably important for the development of AD (Heston, 1984). Hence, both AD and DS have been localized to chromosome 21, specifically on the long arm of the chromosome (Jarvik, 1988; Pirozzolo, Inbody et al., 1989).

The way in which this superfluous chromosome 21 is created is through an error in reproduction. When the cells part in order to make 23

chromosomes out of 46 in the sex cells, an extra chromosome can accidentally generate. This is what causes both DS and AD. Not only does this explain the correlation between the two diseases, but it also explains what role neurofibrillarily tangles play in both. Neurofibrillarily tangles could derive from neurofilaments in the nerve cells which are important for the division of cells. Therefore, both diseases may result from a basic defect in the neurofilaments.

Although there are many similarities between AD and DS, it is difficult to determine when a DS patient really has a form of dementia. The DS patient is by definition limited in intelligence, making it difficult to identify dementia, which is characterized by a loss of intelligence. Even if dementia is evident, it will not appear in DS patients in the same way that it would appear in individuals of normal intelligence. Therefore, it is difficult to diagnose AD once DS is present.

Specific Genetic Causes

APP Mutations

It has been discovered that a number of people with early onset FAD have amino acid substitutions in the amyloid precursor protein (APP) which is encoded by a gene on chromosome 21 (Murrell et al., 1991). Mutations, or changes in how it travels through the cell, cause AD by overexpression of APP through gene dosage effect in exons 16 or 17 (Chartier-Harlin et al., 1991; Goate et al., 1991; Murell et al., 1991; Karlinsky et al., 1992). These exons encode the 42 amino acid fragment of APP which is released as the B-peptide, a principle component of senile plaque. In this manner the balance is tipped because of the change and the beta-amyloid deposition is increase (Science, 1993). The frequency of these mutations occurring as cause for AD is, however, very low (lower than 3%) (Kamino et al., 1992; Tanzi et al., 1992). Further doubt arises because the duplication of the amyloid gene has not been observed in AD. Furthermore, St. George et al. (1987) argued that although they are

located on the same chromosome, the gene for AD and the amyloid gene are not linked in any way. This argument may be debated because a link was discovered between D21S1 and D21S16 by Goate et al. (1989). However, this link was found in only one family out of six tested (Pericak-Vance et al., 1988).

Chromosome 14 and protein S182:

Recently a gene identified as S182 (Sherrington et al., 1995) located on chromosome 14 (Schellenberg, G. D. et al., 1992) was found to be involved in 70-80% of early onset AD cases. St. George-Hyslop et al. searched for an understanding of the gene's product. This product, a membrane protein, seems to be the cause of AD, although its function is yet unknown. Large numbers of mutations of the gene that leads to the disease have been discovered and more are expected. Resemblance of S182 to SP-4, a membrane protein needed for protein transport, might imply that it too is involved in protein transport (Steven L'Hernault, 1996), specifically the formation of beta-amyloid which makes up the senile plaques. Beta-amyloid is clipped off of a larger protein called APP. Some say it is clipped off right in the middle of the beta-amyloid sequence after being transported by vesicles to the cell membrane, releasing beta-amyloid into the cell. Therefore, a mutation in APP or a change in the way it travels may produce an increase in beta-amyloid and in return cause the disease (Science, 22 January, 1993). Tanzi speculates how this change comes about. He maintains that the S182 protein plays a role in the packaging of APP into the vesicle and its delivery to the specific loci in the neuron. Thus it is understandable why a defect in this gene would cause difficulties in transport of APP. He further speculates that APP might be brought to an area during the course of this malfunction, in which it is more likely to cleave to beta-amyloid. But as Selkoe points out (Science, June 30, 1995), it is crucial to bear in mind that this is merely speculation and has not been proven.

Beta-amyloid can also affect the functioning of brain cells in modifying ion-channels (Aripspe et al., 1993), altering the transport of choline (Galdizicki et al., 1994) disrupting phosphatidylinositol metabolism, and reducing synaptic field potentials (Lancet, December 9, 1995). Moreover, beta-amyloid is a constituent of the neurotic plaques which have been observed in every AD patient. In light of the evidence for these multiple functions of beta-amyloid, it does seem that this protein is a central force in causing AD.

Chromosome 1 and STM2

Schellenberg et al localized a protein on chromosome 1 which is homologous in amino acid sequence to S1821 (Clark et al., 1995; Levy-lahad et al., 1995). Because of their similarity they have the same effects on a neuron when mutated. Luciano D'Adamio and his colleagues have found that STM2 may be involved in programmed cell death (apoptosis). A mutation in this protein could trigger the death of neurons; this would account for memory loss and intelligence deficiencies.

Chromosome 19 and Apolipoprotein-E

Recently, Rose et al. found genetic linkages between the gene encoding for apolipoprotein-E (Apo-E) and marks on chromosome 19 in late onset Alzheimer's. One specific allele of Apo-E, E4, is seen at a 35-45% level in AD population as opposed to only 10-20% in normal population (Saunders et al., 1993; Tsuda et al., 1994). Nalbantoglu, however, found a specificity of only 68% from several studies and therefore concluded that this is only a strong risk factor and not a cause.

The Tau Protein

Allen Rosen and Warren Strittmatter (1996) had proposed a radical new theory on the causation of late onset AD accounting for 80%

of late onset AD patients. They suggest that AD is caused by the absence of a protein that helps protect the structure of microtubules (protein filaments needed for normal neuronal functioning). Their solution is not direct. Instead of seeing the problem as explained above in Apo-E4, they propose the other two APO-E variants--Apolopoprotein E2 and E3, help protect against AD by shielding protein Tau from mutation. Tau binds to microtubules, forming and stabilizing them. If Tau receives abnormal additions of phosphate groups it binds less well to microtubuli, allowing them to degenerate, consequently leading to the death of the neurons.

Genetic factors in the causation of AD play a very central role. The research in this field is still very young and far from reaching its end. Taking this and the proofs elaborated upon in this article into account, it seems unlikely that the genetic explanation will be discarded, despite the problems and criticisms. New discoveries are constantly being made, and we are gaining more and more understanding about this disease. This is vital since there is still no cure for AD and one can only be found once the etiology and pathology of the disease are fully understood.

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by Rina-Claire Weinstein

Environmental and Genetic Causes of Antisocial Personality Disorder

The Diagnostic and Statistical Manual of Mental Disorders (DSM IV) states: "The essential feature of Antisocial Personality Disorder is a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood" (1994). Early research that discusses the causality of Antisocial Personality Disorder (ASPD) focuses primarily on the genetic factors that cause, or predispose, a person to this disorder. Though the study of genetic dispositions as causal factors of ASPD is still a strong focus in experimentation, recent studies have begun to focus more upon the importance of environmental factors. Many of the genetic studies are actually in conjunction with studies on alcohol and drug abuse, whereas environmental factors are conceived in lieu of childhood conduct disorders and child abuse as predictors of ASPD.

McGuffin and Thapar (1992) discuss the history of the investigation of a genetic component to personality disorders. They explain that research information comes from three main sources: animal studies, psychophysiological studies, and personality questionnaire inventories. Animal studies have used inbred strains in order to produce identical strains of animals; this process allows researchers to determine if a particular personality trait, such as "emotionality", is a genetic trait. Psychophysiological studies have demonstrated similarities in the EEG patterns and GSR responses of monozygotic twins. Furthermore, there seems to be an abnormality in the smooth pursuit eye movement of schizophrenic patients, indicating a heritable psychophysiological pattern in abnormal behavior. Lastly, personality inventories, such as the Minnesota Multiphasic Personality Inventory (MMPI), have shown that various personality traits are genetically determined. These three types of

studies are the forerunners in studies on genetic components to personality traits.

Additionally, McGuffin et al. (1992) discuss twin and adoption studies in investigating a genetic component to antisocial behavior, one manifestation of ASPD, among adults and juveniles. As cited in their article, McGuffin and Gottesman compiled studies on adult criminality and juvenile delinquency in fraternal and identicle twins. In adult criminality, they found a 51% concordance rate among identical twins and a 22% concordance rate among fraternal twins, suggesting a genetic component. However, there was no significant difference among monozygotic and dizygotic twins regarding juvenile delinquency. Additionally, McGuffin et al. (1992) hypothesize that this genetic predisposition toward adult criminality may only manifest itself in petty crimes and not in violent crimes.

Studies in alcoholism may implicate a genetic factor in the development of ASPD. Cook and Winokur (1993) note that although studies which try to link alcoholism to a specific gene have been inconclusive, twin, adoption, and family studies have all produced evidence of a general genetic linkage to familial alcoholism. They also mention that past studies have shown a correlation between alcoholism and a family history of ASPD, in which the genetic and environmental factors in alcohol abuse and ASPD in male adoptees have been investigated. Cadoret, Troughton, and O'Gorman (1987) interviewed adoptees who had been separated from their biologic parents at birth. Alcohol use in biologic backgrounds of subject participants was defined as "alcohol-related problems;" antisocial behaviors were defined as one or more recorded antisocial behavior. Interviews were conducted with both the adoptive parents and the adoptee to determine conditions in the adoptive home environment. Additionally, background information about the adoptive families, such as socioeconomic status, was obtained from adoption agencies. The study done by Cadoret et al. provides evidence that there is a significant increase of alcohol abuse among adoptees who have a biological history of alcohol-related problems. Additionally, there is an increased risk of Antisocial Personality Disorder among adoptees with a

biologic family of antisocial behavior problems. These two observations indicate that there are both biologic and genetic elements to alcoholism and to antisocial personality disorder.

However, Cadoret et al.'s study is actually flawed. The adoption records used in this study were not of qualitative value in diagnosing antisocial behavioral problems and alcoholic abuse in the biologic families. They used criteria of "alcohol-related problems" and "antisocial behavior problems", which are not necessarily indicative of alcohol abuse or antisocial behaviors that predict future diagnoses of ASPD. Furthermore, there was no control group in this study, and results are therefore inconclusive. At best, this study can show a correlation between genetic factors, alcohol abuse, and ASPD; it cannot show causation or direction of causation.

Another study conducted by Cadoret, O'Gorman, Troughton, and Heywood (1985) provides more conclusive evidence about the genetic factor involved in Antisocial Personality Disorder. The experimenters conducted this investigation in order to determine the genetic contributions of alcohol usage and ASPD on an adult's alcohol-related problem. Two group of adopted children, each one matched with a control group, were used in this experiment. The first group was comprised of adoptees having biologic family members with two or more antisocial behaviors. However, adoption agency records were not detailed enough to define "antisocial behavior" in accordance with DSM-III-R criteria of ASPD. The second group of adoptees had a biological background of alcohol problems. Adoptive parents, adoptees, and the controls for the adoptees were then interviewed over a four year period to investigate childhood development in the adoptive environment. Additional tests were administered to the adoptees and to the controls to assess their psychological adjustment. Records of treatment for psychiatric or behavioral problems were used to identify adult adoptee alcoholism and ASPD.

Results indicate that the development of Antisocial Personality Disorder is indeed a function of genetic background. Four variables were

examined: an alcohol problem in a biologic relative, antisocial behavior in a biologic relative, adult adoptee with alcohol abuse, and adult adoptee with ASPD. In male subjects, the researchers found a significant relationship between biologic antisocial behaviors and adoptee ASPD, and biologic alcohol problems and adoptee alcohol abuse. For both men and women, they found no significant relationship between biologic antisocial behaviors and adoptee alcoholism, or vice versa. Regarding female subjects, a significant association was found only between biologic alcohol problems and adoptee alcohol abuse. These findings demonstrate that there is a connection among men between biologic antisocial behaviors and the adult offspring's development of ASPD, separate from its relationship to alcoholism.

Another study also tried to distinguish the differing variables that predispose a chilsd to ASPD as an adult (Pollock, Briere, Schneider, Knop, Mednick, & Goodwin,1990). Two hundred and one subjects participated in the experiment; one hundred thirty one subjects in the experimental group and seventy in the control group. Subjects' backgrounds were investigated for paternal alcoholism, physical abuse, antisocial behaviors, and criminal activities. Results denote that parental alcoholism alone is not sufficient as a significant predictor of future antisocial behaviors in children. However, childhood physical abuse did significantly predict future antisocial activities. According to these results, earlier studies that associate biological alcoholism with ASPD do not necessarily implicate a genetic component of ASPD as well. Rather, results suggest that environmental factors such as abuse, and not genetic factors, play a significant role in the development of ASPD. Pollock et al. (1990) state that the abuse factor alone, which is wholly environmental, is a significant predictor of antisocial behaviors, and in turn Antisocial Personality Disorder.

Recent studies by Luntz and Widom (1994) have taken the implications of the above experiment one step further. The authors investigated the development of ASPD in childhood victims of abuse and neglect, noting that earlier research had not adequately investigated the relationship between these two issues.

DSM-IV states that persons with ASPD "may repeatedly perform acts that are grounds for arrest (whether they are arrested or not), such as destroying property, harassing others, stealing, or pursuing illegal occupations." Luntz et al. cites evidence that adults previously abused as children have a higher risk of being arrested for illegal acts. Although criminality, a critical element of ASPD, has been shown to have a genetic component (Cadoret et al. 1987), Luntz et al. (1994) suggest that childhood abuse may be at the root of the disorder. This, they hypothesized, could be tested if the variable of criminal behavior is controlled, thereby testing whether the variable of abuse is accounted for.

The experiment was conducted as follows: Abused/neglected children under eleven years of age, were matched with non-abused/neglected children on variables of sex, race, date of birth, and hospital of birth. Approximately twenty years after the victimization of the experimental group, adult records of both the control and experimental groups were evaluated for criminal activities. Interviews were then conducted with both groups for IQ, reading ability and psychiatric assessments, with a particular stress on Antisocial Personality Disorder. In order to control for subject and experimenter biases, interviewers were not informed as to which group each subject belonged; similarly, subjects were uninformed about the precise nature of the experiment.

Results of the experiment were as expected. Experimenters found a significant difference between the frequency of diagnosis of ASPD in adults who had been abused/neglected as children, and the frequency of diagnosis of ASPD in those with no abuse/neglect. Although there was a visible difference between experimental and control groups for women as well as men, statistical significance is only evident among men. Interestingly, men without criminal histories received more diagnoses of ASPD when childhood abuse was a factor than those men with both criminal and abuse histories. Additionally, childhood abuse was a better predictor of ASPD among men who completed high school than among men who had not completed high school. Luntz suggests that this may be

evidence for a "saturation model", in which individuals who are at risk for ASPD have a certain amount of negative influences that can affect their development of the disorder. After that point, any additional negative forces and elements in a person's life will not increase his or her susceptibility. However, the authors maintain that childhood maltreatment was a significant precursor for several symptoms associated with ASPD.

The authors admit that although the study indicates a relationship between childhood abuse/neglect and ASPD, childhood abuse/neglect does not always reliably predict diagnosis of the disorder. 86% of the experimental population were not diagnosed as having ASPD or as being at risk for the disorder; 7% of the control group were diagnosed with the disorder. Luntz et al. suggests that this may be indicative of other variables involved with abuse/neglect and the diagnosis of ASPD. These variables may include other environmental factors, and/or perhaps an genetic predisposition to the disorder. However they conclude that based upon their research, childhood abuse is a glaring contributing factor to ASPD.

Windle, Windle, Scheidt, and Miller (1995) conducted an experiment which similarly provides an environmental model of attribution to the development of ASPD. As cited by the authors, previous studies by Kroll and Schaefer show evidence that childhood abuse is associated with higher rates of psychiatric dysfunction in adult life. With this as a basis for their research, Windle and his colleagues gathered their subject population from an inpatient treatment center through weekly announcements, sign-up sheets, and therapist suggestions. They held interviews using the Alcohol Research Center Intake Interview and asked additional questions which were designed to measure lifetime diagnoses of major depression, generalized anxiety disorder (GAD), and ASPD in recovering alcoholic inpatients. Subjects were also interviewed in accordance with DSM-III-R criteria for measures of psychoactive substance abuse, family history of alcoholism, and history of childhood abuse. Childhood abuse was classified

into four categories: sexual abuse alone, physical abuse alone, dual abuse (both sexual and physical), and neither abuse.

Although childhood abuse proved to be a significant predictor for later diagnoses of ASPD, results differed slightly between ASPD predictors for men and women. The three types of abuse were all significant predictors of ASPD in men, whereas only dual abuse and sexual abuse were significant predictors of ASPD in women. A family history of alcoholism was also a significant predictor of the disorder in men, while neither physical abuse alone nor family history of alcoholism were significant predictors for women. Although family history of alcoholism does seem to implicate genetic factors in ASPD, this finding is only significant in male subjects; gender differences are unaccounted for by the authors. Furthermore, the genetic contributions to ASPD of familial history of alcoholism to ASPD contradicts the findings of Pollock et al. (1990), who found family history of alcoholism to be a non-predictor of ASPD and found that abuse status alone was a significant predictor. Thus the presence of contributing environmental elements in the development of ASPD is agreed upon, while the effect of genetic contributions is still open to debate.

Despite the attractiveness of the experiment, there exist a few methodological limitations. Most outstanding is the non-representative nature of the subject population: all subjects were recruited from an alcoholic inpatient facility, thereby limiting the generalizability of the experiment's conclusions to non-treatment groups. Furthermore, since the patients were recovering alcoholics, this factor in itself may confound the experiment; perhaps the *patient's* alcoholism is related to ASPD, and familial alcoholism and history of childhood abuse play only a minor role in the development of the disorder. Additionally, the measurement of childhood abuse was dependent upon the subject's memory, a possibly unreliable source of information which may confound the assessment of abuse status. Likewise, areas other than physical and sexual abuse, such as emotional abuse, may also play a role in determining environmental affects on the development of Antisocial Personality Disorder.

In another study, Cadoret, Yates, Troughton, Woodworth, and Stewart (1995) investigated a genetic-environmental interaction between both biologic alcoholism and ASPD and negative home environment. They compared these elements with the development of aggressivity and conduct disorders in children and adolescents, two contributing factors to later drug and alcohol abuse and to adult ASPD. Experimental subjects were adopted children with a biologic parent who used alcohol or drugs or was diagnosed with ASPD. The control group had no such biologic record. Biologic parents' information was obtained from hospital or prison records, and was evaluated by a team of psychiatrists. The adoptee and adoptive parents were then interviewed about environmental conditions, including adoptive parent and child stressors which may have affected the adoptee's behavior from childhood through adulthood. Adoptee behavior was measured based on DSM-III-R criteria for childhood and adolescent aggression, conduct disorder, and adult ASPD.

The results of the experiment indicate that the genetic-environmental interaction has different effects upon conduct disorders in children than upon ASPD in adults. The presence of a biologic parent with ASPD will interact with adverse environmental conditions in the adoptive home environment to produce conduct disorder in the child. However, a negative home environment without the presence of ASPD in the biologic parent will not predict conduct disorder in the child. The authors do not discuss the role of genetic factors alone in determining conduct disorder in the adoptive child. Surprisingly, there is no genetic-environmental interaction in predicting adult antisocial behaviors, but rather an environmental effect alone. The authors hypothesize that other genetic or environmental conditions later in life may serve to balance out negative genetic-environmental interactions that lead to conduct disorder and aggression.

One advantage of this study over other similar studies is to be found in its clarification of the biologic and environmental factors examined. Experimenters were therefore able to separate the effects of

familial alcoholism and Antisocial Personality Disorder from one another. The results from this experiment show that conduct disorders and other behavioral problems can be attributed to an interaction between ASPD and environmental factors, but not to familial alcoholism. Additionally, the experimenters were able to use hospital and prison records to determine the presence of substance abuse and ASPD in biologic parents. Environmental conditions were also better defined based upon numerous psychiatric and substance abuse problems in the adoptive parents or siblings.

In an earlier evaluation of their experiment, the experimenters explored the two genetic pathways to drug abuse among adoptees (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995) . As cited by the authors, earlier studies have shown a strong correlation of alcohol abuse and ASPD in the adoptee. This relationship has a possible connection to three genetic pathways, one of which "starts with a biologic parent with antisocial problems leading to an adoptee with antisocial personality disorder." ASPD is then conceived as leading to alcoholism in the adoptee. The authors make this attribution regarding this direction of effect based upon the assumption that conduct disorder in early childhood is a precursor, as defined by DSM-IV, to ASPD. This study tries to understand drug abuse and dependency by examining the relationship between biologic parents' alcohol abuse and ASPD, adoptive parents' psychiatric state, and the adoptee's aggressivity.

Results of the experiment show that there is indeed a pathway to drug abuse that starts with the biological parents' psychiatric assessment and the adoptee's ensuing psychiatric evaluation. The authors find that the biologic parental diagnose of ASPD will in turn lead to adoptee aggression in childhood, and Antisocial Personality Disorder as an adult. This psychiatric dysfunction then influences the adoptee's drug abuse or dependence. This genetic effect, however, is not the sole determinant of drug abuse in adopted children. The authors also cite evidence from their research that "the disturbed adoptive parent variable is significantly correlated with adoptee antisocial personality disorder, one of the

intervening variables between biologic parent antisocial personality disorder and adoptee drug abuse/dependency." According to this observation, a biologic variable must exist in order for ASPD to occur, but a disturbing environmental condition will be the determining factor in the development of the disorder in the adoptee. Genetic factors alone are not attributable to the development of ASPD, but rather to the interaction between genetics and the environment, which will then lead to drug abuse or dependency in the adoptee.

This article has investigated causality in the development of Antisocial Personality Disorder in adults. Adoption studies have primarily been used to investigate the possibilities of genetic and environmental contributions to ASPD. Early studies researched a genetic cause of the disorder in conjunction with studies on alcoholism in adult adoptees. The primary environmental element that has been studied is the effect of childhood abuse on adoptees with ASPD and other behavioral problems. More recent studies have tried to investigate a genetic-environmental interaction factor in the development of Antisocial Personality Disorder, and analysis of the experiments does not implicate an "either-or" mode of causality. Recent studies support the evidence that a genetic-environmental interaction is the source of this disorder. However, more studies will have to be conducted in order to determine the ramifications of that interaction. Perhaps further investigation will show that one element plays a more significant role than the other.

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by Adina Sacknovitz

The Mechanisms of Phototherapy As a Treatment for Seasonal Affective Disorder

Interest in the topic of Seasonal Affective Disorder (SAD) has only prevailed throughout the past decade and a half. The most common form of SAD, referred to as winter depression, is marked by two identifying features. The first consists of recurrent moods that follow a seasonal pattern of depression during the winter months. The second is increased energy, possibly even meeting the criteria for hypomania, during the summer months (Rosenthal, Sack, Gillin, Lewy, Goodwin, Davenport, Mueller, Newsome, & Wehr, 1984). Suicide rates among SAD patients peak during the month of April (Sack & Rosenthal, 1987). This is possibly due to the fact that April is the beginning of spring time, when SAD patients are starting to improve in mood and have the energy to plan a suicide.

Apart from the general signs and symptoms of depression, other symptoms specific to SAD include overeating, weight gain, carbohydrate craving, and oversleeping, all of which occur during the winter months (Checkley, Murphy, Abbas, Marks, Winton, Palazidou, Murphy, Franey, & Arendt, 1993). This disorder predominantly affects females over males by a ratio of 4:1, and generally develops when people are in their twenties (Blehar & Rosenthal, 1989). However, the disorder has been seen in children as well and expresses itself through irritability and refusal to get up in the morning, symptoms which may often be confused with school phobia (Rosenthal & Wehr, 1987).

The acceptance of phototherapy has been widespread, and it has been reliably established as a successful and effective treatment for SAD patients (Blehar & Rosenthal, 1989; Levitt, Joffe, Moul, Lam, Teicher,

Lebegue, Murray, Oren, Schwartz, Buchanan, Glod, & Brown, 1993; Partonen & Lonnqvist, 1993; Rosenthal et al., 1984, 1987; Wirz-Justice, Graw, Krauchi, Gisin, Jochum, Arendt, Fisch, Buddeberg, & Poldinger, 1993). When exposed to bright, artificial lights for a certain amount of time daily, SAD patients show an improvement in their general mood. Possible side effects experienced by subjects after phototherapy treatment were minor and consisted of headaches, nausea, and eyestrain (Levitt et al., 1993). Phototherapy, however, does not seem to be effective for patients with a non-seasonal depression (Partonen & Lonnqvist, 1993; Rosenthal & Wehr, 1987).

Although researchers seem to be in agreement that phototherapy is effective as a treatment for SAD, much controversy remains over the exact mechanisms by which phototherapy operates. The manner in which phototherapy works is considered extremely important, since once these mechanisms are understood, one has in essence uncovered the underlying pathology of SAD, which subsequently may enable researchers to apply this knowledge in other areas and shed light on treatments for related disorders.

One of the major theories regarding mechanisms of phototherapy is referred to as the "melatonin hypothesis" (Betrus & Elmore, 1991; Murray, 1989; Rosenthal, Jacobsen, Sack, Arendt, James, Parry, & Wehr, 1988; Sack & Rosenthal, 1987). Melatonin, a hormone derived from serotonin, is generally secreted by the pineal gland, generally during the night (Betrus & Elmore, 1991). The human body produces an endogenous circadian rhythm that is slightly longer than twenty-four hours. In order to reset the biological clock on a daily basis, light becomes the critical external cue. As the light enters the retina and travels through the retinohypothalamic tract, a message is carried by the neurotransmitter acetylcholine to a part of the hypothalamus called the suprachiasmatic nucleus (SCN). Another neurotransmitter, norepinephrine, then carries the message to the pineal gland. When the "light" message reaches the pineal gland, it The causes the pineal gland to suppress its release of melatonin into the body. This process is essential in regulating the body's circadian rhythm.

The melatonin hypothesis has emerged from what researchers

already know about seasonal changes in animals. Studies have shown changes in melatonin secretion to be essential in certain seasonal changes in animals such as hibernation and migration (Sack & Rosenthal, 1987). Animals that hibernate respond to the decreased amount of daylight during the winter by increasing their secretion of melatonin. The animals then begin to overeat, gain weight, and crave carbohydrates, physiological preparations that are necessary for the process of hibernation. These same symptoms are typical of SAD patients, and do not present themselves in patients with non-seasonal depression.

The observation that an increase in melatonin secretion produces identical symptoms identical to patients suffering from SAD led researchers to believe that melatonin may be the cause of the same effects in humans. As a result of the decrease in light during the winter months, people secrete higher levels of melatonin which may lead to depressive symptoms in individuals who are sensitive to melatonin. According to this hypothesis, phototherapy is thought to operate by increasing the amount of light these individuals are exposed to, thereby further suppressing their production of melatonin (Betrus & Elmore, 1991; Murphy, 1989; Rosenthal et al., 1988; Sack & Rosenthal, 1987).

The melatonin hypothesis appears to be a logical explanation for the mechanisms of phototherapy. If light normally suppresses melatonin secretion, then it follows logically that an increase in light through phototherapy should enhance this natural process and benefit those with a sensitivity to melatonin. However, studies that have been performed do not seem to support this hypothesis.

One experiment to test the melatonin hypothesis was conducted by Sack and Rosenthal (1987). They administered melatonin to SAD patients who were successfully undergoing phototherapy. This was done under the assumption that if melatonin was indeed causing the symptoms of SAD, the administered melatonin would counteract the positive effects of the phototherapy and cause the patients to relapse into a depression accompanied by other typical symptoms of SAD. The results, however, showed that although some of the typical symptoms of SAD worsened in most patients after having taken melatonin, their general moods, as measured

by a depression inventory, remained improved as it had been under the effects of phototherapy alone. This study seems to show that the therapeutic element in phototherapy is not its suppression of melatonin, although melatonin may indeed have a role in causing some of the symptoms specific to SAD (Sack & Rosenthal, 1987).

Another experiment was performed by Rosenthal et al. (1988) to test whether blocking melatonin secretion through the use of a drug would produce the same effects as phototherapy. The drug atenolol was administered to SAD patients to block the pineal gland from secreting melatonin and results were compared with a placebo control group. The level of melatonin secretion in the body was measured by the melatonin metabolite 6-hydroxymelatonin sulfate that appeared in urine samples. Melatonin levels were indeed lower in the urine samples of the experimental group and there was a reduction in pulse rate and blood pressure as well. However, their general state of depression, as measured by the Hamilton Rating Scale for Depression (HRSD), did not improve with the administration of the atenolol and the difference between the experimental and control groups was not statistically significant (Rosenthal et al., 1988). These results are contrary to the melatonin hypothesis. This study is specifically important because it raises the possibility of using a drug as a substitute for the process of phototherapy; however, until the mechanisms of phototherapy are better understood, it is impossible to know which drug would be effective.

Another experiment that can be used as evidence against the melatonin hypothesis was conducted by Meesters, Jansen, Beersma, Bouhuys, and Van Den Hoofdakker (1995). Based on the fact that melatonin is not secreted during the middle of the day, Meesters et al. compared the effects of three different schedules of phototherapy on SAD patients. If phototherapy works by suppressing melatonin, then phototherapy that is administered during the day should not have any effect, as opposed to when treatments are given in the morning or evening. However, contrary to this assumption, the results of the study showed that afternoon phototherapy was equally effective as morning or evening treatment.

The results of these studies all seem to refute the hypothesis that phototherapy operates by suppressing melatonin. Melatonin may have a role in some of the symptoms of SAD but it does not seem to be the crucial factor in producing depression in SAD patients. A second popular theory explaining the mechanisms of phototherapy is referred to as the phase-shift hypothesis (Betrus & Elmore, 1991; Blehar & Rosenthal, 1989; Murray, 1989; Rosenthal & Wehr, 1992). This hypothesis is based on the assumption that most SAD patients have phase-delayed circadian rhythms in relation to sleep-onset, which causes them to have depression symptoms. Therefore, phototherapy administered early in the morning should advance the circadian rhythm and rid SAD patients of their symptoms, whereas phototherapy administered during the evening should delay circadian rhythms even further and possibly even lead to a worsening of symptoms. Afternoon phototherapy should have no effect at all (Lewy, Sack, Miller, & Hoban, 1987).

Lewy et al. (1987) conducted a study to test this hypothesis. Before any treatment was given, they measured melatonin levels in the blood of both normal and SAD subjects every thirty minutes during the day. The melatonin onset times of SAD subjects, used as indicators of circadian phase positions, were found to be significantly delayed relative to those of the normal subjects. After exposing subjects to morning phototherapy for one week and evening phototherapy the next week, results showed that morning phototherapy was much more effective in alleviating depression in the SAD subjects, as measured by the HRSD. Blood samples showed that melatonin secretion was advanced with morning treatment to a position similar to that of the normal controls and delayed with evening treatment. This study is strong evidence for the phase-shift hypothesis, as it shows that when the circadian phase is advanced with light, SAD patients are alleviated of their depression.

There is, however, great controversy with regard to the phase-shift hypothesis due to contradictory evidence. As discussed previously with relation to the melatonin hypothesis, phototherapy has been shown to work even in the afternoon, when light should not have any effect on the circadian rhythm (Meesters et al., 1995). Evening phototherapy has also been shown to be effective treatment for SAD patients (Doghramji, Gaddy,

Stewart, Rosenthal, & Brainard, 1990), which is in complete opposition to the suggestion of phase-shift hypothesis. There is even evidence showing that a schedule alternating between morning and evening phototherapy is equally effective in treating SAD as is a fixed schedule of either morning or evening phototherapy (Lafer, Sachs, Labbate, Thibault, & Rosenbaum, 1994). The possibility exists that circadian rhythms are indeed affected by phototherapy; however, this change is not what leads to the improvement seen by SAD patients after phototherapy. Phototherapy may operate in some other manner, and the change that occurs in circadian rhythm is merely a by-product of phototherapy, rather than the instrumental mechanism in its efficacy (Rosenthal & Wehr, 1992).

Another study contradictory to the phase-shift hypothesis, demonstrated that although the majority of SAD patients have a phase delayed circadian rhythm, not all do. The authors did so by measuring the metabolite of melatonin in urine samples every fifteen minutes for two days prior to any treatment. Despite this finding, when phototherapy was administered, subjects showed lower depression ratings whether they had been exposed to morning or evening light and regardless of their natural circadian phase position (Wirz-Justice et al., 1993).

There is a possible explanation given for the discrepancy between the original experiment conducted by Lewy et al. (1987) and the latter experiment conducted by Wirz-Justice et al. (1993): the original experiment was a within subjects design while the latter experiment was a parallel, or between subjects, design. Each subject in the original experiment was exposed to morning light for a week and then switched to evening light, and the effects of each were compared. In such a case, it is possible that the evening light was not effective; this is because its regular effect may have been somehow altered by the fact that it had been preceded by morning light the week before. This carryover effect could not have occurred in the parallel design, where each subject was randomly placed into either a morning or an evening phototherapy schedule for the entire time and the two groups were compared. Therefore, the second experiment seems to have more accurate results and thus, presents strong evidence against the phase-shift hypothesis.

A study by Checkley, Murphy, Abbas, Marks, Winton, Palazidou, Murphy, Franey, and Arendt (1993) showed similar results. When plasma melatonin was monitored in normal and SAD subjects not undergoing any treatment, there were no significant differences in melatonin rhythms between the SAD subjects and the normal subjects. In other words, there was no phase delay in melatonin onset times of SAD subjects negating the basic assumption that underlies the phase-shift hypothesis.

As these studies show, there is much debate over the phase-shift theory. If this theory was to be supported by further evidence, it might provide considerable insight into other syndromes that affect the circadian rhythm, such as night-shift work and jet lag, and raise the possibility of treating these syndromes with phototherapy (Lewy et al., 1987). However, it is difficult to understand why, if these people also have a phase delayed circadian rhythm, they do not have SAD. In addition, if the circadian phase is indeed moderated by changes in melatonin onset, then it might be possible to use the drug melatonin administered orally at specific times of the day as an alternative to phototherapy in treating SAD (Lewy, Ahmed, Jackson, & Sack, 1992).

There are many other possible theories in determining how phototherapy operates. It is possible that SAD symptoms are not directly due to an increase in melatonin or a sensitivity to it but rather, SAD may be caused by a malfunction or abnormality somewhere else along the process of resetting the biological clock through light. This leaves open a wide range of possible malfunctions that may have occurred that would ultimately require additional light to reset the biological clock and attain the desired response that occurs in normal people. According to these theories, phototherapy should be effective at any time of day that it is given. The amount of additional light is the element causing the effect, not the time that the person is exposed to the light.

Beginning with the first stage in the process of resetting the biological clock, SAD patients may have a reduced retinal sensitivity to light (Rosenthal & Wehr, 1992). This would mean that the retina does not respond to the amount of light generally required in this process and additional light would be necessary for the retina to respond and send a

sufficient "light" message to the suprachiasmatic nucleus (SCN). Another possibility is that there may be direct damage to the SCN causing it to malfunction (Rosenthal & Wehr, 1992). This is a logical theory since the SCN is located in the hypothalamus which is the center that controls and is responsible for the symptoms that appear in SAD, such as overeating, oversleeping, and carbohydrate craving.

There may also be an abnormality in the functioning of one of the neurotransmitters that is essential for the transmission of the "light" message or for some other brain function that would result in the symptoms of SAD. Serotonin is one of the major neurotransmitters that is considered in trying to explain the pathology of SAD. This may be as a result of its role as a precursor to melatonin, as well as its importance in regulating appetite, which is affected in people with SAD, and its proposed link to non-seasonal depression. Rosenthal and Wehr (1992) explain that there is some evidence showing that carbohydrates enhance serotonin activity in the brain. They propose that SAD patients may crave carbohydrates as a behavioral attempt to correct an underlying abnormality in brain serotonin function.

Rosenthal et al. (1984) discussed evidence that brain serotonin levels decrease in the fall and winter even in normal people who have no affective disorder. If this is true, then SAD may be due to an increased sensitivity in these people to the reduced levels of serotonin in the brain. This may also explain the symptoms of SAD, such as overeating, weight gain, and carbohydrate craving which are all normally influenced by serotonin. O'Rourke et al. (1987), as cited by Blehar and Rosenthal (1989), administered a serotonin agonist, D-fenfluramine, to SAD patients. This resulted in an improvement of SAD symptoms that was statistically significant in comparison to a placebo.

Rosenthal and Wehr (1992) have considered abnormal norepinephrine function as another possible cause of SAD. Norepinephrine, like serotonin, is known to influence eating habits and appetite. Norepinephrine is also the neurotransmitter that carries the "light" message from the SCN to the pineal gland. Plasma norepinephrine levels have been found to be negatively correlated with depression. Skwerer et al. (1988), as cited by Blehar and

Rosenthal (1989), found that after phototherapy, there is an increase in norepinephrine levels that is in proportion to the improvement seen in patients' moods.

Currently, there is not much research available that has dealt with the role of neurotransmitters in SAD. This may be because it is difficult to test these hypotheses, which try to demonstrate that there is some abnormality in the function of a certain neurotransmitter. Neurotransmitters have so many different tasks in the body and influence so many different areas that it is hard to isolate their effects on specific symptoms. There are also numerous theories on the role of specific neurotransmitters in non-seasonal depression and it is difficult to understand precisely how SAD is related to non-seasonal depression and whether it may in fact be a separate and distinct disorder with its own etiology.

There is still much controversy over the mechanisms of phototherapy and no conclusions have been reached in terms of how it operates. The only fact of which researchers are sure is that phototherapy is an effective treatment for people with SAD. An understanding of how phototherapy operates would explain the pathology of SAD. This could provide insights into new possibilities in treating related syndromes and psychological disorders with phototherapy and in treating SAD with drugs. However, until the mechanisms of phototherapy are understood, its uses and applications remain limited to the treatment of SAD.

In terms of research, better controlled studies are required to verify whether there is an optimal time of day that phototherapy should be administered as this seems to be a key element in uncovering the mechanisms behind phototherapy. Clarification in this area would also be clinically useful since it would be much more convenient if SAD patients were able to benefit from phototherapy at any time of the day or evening.

Researchers must also continue to search for a drug that will be as equally effective as phototherapy in the treatment of SAD since phototherapy is time consuming and inconvenient. The idea of taking a pill rather than sitting under artificial light for a couple of hours a day, would be an attractive alternative for SAD patients. Even so, it is beneficial to

have an alternative to drug therapy such as phototherapy, as there may be fewer side effects and fewer problems than those that occur with drugs, such as addiction. Although it is frustrating that there is so little known about SAD and even less consensus of researchers on the issue of the theory behind phototherapy, it is important to keep in mind that people who suffer from SAD do at least have an effective treatment currently available to them in phototherapy.

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by Shaindi Steiner

Immunology and Stress: The Effects of Psychological Stress on Biological Functioning

The immune response, arguably the body's most elegant protective system, mobilizes a formidable line of defense. The B and T lymphocytes alone "can recognize ten million different antigenic structures" (Stein, 1986 p12.) This line of defense, however, is not infallible. Different psychological states and stressors (loneliness, stress, or depression, for example) have been shown to affect its functioning and competence. This has particular relevance in light of the increased morbidity and mortality following bereavement (Kiecolt-Glaser et al., 1984b).

The immune system's attack is either nonspecific or specific. The non- specific response is the body's immediate reaction to any antigen (a foreign body or a non-self substance). It makes no difference who or what the antigen is--the body automatically attacks. One type of non-specific cell that attacks microbes is the natural killer cell. Rather then attacking the microbe directly, it destroys the body's infected cells. By puncturing the infected cell wall, the NK cells cause the infected cell to fill up with water and explode. Their specialty is detecting and causing explosion of cancer cells before the cells grow into tumors. Many immunocompetence experiments measure the NK levels, since "the vigilant surveillance by natural killer cells is one of the body's most potent defenses against cancer" (Raven & Johnson, 1992 p1084).

Inflammation is another of the body's non-specific responses to infection. Cortisol, a hormone produced by the adrenal cortex, reduces

inflammation. Elevated levels, however, when no inflammation is present can indicate suppression of immune function. Cortisol levels, interestingly enough, are higher in patients diagnosed with major depression than in the general population (Stein, 1986).

The body's specific defense system is organized along two pathways; the cell-mediated immune response and the humoral or antibody immune response. The T and B cells are a type of lymphocyte or white blood cell. The helper T cells are the "commanders" of the cell-mediated immune response (Raven & Johnson, 1992). They are responsible for activating the immune response. The cytotoxic T cells rupture the infected cells, and the suppressor T cells call off the immune response.

The B cells are the main players in the humoral immune response which is a "longer-range defense" (Raven & Johnson, 1992 p1098). The B cells produce antibodies which attach themselves to the antigens like a flag which mark the antigens for destruction. These antibodies are also called immunoglobulins, and they include IgM, IgG, IgA, IgD, and IgE.

The six studies discussed below attempt to determine the effects of various stressors on subjects' immune response. Difficulties arise in defining the nebulous term, "stress;" the studies do not address this problem, but deal with more concrete stressors, such as final exams in medical school, loneliness, separation/divorce, caring for an ailing relative, and anticipation of bereavement.

In the study, "Psychosocial modifiers of immunocompetence in medical students," Kiecolt-Glaser et al. tested the effects of a very potent and "naturally occurring stressor"- final exams - on seventy five first year medical students (1984a p8). Blood was drawn a month before finals and on the first day of finals, after students had written their first two exams. The first blood sample served as the baseline.

The experimenters examined NK cell and IgG, IgA, IgM, CRP, and salivary IgA levels. Three self-report questionnaires were administered when the blood was drawn: the Brief Symptom Inventory (BSI); the Social Readjustment Rating Scale (SRRS); and the UCLA Loneliness Scale.

The authors analyzed the effects of three main variables--stress (low, high), loneliness (low, high), and trials (first, second). Since gender was only significant for plasma IgA, gender was collapsed across the other data. There was a significant decrease in NK activity from the baseline to their first day of exams. Those students that scored high on stressful life events or on the loneliness scale also showed significantly lower NK activity.

The blood samples for the plasma and salivary immunoglobulins and CRP were only analyzed for one main effect, the trials, since complete data was unavailable due to "technical difficulties" (Kiecolt-Glaser et al., 1984a p10). IgG, IgA, and IgM levels all increased, but only plasma IgA reached significance. Women had lower levels of the immunoglobulins overall, and their levels of IgA exhibited a smaller increase than the men's. Analysis of salivary IgA did not reach significance. CRP levels were not analyzed due to the "very small number of deviant samples" (Kiecolt-Glaser et al., 1984a p11).

Glaser and her colleagues explored other factors that might have affected the data. Previous medical problems were investigated. Eleven students reported allergies, but they were randomly distributed across the groups. The experimenters also examined the possibility that the self-selection of the subjects might have influenced the data. Perhaps students earning poorer grades volunteered for the study, since the reward for participating consisted of a report of the subject's immunological data. However, after a comparison of grades, a trend (non-significant) in the opposite direction was observed, with students who earned better grades participating. (The grades were also analyzed for effects of loneliness and stressful life events. While no significant effects were found for loneliness, students reporting high stressful life events scored lower on average on their exams.)

The authors' conclusion that, "the immune system is sensitive to milder stressors than those sampled previously, e.g., bereavement and forty eight hours of noise and sleep deprivation" (Kiecolt-Glaser et al., 1984a p12), is problematic. The authors themselves note that they did not obtain any data on "changes in the use of drugs or alcohol, amount of sleep, or

nutritional status in relation to the baseline and final examination weeks" (Kiecolt-Glaser et al., 1984a p13). The students were undoubtedly sleep deprived and probably neglectful in obtaining proper nourishment.

The relationship found in this study between loneliness and lower NK activity, however, is especially compelling in light of the fact that depression and anxiety are associated with increased levels of loneliness.

The study, "Urinary cortisol levels, cellular immunocompetency, and loneliness in psychiatric inpatients," was done by the same authors who performed the "Psychosocial Modifiers" study (Kiecolt-Glaser et al., 1984b) in order to reproduce and expand upon their previous findings. Psychiatric patients were used as subjects, since this populations tends to report more loneliness and fewer social support networks.

Urinary cortisol levels (correlated with isolation), NK activity, and reactivity of lymphocytes to certain antigens (phytohemagglutinin and pokeweed mitogens) were measured. Self-report measures administered included the UCLA Loneliness Scale, the MMPI, and the Psychiatric Epidemiology Research Interview Life Events Scale (PERI).

The two independent variables were loneliness and stressful life events. Gender was initially included as a variable, but because no main effects were found, the data were collapsed across the other two variables. The high loneliness patients exhibited significantly higher levels of urinary cortisol, lower levels of NK activity, and a worse response to PHA stimulation. There were no significant main effects for stressful life events on cortisol or NK levels. (PWM stimulation was only significantly correlated with stressful life events at a 5.0 ug/ml concentration.) Once again, "loneliness emerged as the best predictor of immunocompetence" (Kiecolt-Glaser et al., 1984b p20).

A weakness of this study is the authors' failure to collect nutritional data from the subjects. It is possible that the conclusions reached were skewed because of this omission. The depression in immune response could have been caused by a poor diet or lack of sleep, or because the blood obtained from these patients was drawn at different times of day.

Another study, "Marital quality, marital disruption, and immune function," analyzed the correlations between marital quality and separation and immunocompetence " (Kiecolt-Glaser et al., 1987b). Thirty-eight separated/divorced women were matched with 38 married women. The subjects were recruited from various sources [advertisements, the church, the undergraduate population, and the group Parents Without Partners (PWP)]. Only subjects that were not currently taking any immunosuppressive medication, or suffering from any health problems that had an "immunologic or endocrinologic component" were accepted (Kiecolt-Glaser et al., 1987b p15). Separated or divorced women were restricted to those who had separated within the last 6 years.

Questionnaires administered included the Brief Symptom Inventory (BSI), the Dyadic Adjustment Scale (DAS), the Attachment Scale, the survey version of the UCLA Loneliness Scale, and the Psychiatric Epidemiological Research Inventory Life Events Scale (PERI). The immunologic assays evaluated the subjects responses to concanavalin A, phytohemagglutinin, and antibody titers to the Epstein-Barr virus, and additionally determined the levels of NK cells, helper and suppressor T cells. Nutritional assays, assessing the subjects' levels of albumin and transferrin, were used to determine their overall nutritional condition.

Lower DAS scores among the married women, indicating poorer marital quality, were associated with higher EBV VCA antibody titers. (High levels of antibody titers to EBV indicate incompetence on the part of the cellular immune response. Presumably, the humoral immune response is overloaded with viral antigens, and this accounts for the increase in antibodies.) There was also a significant difference between the high and low marital quality scorers and the response to PHA stimulation.

In comparisons between the recently separated/divorced women and the married women, the separated/divorced women exhibited higher EBV VCA titers, lower levels of NK cells, and lower levels of helper T lymphocytes. (The suppressor T lymphocytes showed no significant difference.) There was also a significant difference in the PHA response between the two groups. While a significant difference in transferrin levels was observed between the two groups, both counts were within the normal

range.

The experimenters suggest one way in which marital quality could affect one's health. Just as greater autonomic arousal has been associated with marital dissatisfaction, "...it is quite possible that there are concurrent persistent alterations in endocrine function that mediate immunologic changes" (Kiecolt-Glaser et al., 1987b p29).

Once again, the fact that blood was not drawn at the *same time* is a possible confounding variable. In addition, the experimenters report that the difference in transferrin levels was significant, although both were within the normal range. If the difference is significant, a more sensitive indicator of nutritional fitness should be implemented. This is especially important if one wishes to completely rule out the role of poor nutrition on immunocompetence.

Another study on marital quality, this time conducted on males, was carried out by the same authors, entitled "Marital Discord and Immunity in Males" (Kiecolt-Glaser et al., 1988 p30). The article suggests that marital dissatisfaction may be a greater stressor than previously thought, since in addition to the partner in the marriage being a source of stress, the spouse feels constrained from relying on, and exploring outside social networks. In addition, men may suffer more from separation, as the woman is frequently the one to initiate the separation. The initiator subordinates and makes powerless his or her hapless mate.

The subjects, 32 separated/divorced men matched with 32 married men, were recruited from the college population, and from the local population by ads and a newspaper article about the experiment. The same screening procedures were used as in the previous study. Similar self-report measures were administered, as well as Rotter's locus of control scale, in the event that the role of initiator might be associated with its own constellation of psychological and health correlates. Similar nutritional (albumin) and immunological assays (helper and suppressor T cells and EBV and HSV-1 antibody titers) were analyzed.

Married men who reported greater satisfaction with their marital life

exhibited greater percentages of suppressor T cells and a greater helper to suppressor ratio. Marital quality was also significantly related to EBV and HSV-1 antibody titers. Poorer marital quality was significantly correlated with greater depression, distress, and loneliness. Separated/divorced men who initiated the divorce, or reported that the separation was mutual, had significantly lower EBV antibody titers than the passive separated/divorced men.

The difference in antibody titers between the two groups was significant, with the married subjects displaying lower levels of titers for both EBV and HSV-1. The differences in helper and suppressor T cells and the albumin levels failed to reach significance. Similarly, other weight and sleep measurements did not differ significantly between the two groups. On a more subjective measure, separated/divorced men recalled more recent illnesses.

In an age where therapy is becoming increasingly common, it is surprising that the experimenters failed to ask if any of their subjects were undergoing therapy. Even self-help groups have a possible beneficial value, and could be a potentially confounding variable. In addition, it is also possible that the separated and divorced members exhibited "...preexisting depressive symptoms [which] were an underlying cause for the divorce; moreover, such symptoms might have subsequently hampered the formation of alternative relationships, a factor associated with lower attachment" (Kiecolt-Glaser, et al., 1988 p30). The direction of these correlations is very hard to prove.

Surprisingly, studies of rodents have found that exposure to chronic physical stressors actually leads to an improvement in immunocompetence. Glaser, in the study, "Chronic stress and immunity in family caregivers of Alzheimer's disease victims," wished to extend these findings to humans. Caregivers of patients with a chronic disease, "provides an opportunity to examine the immunologic psychologic consequences of a more chronic psychosocial stressor" (Kiecolt-Glaser, et al., 1987a p524).

The caregivers were only screened for immunosuppressive medication and illnesses that had an "immunologic component" (Kiecolt-

Glaser, et al., 1987a p525). Caregiver subjects had an equal number of controls who were matched for similar medications, age, sex, and education.

Self-report measures administered included the Beck Depression Inventory and the Older Americans' Resources and Services Multidimensional Functional Assessment Questionnaire (OARS). Demographic and health data were collected, and the patients' histories and present functioning were also assessed. The levels of T lymphocytes, including helper T lymphocytes and suppressor T lymphocytes, NK cells, and Epstein-Barr virus (EBV) antibodies were examined.

The caregiving variable was the only variable analyzed since gender was found to have no effect. Caregivers had significantly higher scores on the shortened version of the BDI, conveyed lower satisfaction with life, and reported lower ratings of mental health. There were, however, no significant effects of loneliness and social support. The caregivers had significantly higher levels of antibody titers to EBV VCA, and lower percentages of total T lymphocytes and helper T lymphocytes. There were no significant differences in the suppressor T cells, in the helper-to-suppressor ratio, or in the NK cells.

The caregivers' nutritional condition was analyzed by assessing their plasma and transferrin levels. The experimenters wished to discount the possibility that their compromised immunocompetency was due to poor nutrition. The results supported their hypothesis since all caregivers were "within normal range for both markers" (Kiecolt-Glaser, et al., 1987a p530). While there was a significant difference in the amount of sleep the two groups reported, the correlations of amounts of sleep with "immunologic parameters" failed to reach significance.

The level of NK activity was correlated with the Alzheimer patient's place of living. The caregivers whose AD relative was in an institution had the highest level of NK cell activity compared to those caregivers whose AD relatives lived with them or elsewhere. The caregivers who belonged to a support group were analyzed separately, and were found to report significantly less loneliness and sustain higher levels of NK activity.

Unlike rodents exposed to a chronic physical stressor, humans did not seem to adapt as well or exhibit the improvement of immune function that the rodents did. The difference may be due to the magnitude and nature of the stressor. Caring for an AD patient (especially a family member) is a psychosocial stressor as opposed to a physical stressor. It is an extremely intense experience, both emotionally and physically draining. Hope is a powerful and significant contributor "to the maintenance of healthy physiologic states under the stress of significant loss" (Udelman & Udelman, 1983 p217). Yet the caregiver sustains no hope that the AD patient will recover.

One would assume that bereavement, or anticipation thereof, would be a potent combination of stress and loneliness. (See "Life events, depressive symptoms, and immune function," (Irwin et al. 1987) and "Alterations in immunocompetence during stress, bereavement, and depression: Focus on neuroendocrine regulation" (Calabrese, et al. 1987)). Indeed, these studies have shown that women anticipating or undergoing bereavement, exhibit a compromised immune system.

However, a more recent study, "Lymphocyte response in depressed patients and subjects anticipating bereavement," contradicted the above findings. The experimenters wished to rule out sleep and weight changes as the main effecters in immunocompetence, while concurrently evaluating the lymphocyte response in depressed subjects and those anticipating bereavement.

Eleven subjects who had been diagnosed with major depression and eight subjects whose spouses were being treated for lung cancer participated in this study, together with nine healthy controls. The Hamilton Rating Scale for Depression and the BDI were administered. Lymphocyte stimulation with PHA was designated as the gauge for immunocompetence. As predicted, sleep and weight changes were not associated with lymphocyte activity for either group. However, while there was a significant negative correlation between lymphocyte activity and BDI scores in the depressed subjects, there was a positive correlation between lymphocyte activity and BDI scores in the subjects anticipating bereavement. It appears that anticipating bereavement can enhance ones

love was reciprocated" (Smith & Hokland, 1988 p44). Junior college students in a reciprocated love relationship exhibited lower NK activity levels. Apparently, all the world loves a lover, including the immune system.

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immune response! The authors plot a "significant curvilinear relationship... with decreased immune response found for mild and severe depressed states" (Spurrell & Creed, 1993 p62).

There are several factors that might have influenced these results. While the experimenters did collect information about "changes in diet," clinical nutritional assays would have constituted a more sensitive measure. In addition, studies have shown the herpes virus antibody titers to be the more discriminating measure of compromised immunocompetence as opposed to lymphocyte response to PHA (Kiecolt-Glaser, et al., 1988).

The authors mention two other potentially confounding variables. Firstly, half of the patients were on medication (benzodiazepines). Secondly, the group of subjects anticipating bereavement were selected by doctors who might have picked "copers," as opposed to "non-copers". In this case, it is not so surprising that the "coping" subjects anticipating bereavement displayed a better immune response.

It is unclear exactly how or why stress affects the immune system. Even when it does depress the immune system, it is also unclear that the person is more sickly than the average person. Subjects with compromised immune systems do not report significantly more visits to their doctor. One hypothesis is that prolonged stress causes the body to continually release cortisol which, although helpful in small amounts for the "fight or flight" reaction, is damaging in large amounts. Cortisol raises the level of blood sugar and increases metabolism. It also transfers the body's energy away from synthesizing new proteins that are integral for the immune system (Kalat, 1995)

Comprehensive studies on factors that enhance the immune response have yet to be performed. So far though, laughter does indeed seem to be the "best medicine." Studies done by Dr. Lee S. Berk have shown that, "an hour spent laughing lowers levels of stress hormones like cortisol and epinephrine. At the same time, the immune system appears to grow stronger, the body's T cells, natural killer cells and antibodies all showing signs of heightened activity"(*New York Times,* 1996 sec.C p5). Another study reveals that "being in love was salutogenic, provided that the

love was reciprocated" (Smith & Hokland, 1988 p44). Junior college students in a reciprocated love relationship exhibited lower NK activity levels. Apparently, all the world loves a lover, including the immune system.

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Clinical Psychology and Managed Health Care, "Quo Vadis"?

by Marcel Isaac Perlman, PhD

A Shrink's Lament

Doctor, Doctor tell me...

Why am I so?
G-d made you so!

Why am I so?
Mother's milk made you so!

Why am I so?
Chromosomes made you so!

Why am I so?
Chemicals made you so!

Why am I so?
Poverty made you so!

Why am I so?
Drugs made you so!

Why am I so?
Pollution made you so!

Why am I so?
Abuse made you so!

Why am I so?
How the H--l do I know ???[1]

When I first wrote the lines above, I didn't realize that there was another line I could have added ... **Managed Care made you so!** Managed Health Care is the "new boy on the block." And we don't quite know what to make of him. Traditionally, health care in this country was paid for by either the patient or family, or, alternatively, by an employer as part of an employee's compensation package. The employer would purchase this coverage from an insurer in what was known as a Group Plan. Obviously, the larger the group was, the lower the cost of the premiums was. If individuals wished, they could, (and many did), purchase individual or family coverage, but at a much higher cost. This was usually the case when employer-provided coverage was not available. This was the model followed since before World War Two, and in ever-increasing numbers afterwards. It seemed to be a relatively fault-free approach for many years. If you needed health care, you went to the appropriate provider, (Doctor, Dentist, Psychologist or Chiropractor), received the required care, and were either reimbursed for the cost, or the provider billed the carrier directly and was paid in due time.

We went along blissfully for several decades until, suddenly, with what appeared to be little warning, we (providers and patients both) found ourselves in the era of Managed Health Care. How did this happen? I am afraid we must stipulate that there is quite enough blame to go around. It seems that two words would cover the situation adequately....**Greed and Apathy**. Whose greed? Everyone's. Who's apathy? Equally, everyone's. Medical and clinical costs soared and no one seemed to care because in the main, someone else was paying the bill. There appeared no valid reason to strive for greater efficiency and economy because no one seemed to give

[1] Shrink's Lament ... Whispers at Dusk
Ed. Diana Zeiger, National Liberty of Poetry, 1997
Publisher, Jeffry Franz, Watermark Press
Owings Mills, MD 21117

a hoot. Costs went up, premiums went up, and Status Quo prevailed.

Then the concerns began. Corporations started to examine the cost of "fringe benefits" provided to their employees and began to feel their costs. Insurance companies began to look at their "loss" experience ratios and hiked the premiums even more. A perfect and fertile ground for a new breed of entrepeneure. Someone who could step in and control the "Demon" of costs. How would such a person make money for himself? Very simple. He would get paid either a fee, or in the majority of cases, would get a percentage of the costs he had saved. And like mushrooms in a dank forest, Managed Health Care companies in various formats sprung up all over the land. The formats basically follow two forms. The first is groups of providers under contract to the MHC. They agree to charge a lower fee in return for assured referrals. They further agree to abide by a host of restrictions on the nature and length of care they will provide and the recommendations for further care they will make. Violations of these caveats will lead to expulsion from the plan and from access to the patients in the plan, since patients will be reluctant to go out of the network, as doing so will mean that they have to bear the cost themselves. The second and equally frequent format is that of the HMO. These are Health Maintenance Organizations which generally consist of a group of providers from various specialties housed under one roof. For a fixed annual cost, a patient, in theory, has access to any of the practitioners in the HMO. In reality, this access is governed by what is called a Primary Care Physician who is charged with the authority to approve or deny access to other practitioners. The responsibility for cost containment rests upon his or her shoulders. The compensation to the staff is largely determined by how effectively the expenses are curbed.

How do these approaches contain costs? Very simply...By denying service to their subscribers. There appears to be little doubt that the most effective way to save money is by not allowing it to be spent. There appears to be equally little doubt that these decisions are being made almost exclusively on economic grounds rather than clinical ones.

How then does all of this bear upon Clinical Psychology? Very directly, and in some cases, in a highly Draconian fashion. The emphasis

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today has shifted from a psychodynamic or analytic approach to a short-term cognitive or directive orientation. At the risk of over-simplification, the working assumption is that one can deal with a problem of many years' standing in six sessions or less. If not, the alternative or perhaps preferred course of treatment is an organically based one of medication or electro-convulsive therapy, or, in some extreme cases, psycho-surgery. To many people's dismay, lobotomies have reared their unlovely heads once again.

To illustrate the problem anecdotally, let me cite the instance of a bright, attractive, thirty-one year old woman who sought therapy to deal with a problem she was experiencing. She had just discovered that she was pregnant. Upon receiving confirmation, she immediately went into a state of high anxiety. She consulted her internist, who, recognizing her pregnant state, realized that medication was contra-indicated. He then made the referral for psycho-therapy. She was in a Managed Health Care plan, so we immediately applied for approval. This meant calling and making an appointment to speak to a case review person no sooner than one week later. In presenting the case to the reviewer, it was not possible to establish the etiology of the anxiety after only one session with the patient. The reviewer, (a young, unmarried woman with a B.S. in nursing), commented to me that in her experience, all first time mothers panic somewhat upon learning that they are pregnant. In her view, therapy was unnecessary, and in fact, inappropriate since it would only emphasize these normal and ordinary feelings. Therefore, she could not see her way clear to approve any further sessions other than the *one* the patient had already had. She suggested that there were mild tranquilizers which might present *only* minimal risk. I then relayed this information to the patient. Quite fortunately she was willing to bear the cost of treatment herself, and after negotiating a fee that she could handle, we began. What emerged was that the basis of her panic and fear was her own childhood with a mother whose sadism and abusiveness would qualify her for the "Witch of the Decade" award. What the patient was fearful of was that she would parent much in the same way she had been reared. She feared this so greatly, that while she desperately wanted to be a mother, she was seriously considering termination of her pregnancy or adoption, rather than exposing her unborn child to the risks that having her as a mother might hold.

The above case study illustrates several issues inherent in the current iteration of Managed Health Care. Firstly, the relatively limited clinical experience of the reviewer, as well as the inappropriateness of her field of training to the request at hand. Secondly and no less importantly, the immediate reference to a drug alternative. Obviously, the reviewer had been trained by the MHC to respond in this manner. I am not suggesting that approval should have been issued for an unlimited number of sessions, but rather that a sufficient number of sessions should have been allowed to determine what the basis of the problem was, and if therapy was in fact the most efficacious way to proceed. For the sake of closure, the patient above delivered a healthy baby boy who is currently about six months of age, and she is having, in her words, the time of her life with him.

There is a broader impact upon Clinical Psychology beyond a case by case one. The current approach is having a massive effect upon the kind and nature of Graduate Training as well as the kind and direction of research that is being conducted. Training is being impacted because it is incumbent upon Ph.D. and Psy.D. programs to prepare their students for a discipline that actually exists, and one in which their students can ultimately make a living. Given the limitations imposed upon the practice of classical psychotherapy, graduate schools are being pressured to come up with programs that conform to the times, regardless of whether or not what is being discarded continues to have real merit or not. To illustrate, there is an increased call to teach and allow Psychologists to prescribe medications so that they can compete in the marketplace. In my view, this would relegate Psychologists to the role of semi-Physician, rather than that of full-Psychologist. If we truly feel that Psychology has nothing unique to contribute to the Human Condition, then we should cease and desist until such a time as we re-invent ourselves. I do not believe for a moment that this is the case! I do not believe we need to be theoretical Luddites, but at the same time we should not rush pell-mell to adapt strategies which could only be justified because they have economic value. Research today appears to be enmeshed in one enormous "self-fulfilling prophecy." Because of the clinical climate, the emphasis is on organic and biological approaches to human behavior. This in turn, ensures that the funding available for new research will, in the main, be limited to work in the "hot" areas. This leads to the increasing corroboration that this is the way to go,

when in fact there is a built-in bias in this approach. Obviously, if the field only concentrates on developing expertise in only one area, then that will be the area that appears to "work." Managed Health Care clearly has a major role to play in determining the direction in which research goes. It influences the flow of funds both directly and indirectly.

In my view, Managed Health Care has a real and valid opportunity to impact upon Clinical Mental Health. Not from the point of view of curbing costs by denying care, but rather by involving itself in an ongoing collaboration with patients, providers, and the institutions which train those providers, to raise the level of the quality of that care that they seek to manage. When we shift the emphasis from *less to better*, there is no question in my mind that we will also have shifted to *less costly*. One follows from the other. It is vital that Managed Health Care in all of its forms must move from an adversarial relationship with clinical providers and clients to a cooperative one. Doing that will provide both with a synergy from which all can benefit.