International Journal of Mental Health, Vol. 31, No. 2, Summer 2002, pp. 51–60.
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ISSN 0020–7411/2002 $9.50 + 0.00.

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A Biopsychosocial Model of Schizophrenia

The view that schizophrenia is by nature a somatic, not a heterogenic, disorder simplifies estimation of its evolution, according to M.M. Kabanov [26]. As a result, some investigators consider that its psychopathological symptoms arise from the disorder itself, from a theoretically specific process of a brain lesion.

Nevertheless, psychiatry has another, less reductively physiological, standpoint based on the notion that features of adaptive and compensatory mechanisms are of essential importance in the origin and clinical manifestations of schizophrenia. Most researchers consider *adaptation* a dynamic process of adjusting to changeable life conditions [54], the whole of which appears to be an optimally functioning organism (like a complicated system) in interaction with the environment [15], and *compensation*, a reaction of the organism to obstacles to adaptation that maintains the integrity and essential functions of the organism and compensates for the insufficiency of damaged parts thanks to the activity of the intact part of the system [4, 55]. T.B. Dmitrieva [20] thinks that the adaptive and compensatory mechanisms maintain an optimal dynamic balance (i.e., homeostasis) between organism and environment through antagonistic regulation of functions. From this viewpoint, adaptation

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English translation © 2002 M.E. Sharpe, Inc., from the Russian text © 2001 V.M. Bekhterev Psychoneurological Research Institute. "Biopsikhosotsial′naia model′ shizofrenii," *Sbornik nauchnykh trudov Sankt-Peterburgskogo nauchno-issledovatel′skogo psikhonevrologicheskogo instituta im. V.M. Bekhtereva*, 2001, *137*, 230–41. Translated by the author.

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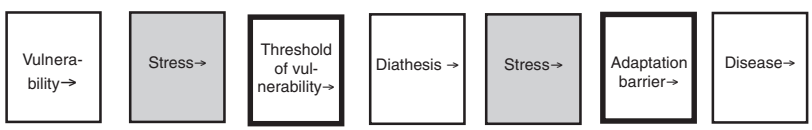
and compensation may be regarded as composite elements of a common process of adaptation for survival and development of the individual. However, it is necessary to emphasize that any defense process easily becomes pathological in the presence of aggravation; therefore, the concept of "accommodation" and its categories "adaptation" and "compensation" are applicable to both normal and pathological phenomena [19].

Within the concept of a network of adaptation and compensation, an interpretation of schizophrenia as a diathesis-stress disease is being developed. Generally, the main point of diathesis-stress theories is that they acknowledge the presence of a dynamic totality: first, of biological vulnerability as a genetic predisposition to schizophrenia [63–66] and, second, of an understanding of clinically determined psychopathological [50, 51] and schizotypal [18] "diatheses" as a special "organism's" [59] sensitivity to stimuli representing stressors under whose influence illness develops. Among these stressors researchers consider, first, "any supernormal external irritants" [33] or macroevents (i.e., critical life events); second, microevents of day-to-day living [1], ordinary psychosocial pressures [57, 58, 61] that do not affect healthy people, but act as nonspecific stressors for "vulnerable" persons [62, 65]. These stressors cause overexertion of adaptation structures and thus disturb relations between the person and the environment [11]. The forming of stressful complexes is a permanent attribute of organisms struggling "with the negative influence of the environment long before the patient becomes aware of that struggle" [46]; and in the case of adaptive-compensatory systems, the whole [9, 10, 12] may "erupt," producing the beginning of a schizophrenic process [27].

Differentiation of the terms *vulnerability* (i.e., a genetic risk factor that may not manifest itself), *schizophrenic diathesis* (i.e., a risk symptom of premorbidity), and *disease* produces a supposition about the presence of a "threshold of vulnerability" between vulnerability and diathesis [64] and about the "adaptation barrier" between diathesis and disease [2, 9] that is the dynamic formation preventing disorganization of the functional systems the organism is using for adaptation to a specific situation.

A simple diagram of this model is:



A theoretical concept of the mechanisms of development and relapse of schizophrenia may be presented as follows.

Each person has biological health resources that prevent the influence of pathogenic agents as a fixed (up to zero) degree of diathesis to schizophrenia, i.e., vulnerability is the main risk factor in the genesis of schizophrenia.

At the same time, each person also has certain psychological health resources. The concept of psychological health is linked with the idea of psychological adaptation, based on a three-component structure of individual relations and associated spheres of mental activity: (*1*) the perceptual-cognitive; (*2*) the emotional, as a relatively autonomous psychological system, including a controlled cognitive process of environmental assessment and influencing the manner of individual behavior [53]; and (*3*) the motivational [24, 37].

It is important to take psychological and biological adaptations into account during examination of etiopathogenic aspects of schizophrenia. It is necessary to point out that the nature of the relation between biological adaptation mechanisms (concerned with corresponding brain structures) and psychological adaptation mechanisms remains as unclear as discussions of the problem of the correlation between brain and mind.

Suppose, first of all, that each event is assessed from without by means of a cognitive motivational instrument. The total assessment reveals a certain level of conflict experience peculiar to each person, i.e., "antinomic reactions" [23] that are mental processes "of constant analysis and intense orientation" (difficult situations, subjective experiences), concerned with reciprocally expostulative purposes, tendencies, wishes, and attractions of the person. Consequently, any mental instrument always contains a certain set of conflict experiences (total intensity of conflict experiences in a sense), which, as a rule, is background, but intensifies during acceptance of motives or inability to realize a motive [49]. "It means that we have a relationship between the semantic and the cognitive, the semantic and the emotional, and the semantic and the motivational spheres; we have mechanisms of goal-formation and future planning" [48].

A person's psychological defense mechanisms serve as psychological coping means that aim to keep the level of conflicts within the limits of the background. However, in case of influence of external additional risk factors (the environment, micro- and macropopulation), a person's

psychological defense mechanisms cannot cope with a problem, and a psychological stress condition results. If the influence of the stress factor is halted and/or reserve defense mechanisms are marshaled, the psychological stress condition may be reversible. Otherwise, "return to the initial position" does not occur, which contributes to a distorted response or to different social effects. As a result, the reciprocal reaction is part of a "vicious circle" mechanism that intensifies the person's psychosocial vulnerability [6] to the pathogenic influence of many life events. This leads to activation of the genetic mechanism of vulnerability.

In cases in which psychological stress takes place under conditions of an insufficiency or exhaustion of psychological defense mechanisms, damage to the vulnerability threshold [64] may occur and may initiate schizophrenic diathesis and, in the future, damage to the adaptation barrier level [2] (called by L. Ciompi [56] "destruction of a vulnerable person"); and symptoms of a disorder may occur. Different genetic defects, various external damages, or excessive stress loads may lead to a common result: a failure of regulation and a move to another pathological level.

We theoretically hypothesize that a "primary breakdown" provokes the subsequent brain response in itself in the form of its activation (a first-order compensatory reaction by Iu.L. Nuller's interpretation [40]). It is no accident that Nuller thinks that "in the presence of neuropsychic diseases, initially the pathological process is inseparably linked with inclusion of compensatory mechanisms" [47]. On the clinical level, as it intensifies, this corresponds to a manifestation of psychosis in the form of anxiety and disintegration of mental functions [40]. Under conditions of "distorted balance" tending toward destructive processes, the insufficiency of defense mechanisms stimulates second-order compensatory reactions that contribute to a decrease in anxiety and stress levels, such insufficiency being "standard" and essentially displaying little differentiation in terms of an etiologic factor [40]. These *pathogenic compensatory reactions* become productive pathological disorders on the clinical level [19]. Iu.L. Nuller's [40] viewpoint consists in reducing nonspecific excitation realized by the formation of such psychopathological phenomena as depression, autism, and depersonalization.

It is a well-known fact that the appearance of experiences with a substantial plot arising from painful, maladaptative, unstructured emotions, such as anxiety, fear, panic, despair, etc., contributes to a decline in the internal tension level.

However, compensatory formations result from a regressive response since "the subject returns to the ontogenetic earliest stages of behavior under conditions of disturbance of his adaptation system" [44]. Evidence of compensation realized because of the attraction of ancient mental organizational mechanisms is the typological similarities between some schizophrenic productive disorders and the phylogenetic earliest stages of mental development [45]. Several authors, expressing fundamental permissibility with regard to psychopathological symptoms as exaggerated adjustment [29], agree with this assertion.

The features of pathogenic compensatory reactions depend on the organism's reactivity. In the presence of acute reactivity, acute and subacute productive disorders may be, as is known, prognostically more favorable versions of disease development. In this case, when compensatory formations become hyperactive, the most acute psychotic disorders (including hypertoxic schizophrenia) may prevail. Thus, in the presence of febrile schizophrenia, autoimmune reactions are hyperergic [13, 41]; in the presence of borderline disorders, autoimmune reactions are hypoergic [35]. We may draw a parallel with hyperthermia in the presence of somatic illness, which can be beneficial in some cases but, when the limit is exceeded, can create life-threatening conditions. When hyperthermia has declined to an acceptable level, a favorable outcome of the disease may be observed. In the presence of hyporeactive compensatory formations, acute psychopathological disturbances have a tendency to become chronic. An absence of productive symptoms as clinically confirmed in the simple form of schizophrenia may occur under conditions of areactive compensatory formations.

It is probable that when the organism's resistance to disease is limited by adaptive mechanisms, a situation of areactive compensatory formations may be created. A pathogenic process devoid of the opposition of compensatory formations leads to exhaustion of defense mechanisms and a decline in the organism's total functional possibilities that takes the form of negative symptoms. Increased negative symptoms form a new adaptation level that is depressed in comparison with the initial level. The difference between premorbid and morbid adaptation levels is called an adaptation value [15]. Thus, a negative symptom is a clinical equivalent of an adaptation value.

This standpoint is in accord with the concept of a defect as a "special form of adaptation to reality" [28] because of the person's decreased functional level, which is a defect, as is the price of adaptation.

During regression of a disease, when remission becomes the prevailing "pathogenic compensatory reaction," the acute phase of the disease changes into "true compensatory reactions" [34]. During remission, "neurodynamic, reparative, and restorative character modifications" [36], representing a "safety valve" [16], result in a decrease in the acute psychotic state and promote a reduction in psychopathological symptoms. We distinguish true compensatory reactions (profound reliving and dual assessment of pathologic experiences, rationalization, exclusion, constraint of psychotic contents to the limits of consciousness, amalgamation and stereotyping), which are the result of a certain process leading to negotiation of psychopathologic symptoms.

Besides the foregoing pathogenic and true compensatory reactions formed in the presence of a full-blown disorder, the mechanisms of psychological adaptation typical for a person as a whole may occupy an important place in the adaptation system. We proceed from the hypothesis [22–23, 30, 31] that the general, fundamental rules of the mental activity of healthy people are valid and common.

Psychological adaptation includes a complex of defense formations that include: *coping* [25, 52, 60], *psychological defense* [3, 5, 17, 43], and the *internal picture of the presenting illness* [12, 38, 42]. The same *personal characteristics* have an essential influence on psychological adaptation mechanisms.

Thus, the complete concept of disease in terms of the network of a biopsychosocial model is linked not only with adaptation to environmental conditions, as I.V. Davydovskii thought [19], but with the concept of a complex of compensatory adaptation reactions. Moreover, the formation of negative psychoproductive symptoms is connected primarily with adaptation mechanisms, and productive symptoms, with compensatory mechanisms. Psychopathology reflecting the intensity of mental damage is determined by features of psychological adaptation, including subjective reactions to symptoms of a disease and treatment conditions, and also by external psychosocial factors.

The foregoing characteristics of psychological adaptation, together with the system formed by the activities of many biological subsystems, is termed *mental adaptation*. The latest hypothesis connects this with a *social adaptation* that arises from an adaptation process of the individual mind to external conditions and requirements [32].

We distinguish qualitative and quantitative characteristics of *social adaptation*. A qualitative characteristic of social adaptation is adaptive

behavior represented as a "biographicaly existing, disease- and situation-modified method of interaction with reality" [14]. A quantitative characteristic takes into consideration the level of patient functioning in different social spheres. In recent years, quality of life is a subjective characteristic of social adaptation [8].

An analysis of correlations between the level of social functioning and the nature of adaptive behavior has shown that productive forms of adaptive behavior have a high level of social progress. It is known that different psychosocial factors have an essential influence on qualitative and quantitative characteristics of social adaptation.

We could say that premorbid biological characteristics partly determine the probability of schizophrenia's beginning and the extent of its progress. In the presence of a disorder, clinical prognosis depends first on the nature of the disorder and, second, on the patient's psychological and psychosocial characteristics. However, the social prognosis is determined primarily by psychological and psychosocial characteristics. Medical improvement is not the end of treatment: it is only the basis for realization of a varied rehabilitation program that will permit the patient to make maximal use of compensatory possibilities.

References

1. Ababkov, V.A., Perre, M., & Plansherel, B. (1998) [A systemic study of family stress and coping]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhterev*, No. 2, p. 4.

2. Aleksandrovskii, Iu.A. (1976) [*Mental maladaptation and compensation*]. Moscow: "Nauka" Publishers. 272 pp.

3. Aristova, T.A. (1999) [The difference between biological and psychological components of adaptation in patients with neurosis and borderline disorders]. Author's abstract of dissertation. St. Petersburg. 24 pp.

4. Anufriev, A.K. (1974) [Compensation and adaptation as a biosocial basis of rehabilitation]. In V.P. Belov (Ed.), [*Expert medical and social-rehabilitative workup of patients with mental disorders*]. Moscow. Pp. 18–22.

5. Beznosiuk, E.V., & Sokolova, E.D. (1997) [Mechanisms of psychological defense]. *Zh. Nevropatol. i Psikhiatr. im. S.S. Korsakova*, *97*(2), 44.

6. Berner, P. (1994) [A multifactoral model of pathogenesis. Hans Hoff—A pioneer of modern psychiatry]. *Sotsial´naia i Klinich. Psikhiatr.*, *4*, 121.

7. Bekhtereva, N.P. (1977) [The human brain. A century and its last decade in study of the human brain]. *Zh. Nevropatol. i. Psikhiatr. im. S.S. Korsakova*, *77*(10), 47.

8. Burkovskii, G.V., Kabanov, M.M., Kotsiubinskii, A.P., Levchenko, E.V., & Lomachenkov, A.S. (1997) [Testing patients with schizophrenia during rehabilitation with the "Quality of Life —100" test]. In V.S. Yastrebov & V.G.

Rotstein (Eds.), [*Reform of mental health services: Problems and perspectives: Materials from an international conference*] (21–22 October). Moscow. Pp. 171–77.

9. Val′dman, A.V., & Aleksandrovski, Iu.A. (1987) [*Psychopharmacotherapy of neurotic disorders (experimental-theoretical and clinical-pharmacological analysis*]. Moscow: "Meditsina" Publishers. 287 pp.

10. Wasserman, L.I., Berebin, M.A., & Kosenkov, N.I. (1994) [A common assessment of mental adaptation]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 3, p. 16.

11. Wasserman, L.I., & Berebin, M.A. (1998) [Social frustration and its role in the genesis of schizophrenia]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 1, p. 33.

12. Vid, V.D. (1993) [*Psychoanalytic psychotherapy for schizophrenia*]. St. Petersburg. 236 pp.

13. Vilkov, G.A. (1988) [The neuroimmune processes and pathogenesis of schizophrenia]. In [*Advances in immunology and the therapy of mental disorders*]. Moscow. Pp. 48–51.

14. Volovic, V.M., Kotsiubinskii, A.P., & Sheinina, N.S. (1984) [On the characteristics of the formation of adaptative behavior in borderline patients]. In [*Early rehabilitation of patients with schizophrenia*]. Leningrad. Pp. 39–46.

15. Volozhin, A.I., & Subbotin, Iu.K. (1987) [*Adaptation and compensation—a universal mechanism of adaptation*]. Moscow.

16. Volkov, P.P. (1979) [The personality and initial mental disorders. In [*Problems in the early diagnosis and treatment of nervous and mental disorders: Thesis of the VIth Scientific Conference on Neuropathology and Psychiatry*]. Kaunas. Pp. 189–90.

17. Vorob′ev, V.M., & Konovalova, N.L. (1993) [Prophylaxis and treatment of disturbances in mental adaptation]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 1, p. 71.

18. Goncharov, M.V. (1998) [The evolutional biology of schizophrenia (Approaches to problems)]. *Rossiiskii Psikhiatrich. Zhurnal*, No. 3, p. 31.

19. Davydovskii, I.V. (1962) [*Problems of cause in medicine (etiology)*]. Moscow.

20. Dmitrieva, T.B. (1997) [Contemporary assessment approaches to psychopathologies]. *Zh. Nevropatol. i Psikhiatr. im. S.S. Korssakova*, *5*, 4.

21. Zeigarnic B.V. (1962) [*The pathology of thinking*]. Moscow.

22. Zeigarnic B.V. (1973) [*Foundations of pathopsychology*]. Moscow.

23. Zurabashvili, A.D. (1973) [Personality and antinomic reactions]. In [*Current questions in psychiatry*]. Saratov. Pp. 15–19.

24. Iovlev, B.V., & Karpova, E.B. (1998) [V.N. Miasishev's concept. Article 6. Components of attitude toward disease]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 2, p. 9.

25. Isaeva, E.R. (1999) [Coping mechanisms in adaptive behavior of patients with schizophrenia]. Author's abstract of dissertation. St. Petersburg.

26. Kabanov. M.M. (1995) [Rehabilitation of patients with schizophrenia]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 4, p. 175.

27. Kabanov, M.M. (1998) [*Psychosocial rehabilitation and social psychiatry*]. St. Petersburg.

28. Kogan, V.M. (1959) [The role of the mind in compensation]. In [*Technical*

documents from the First Congress of the Psychological Society]. Vol. 1, pp. 192–93.

29. Korolenko Ts.P., & Kolpakov, V.G. (1976) [Evolutional and biological aspects of psychosis]. *Zh. Nevropatol. i Psikhiatr. im. S.S. Korsakova*, *76*(10), 134.

30. Kochenov, M.M. (1970) [Disturbances of the sense-formation process among patients with schizophrenia]. In [*Psychological investigations*]. Moscow. Vol. 2, pp. 179–86.

31. Kochenov, M.M., & Nikolaeva, V.V. (1978) [*Motivation and schizophrenia*]. Moscow.

32. Kotsiubinskii, A.P., & Sheinina, N.S. (1996) [On the adaptation of patients with schizophrenia]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 2, p. 203.

33. Marishuk, V.L. (1995) [*Emotion and stress in sports* (*Material from an investigation*)]. St. Petersburg.

34. Melekhov, D.E. (1993) [*Clinical prognosis of the ability to work of patients with schizophrenia*]. Moscow.

35. Mikolaiski, M.V. (1985) [The ratio between biological and social factors in different clinical forms of schizophrenia]. *Vrachebnoe Delo*, No. 11, p. 103.

36. Morozov, V.M. (1953) [Schizophrenic remissions and working capability examinations]. *Zh. Nevropatol. i Psikhiatr. im. S.S. Korsakova*, *53*(10), 770.

37. Miasishev, V.N. (1960) [*Personality and neurosis*]. Leningrad.

38. Nikolaeva, V.V. (1970) [Internal picture of the disorder during mental disturbances]. Author's abstract of dissertation. Moscow.

39. Nuller, Iu.L., & Mikhalenko, I.N. (1988) [*Affective psychosis*]. Leningrad.

40. Nuller, Iu.L., Pegashova, A.E., & Kozlovskii, V.L. (1998) [Anticipation in families with mental patients]. *Sotsial´naia i Klinicheskaia Psikhiatriia*, No. 2, p. 5.

41. Perekhov, A.Ia., & Duvatova, I.V. (1998) [Treatment of febrile schizophrenia in a rehabilitation department]. *Obozrenie Psikhiatrii i Med. Psikhologii im. V.M. Bekhtereva*, No. 2, p. 31.

42. Resnikova, T.N., & Smirnov, V.M. (1976) [Modeling of the internal picture of a disorder]. In M.M. Kabanov (Ed.), [*Problems in medical psychology*]. Leningrad. Pp. 122–24.

43. Romanova, E.S. (1998) [An investigation of psychological defense mechanisms in patients with deviational behavior]. *Rossiiskii Psikhiatricheskii Zhurnal*, No. 3, p. 18.

44. Rotenberg, V.S., & Arshavskii, V.V. (1984) [*Search activity and adaptation*]. Moscow.

45. Samokhvalov, V.P. (1985) [A phylogenetic principle in analysis of schizophrenic psychopathology]. In A.S. Timofeyeva (Ed.), [*Genetic and evolutional problems in psychiatry*]. Novosibirsk. Pp. 150–74.

46. Sarkisov, D.S., Gelfand, V.B., & Tumanov, V.P. (1988) [Theoretical and practical aspects of the ratio between structure and function]. In [*Psychiatric practice*]. Moscow. Vol. 1, pp. 51–74.

47. Semenov, S.F., Mogilina, N.P., Kogan, R.D., Dotsenko, N.M., & Trekova, N.A. (1979) [Processes of compensation and adaptation during neuropsychic disturbances]. In A.A. Portnov (Ed.), [*Clinical, social, and biological aspects of compensation and adaptation during neuropsychic disturbances*]. Moscow. Pp. 10–20.

48. Tikhonenko, V.A. (1998) [The position of the personality in a situation of

conflict and suicidal behavior]. *Rossiiskii Psikhiatricheskii Zhurnal*, No. 3, p. 251.

49. Faizulaev, A.A. (1985) [Principal motive of the personality]. *Psikhol. Zhurnal*, *6*(4), 87.

50. Tsirkin, S.Iu. (1995) [Conceptual diagnosis of functional disturbances in schizophrenia: Diathesis and schizophrenia]. *Sotsial´naia i Klinicheskaia Psikhiatriia*, No. 2, p. 114.

51. Tsirkin, S.Iu., & Kulygina, M.A. (1998) [Characteristic types of personality and style of interaction with patients during psychotherapy]. *Rossiiskii Psikhiatricheskii Zhurnal*, No. 2, p. 44.

52. Chekhlatyi, E.I. (1994) [Personal and interpersonal conflicts and coping behavior and their dynamics under group psychotherapy of patients with neurosis]. Author's abstract of dissertation. St. Petersburg.

53. Schneider, F. (1997) [Neuropsychiatric research on the emotions]. *Sotsial´naia i Klinich. Psikhiatriia*, No. 2, p. 47.

54. [*Encyclopedia of medical terms*]. Moscow, 1982. Vol. 1.

55. [*Encyclopedia of medical terms*]. Moscow, 1983. Vol. 2.

56. Ciompi, L. (1980) The natural history schizophrenia in the long run. *Brit. J. Psychiatry*, No. 136, p. 413.

57. Eaton, W.W. (1978) Life events, social supports, and psychiatric symptoms: A reanalysis of the New Haven data. *Journal of Health Social Behavior*, *19*(2), 230.

58. Hurst, M.W. (1979) Life changes and psychiatric symptom development. In J.E. Barrett, R.M. Rose, & L.Kleerman (Eds.), *Stress and mental disorders*. New York: Raven Press. Pp. 17–36.

59. Laury, G., & Meerloo, J.A.M. (1969) Adaptive disability: Some precipitating factors in schizophrenic decompensation. *Psychiatric Quarterly*, *43*(2), 319.

60. Lazarus, R.S. (1966) *Psychological stress and the coping process*. New York: McGraw-Hill.

61. Lukoff, D., Snyder, K., Ventura, J., & Nuechterlein, K.H. (1984) Life events, familial stress, and coping in the developmental course of schizophrenia. *Schizophrenia Bulletin*, *10*(2), 258.

62. Olbrich, R. (1987) Die Verletzbarkeit der Schizophrenen: Zubins Konzept der Vulnerabilität. *Nervenarzt*., No. 58. p. 65.

63. Pino Morales Socorro, Lorez-Ibor Alino, J.J. (1995) [Psychosocial factors and adaptation mechanisms in stressful events]. *Actas Luso. Esp. Neurol. Psiquiatr. Cienc. Afines*, *23*(3), 124.

64. Zubin, J., & Spring, B. (1977) Vulnerability—A new view of schizophrenia. *Journal of Abnormal Psychology*, *86*, 103.

65. Zubin, J., Magaziner, J., & Steinhauer, S. (1983) The metamorphosis of schizophrenia: From chronicity to vulnerability. *Psychological Medicine*, *13*, 551.

66. Zubin, J., Steinhauer, S.R., Day, R., & van Kammen, D.P. (1985) Schizophrenia at the crossroads: A blueprint for the 80s. *Comprehensive Psychiatry*, *26*, 217.

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