Review Article:

Vitamin Deficiency and Mental Health

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The evidence for an association between nutritional deficiencies and psychiatric disorders is abundant. Subclinical deficiency in members of vitamin B group and vitamin C are common occurrences in a variety of psychiatric disorders. Folate deficiency in particular has been implicated in psychiatric disorders associated with epilepsy, in dementia, and depression. In the latter condition, folate deficiency has been proposed as an aetiological factor, and associated with severe illness and resistance to treatment. Similar conclusions were reached in connection with alcoholism. Folic acid therapy has been linked to improvements in psychiatric disturbances during anticonvulsant therapy, depressive symptoms, neurasthenic symptoms, and in overall improvement in schizophrenia, endogenous depression and organic psychoses.

The mechanisms mediating the association between folate deficiency and depression are not settled. Methylation and hydroxylation of amino acids implicated in depression are influenced by folic acid. Still folate deficiency may be secondary to depression rather than the cause of it.

A nutritional deficiency model for the psychoses is proposed. Neurotransmitter changes may result from or drain vitamin resources which are needed as cofactors in their synthesis. In turn these changes may aggravate nutritional deficiency and a vicious circle sets in.

There are several reasons why geriatric patients are likely to suffer from nutritional deficiencies. These possibilities should not be neglected in the aetiology of psychogeriatric disorders and their management.

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Introduction

Nutritional deficiencies commonly occur in a variety of psychiatric disorders. Their prevalence, however, is highest in the elderly in association with increased physical and psychiatric morbidity and with psychosocial disadvantage. Dietary surveys of elderly people in Great Britain and Sweden, showed that a substantial proportion of subjects had intakes below recommended standards. Two United States National Health and Nutrition surveys showed that 50% of the population, especially the elderly are deficient in one or more

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nutrients. The reasons for this are multiple and they include social factors such as poor financial reserves, poor housing, isolation and lack or loss of family support. Health factors include, dental problems, medication that is associated with vitamin deficiencies and commonly with gastrointestinal disorders that interfere with the absorption and digestion of nutrients. Malnutrition is commonest in the elderly living in institutions including hospitals as shown in several surveys in Europe and the United States. For these elderly, the dietary problems are more quantitative (inadequate intake) than qualitative (e.g. lack of ascorbic and folic acid) and are related to the aforementioned social and health factors.

In this paper, the evidence for an association between nutritional deficiencies and psychiatric disorders will be reviewed, laying emphasis on vitamin deficiencies and among these, focusing on folate deficiency, which offers a unique model for examining the contribution of nutritional deficiency to the aetiology of psychiatric illness and the role of folate deficiency in the aetiopathogenesis of depressive illness.

Vitamin Deficiency and Psychiatric Illness

Studies of the association of vitamin deficiency and psychiatric disorder have been predominantly ones of the prevalence of vitamin deficiency in psychiatric illness rather than studies of the psychiatric illness associated with vitamin deficiency syndromes. Extreme vitamin deficiency syndromes such as Wernicke's encephalopathy resulting from thiamine deficiency and other organic psychosyndromes caused by deficiencies in niacin, vitamin B12 and folate are relatively rare. Sub-clinical vitamin deficiency is, however, more common in the elderly, particularly those with psychiatric disorder. Morgan and Schorah (1986) compared nutritional indices in healthy elderly women with mentally ill elderly and reported lower levels in vitamin C, riboflavine, pyridoxin (B6) in the elderly mentally ill, particularly in the long-stay population. Within the elderly mentally ill, those with dementia had particularly lower values compared with the healthy controls, than did those with depression, particularly for vitamin C levels, which was also found in newly admitted patients with dementia.

Alcoholism is commonly associated with deficiencies in thiamine, riboflavine, pyridoxin, vitamin B12 and folic acid. Irritability and aggressive behaviour were reported in human volunteers undergoing thiamine restriction (Word et al., 1980). Carney (1990) reported thiamine deficiency in 58 of 154 (38%) and 30% of two series of patients admitted consecutively to the psychiatric unit of a district general hospital, with significant excesses of patients with schizophrenia and alcoholism. Thiamine deficiency appeared to be secondary to anorexia and poor diet in this population. Amongst these patients 29% were riboflavin deficient, particularly those with depressive illness, which was also reported by Nobbs (1974). The rate of pyridoxin deficiency was also high at 44% in these patients and highest for those with endogenous depression (79%). Carney et al., (1979) re-evaluated the results by combining the two series of 238 patients assessed for riboflavin and / or pyridoxin status. They showed association between deficiency and endogenous depression.

Hallert et al., (1983) showed that in patients with coeliac disease, vitamin B6

supplementation was associated with improvement in depressive symptoms when no improvement was observed after 12 months of gluten-free diet. The prevalence of low B12 is around 6%. Vitamin B12 deficiency is common in organic brain syndromes and alcoholism and is associated with long-term use of psychotropic drugs phenothiazines, tricyclics, barbiturates and benzodiazepines.

In summary, sub-clinical deficiency in thiamine, riboflavine, pyridoxin, folic acid (ascorbic acid) and Vitamin B12 are a common occurrence in a variety of psychiatric disorders, particularly in the elderly. Vitamin B12 deficiency is particularly common in organic brain syndromes, whilst all the other deficiencies are particularly common in depressive illness and alcoholism. The role of folate deficiency in psychiatric illness is examined in the next section, focusing on its role in the aetiopathogenesis of depressive illness.

Folate Deficiency in Psychiatric illness

Folic acid deficiency was shown to be a common occurrence in the general population in the United Kingdom, particularly in the elderly mentally ill (British Medical Journal, 1968). Surveys showed that up to 80% of those admitted into old people's homes had low plasma folate concentrations (Read et al., 1965).

In the U.S.A., Garry et al., (1984) studied the nutritional status of 270 healthy individuals using both dietary and biochemical measures of folate and vitamin B12. Of these, 40% had dietary intakes of folic acid and 13% of vitamin B12, less than half the recommended daily dietary allowance. Only 8% of these individuals, however, had low plasma folate and a smaller proportion low plasma vitamin B12 concentrations (3%). These workers have previously reported that individuals in the same population with low levels of plasma folate and vitamin B12 (bottom 5%) scored significantly less than the rest of the population on the Halstead-Reitan categories test, a non-verbal test of abstract thinking ability and on the Wechsler memory test, results, they argued, that it would indicate a need for further investigations of the possible consequences of subclinical deficiencies in folic acid and vitamin B12 (Goodwin et al., 1983). Surveys of folate deficiency in psychiatric patients have found variable rates of low serum folate, ranging from 10% of patients attending a psychiatric clinic to 50% of hospital patients, including alcoholic patients, drug dependent individuals and epileptic patients receiving anticonvulsant drugs. Carney (1967) examined the prevalence of folate deficiency in 423 successive admissions to a general hospital psychiatric unit and found a rate of 23%. The proportion of folate deficient patients in various diagnostic groups were 86% of epileptic patients, 30% of depressed patients, 24% of organic psychotic patients and 20% of schizophrenic patients.

Reynolds (1968) suggested that the later occurrence of schizophrenia like illness in patients with temporal lobe epilepsy might be related to folate and B12 deficiencies induced by anticonvulsant therapy. Epileptic patients with psychiatric symptoms are more likely to have folate deficiency that responded to folate treatment (Freeman et al., 1975).

Folic acid deficiency is a causative factor in certain types of dementia and lower plasma concentrations of folic acid, ascorbic acid and total tryptophan

were found in patients with senile dementia in comparison to normal subjects (Shaw et al., 1984). Bober (1984) however, found that folic and ascorbic acid deficiencies are common findings in a variety of psychiatric diagnoses in the elderly, including organic, functional and mixed states.

In a recent study of patients with dementia, folate deficiency was observed in 35% and was associated with more advanced age and greater "metabolic strain", as determined by raised erythrocyte sedimentation rate and increased plasma thyroxine concentrations (Abou-Saleh, et al., 1986). Medical conditions associated with heightened demand for folate (chronic inflammatory and malignant diseases) or with excessive loss of folate (congestive heart failure, longterm hemodialysis and acute liver disease) often present with dementia or confusional states (Shulman, 1979).

Folate Deficiency in Depression

In a study of 100 consecutive admissions with severe depression, low serum folate and vitamin B12 concentrations were found in 24% and 13% of the cases respectively (Reynolds, 1970). Folate deficient patients had more severe illnesses, had lower validity (psychic energy) scores on admission and discharge, greater neuroticism scores on discharge and responded less well to ECT and antidepressants than those with normal serum folate concentrations. Surprisingly, folate and vitamin B12 deficiencies were not related to poor nutritional history in these patients. In a study of 107 patients with recurrent affective disorders receiving long-term lithium therapy, those with lower plasma folate concentrations had a higher affective morbidity, both manic and depressive, than those with higher folate, both at the time and during the previous two years. The association was not a result of weight change or concomitant use of other drugs (Coppen and Abou-Saleh, 1982). In abstinent alcoholic patients receiving psychotherapy and social rehabilitation, low plasma folate concentrations were associated with greater affective morbidity and there was no association between low folate levels and the presence of liver function disturbance or greater severity of alcohol dependence (Merry, et al., 1982). In a series of 95 severely depressed in-patients, plasma folate deficiency was observed in 20%, particularly in those with endogenous and more severe illnesses (Abou-Saleh and Coppen, 1989) and red blood cell folate concentrations were significantly lower than in normal control subjects. Low plasma and red blood cell folate levels were not associated with weight loss in these patients.

In a study of the neuropsychiatric status of 84 consecutive admissions with megaloblastic anaemia secondary to pure folate or B12 deficiencies, patients with folate deficient megaloblastosis had significantly higher prevalence of affective disorders (56%) than those with vitamin B12 deficiency megaloblastosis (20%) (Shorvon et al., 1980).

Therapeutic Trials

Other supportive evidence for an association between folate deficiency and psychiatric illness comes from therapeutic trials of folic acid therapy. The earliest studies were on epileptic patients with psychiatric disturbance during anticonvulsant therapy who showed considerable improvement in their mood, drive and sociability following folic acid treatment (Reynolds et al., 1966). Botez et

al., (1976) described a syndrome of long-standing functional gastrointestinal disorder, abnormal diet, occult malabsorption and folate deficiency associated with fatigue, lassitude and depressive symptoms proving unresponsive to conventional antidepressant therapy. These neurasthenic symptoms responded to oral folic acid (10 mg daily) within 2-3 months of treatment, whereas the gastrointestinal and neurological symptoms improved after five months of treatment with maintenance doses of folic acid (5-10 mg weekly). Significant improvement was also observed in psychometric measures, including verbal performance intelligence tests. Carney et al (1979) noted that folate deficient patients with schizophrenia, endogenous depression and organic psychoses and who had received folate supplements showed more improvement in their mental state on discharge and spent significantly less time in hospital than similar patients with low folate levels who received no folate supplement.

In a recent study, Botez et al., (1984) explored the value of longterm therapy with folic acid in folate deficiency related disorders, including 49 patients with mild depression, 12 with folate neuropathy and 37 with soft neurological signs who had low serum and CSF folate concentrations. Sixty-eight per cent of them showed evidence of cerebral atrophy on computerized axial tomography and / or radio-nucleide cisternograms in association with cognitive impairments in psychometric tests. Patients showed significant improvement in cognitive functioning following one year of folate therapy, particularly in the block design and digit symbol sub-test; of five patients who had their radionucleide cisternograms repeated three showed improvement in cerebral atrophy.

Pathogenesis

The association between folate deficiency and depression invites speculation on the mediating mechanisms. The likelihood that such deficiency is secondary to diminished appetitive behaviour, a cardinal symptom of depression, has been seriously considered. Three studies found no significant association between dietary histories and serum folate levels on admission to hospital (Reynolds et al., 1970; Thornton and Thornton, 1978; Shulman, 1967) Another did find an association between low folate levels and poor nutrition (Carney,1967). Botez et al., (1979) put forward an alternative hypothesis suggesting that anxiety / depression causes or is associated with colitis which in turn results in folate deficiency; this is conducive to the development of neuropsychiatric symptoms, including depression, that in turn aggravates the colitis and a vicious circle is therefore created. Their suggestion must be set against the notion that the gastrointestinal system is extremely efficient in absorbing folate from all segments of the small intestine (Steinberg, 1984). The possibility that depressive patients consume or require excessive amounts of folate could be entertained. At the neurochemical level of analysis the link between folate deficiency and depression may be forged by metabolic processes such as methylation and hydroxylation. The methylation hypothesis put forward by Reynolds and Stramentinoli (1983) proposes a methylation deficit in depression. This was based on the observation that S-adenosylmethionine (SAM) has antidepressant properties. It was noted to be particularly effective in endogenous and bipolar depressive patients who later developed hypomanic

symptoms (Carney, et al., 1983). The link between folate and deficient methylation in depression was provided by Reynolds and Stramentinoli (1983) 5- methyltetrahydrofolate, which is actively transported into the nervous system and highly concentrated in the CSF and synaptic regions, is involved in the synthesis of SAM, donating its methyl group to homocysteine to form methionine which in turn is converted to SAM by the enzyme methionine adenosyl transferase.Vitamin B12 and SAM are required in catalytic amounts for the formation of methioninefrom 5-methyltetrahydrofolate and homocysteine (Spector, et al., 1980).. An alternative mechanism for the role of folate in the aetio-pathogenesis of depression involves the hydroxylation of tyrosine and tryptophan, the rate limiting step in the synthesis of catecholamines and 5-HT respectively. Folate reductase and pteridine reductase are both involved in the regeneration of tetrahydropterin (BH4), an essential co-factor in this hydroxylation process in mammalian brain (Spector, et al., 1977). Swade and Coppen (1980) have studied seasonal variation in 5-HT uptake by platelets and serum folate concentretions in depressed patients, factors that may well be related to depressive illness (Coppen et al., 1978). Interestingly, both 5-HT uptake and serum folate showed significant reductions in the months of May and June, when patients showed less favourable responses to antidepressant medication. Coppen et al., (1978) demonstrated a positive correlation between urinary indices of BH4 synthesis and plasma folate.

Implications for Nutritional Hypotheses of the Psychoses

The aforementioned data strongly suggest that folate deficiency is a common occurrence in depression and other psychiatric disorders. The nature of this association, however, remains to be further examined. It is conceivable that even if folate deficiency is simply a secondary phenomenon caused by anorexia and poor nutrition, as commonly observed in depression and other psychiatric illness (Post, 1956) it could still have an aggravating effect: it could cause further clinical deterioration, delayed recovery and contribute to chronicity and refractoriness to treatment. Such effects might be more pronounced in those predisposed to a particular monoamine deficiency, e.g. 5-HT deficient depression or in those who might need folate in excess. Accompanying deficits of methylation and hydroxylation might well be reconciled with the amine hypothesis of depression and further implicate a variety of nutritional factors involved both in the biosynthesis of monoamines and in the pathogenesis of depression such as monoamine precursors (tyrosine, tryptophan), methionine and vitamin B6, B12 and C.

A Nutritional Deficiency Model for the Psychoses

In the light of the evidence marshalled here, a nutritional deficiency model for the psychoses is proposed. As shown in Fig. 1, emotional strain may provoke neurohumoral changes including functional overactivity of noradrenaline, 5-HT and acetylcholine production (a). This would lead to increased demand on the precursors of these neurotransmitters (tyrosine, tryptophan, choline) ad catalytic co-factors (vitamins B6, B12, C and folate) (b), a situation that might be conducive to a state of negative balance (c) in these neurohumoral functions in the presence of pre-existent poor nutri-

tional (vulnerability model). These neurohumoral changes, e.g. 5-HT excess, may also induce diminished appetitive behaviour (d) with consequent poor nutrition (e) which would precipitate and / or aggravate the aforementioned negative balance (g). An alternative / additional mechanism involves the effects of strain and these neurohumoral changes on gastrointestinal function (h) affecting the absorption of these nutritional factors (i) and it is conceivable that a deficiency in one nutritional factor, e.g. folate and vitamin B12 is conducive to other nutritional deficiencies if not self-perpetuating. This may lead to a deficit state in neurohumoral function (j), deficits that may predispose, provoke, promote or aggravate psychiatric illness such as depression (noradrenaline and / or 5-HT deficit), chronic schizophrenia (type II) (dopamine deficit) and dementia (acetylcholine deficit). It is conceivable that these neurohumoral deficits are conducive to brain damage that is associated with these illnesses.

This model is reminiscent of Pauling's concept of "orthomolecular psychiatry" (1968) yet may have a heuristic value. The studies of Osmond and Hoffer (1962) had shown that large amounts of niacin are beneficial in the long-term management of schizophrenia. Folate, a highly vulnerable vitamin that was synthesized relatively recently, remains to be evaluated in the management of psychiatric illness.

Considerations for Psychogeriatric Practice

Beside the unanswered questions as to how nutrition affects the mental health of elderly people there are difficulties in both the assessment of nutritional status and establishing clear recommendations for the daily requirement for nutrients. For example, the recommended daily intake of vitamin C for the elderly in the U.S.A. is 60 mg. but only half this in the U.K. It is also unclear how illness alters these requirements.

When considering mental disorders in old age it is impossible to disregard physical health and here there is information about the effects of undernutrition which are of direct and important significance for the elderly mentally ill and their management. Low body weight is associated with increased mortality of elderly people living at home (Matilla, et al., 1986) and admitted to hospital (Bienia, et al., 1982). Poor nutrition increases the risk of hip fracture (Farmer et al., 1989) is associated with excess winter deaths (Keating, 1986), the development of pressure sores (Pinchcofsky-Devin and Kaminski, 1986) and infections (Chandra, 1989) Clearly when a specific deficiency occurs in the context of a mental disorder with known aetiological connections like the Wernicke-Korsakoff syndrome, dementia or depression-like symptoms this should be corrected as part of the primary treatment. Other deficiencies are rarely detected in clinical practice as only B12 and folate screening is performed with any regularity, yet Asplund et al., (1981) found 30% of 91 patients with a variety of diagnoses admitted to psychogeriatric inpatient care were undernourished. Nutritional status, therefore, needs to be considered as part of the clinical assessment of elderly patients. On an empirical basis nutritional supplementation should be considered whenever deficiency is established. The assessment of nutritional status is complex and a single measure is usually insufficient. It is suggest-

ed that evidence of undernutrition should be established from a combination of history and examination, anthropometry and haematological and biochemical indices (Lehman, 1991). Of the 10 risk factors from the history described by Davies (1989), depression, loneliness and alcoholism feature in addition to social variables and factors relating to dietary intake and weight change.

There are few studies of nutritional supplementation in elderly populations and more information is clearly needed. While the full import of nutritional intervention is being established a pragmatic position recommending nutritional replacement and the prevention of undernutrition should be accepted in the light of evidence demonstrating the association between nutrition and health.

The major impetus for prevention must ultimately come from a change in public policy that improves the elderly persons social, material and financial position in society and improves the availability and delivery of services to old people in their homes. The market led approach to nutrition that operates in many food rich countries has been found to increase the disparity between the nutrition and health of the rich and poor (Milio, 1989). The provision of domiciliary services is inequitably distributed, inefficiently organised and delivery is usually determined by demand rather than need (Sinclair, 1990). Consequently, valuable services like meals on wheels or home helps (often the only people to provide food for isolated mentally ill old people) may not reach those most at risk. Little attention or imagination has been given to the meals on wheels service, a potentially important resource, which suffers from a complex, multi-agency organisation, inflexibility in delivering meals of a type or at a time to suit individuals and often arriving cold. Only half the recipients find the meals at least moderately satisfactory (Sinclair, et al., 1988), while 15% never eat them and 15% only eat some of those delivered (Bebbington, et al., 1986). There is little evidence that they are targeted at those at risk of undernutrition and some evidence that the meals themselves are nutritionally inadequate (Sinclair, 1990). This has led Johnson et al., (1981) to conclude that the service acted only as a symbol of concern for elderly people.

prevention of undernutrition can be assisted by simple measures. providing meals in a form that is easily edible and for seriously cognitively impaired individuals maximising food intake during the time of day when cognitive abilities are at their peak can improve dietary intake (Suski and Nielsen, 1989). Intensive and invasive nutritional supplementation is not likely to affect the prognosis of dementia, but improved nutrition may reduce the risk of falls, infections and pressure sores, which can complicate the management of dementing people especially those in hospital. poor dentition is associated with undernutrition (1979) and the fitting of dentures to elderly people in institutional (Anderson, 1971) care has been shown to increase the consumption of raw vegetables.

Burns et al., (1989) found a group of elderly demented people in institutions to be more undernourished than those living in the community alone though they were not more cognitively impaired. It is of concern that elderly people in institutions seem more at risk of undernutrition though many factors may operate to explain this finding. Often

little attention is given to diet in institutional care and in U.K. hospitals the popular practice of expanding menus to include less colloquial dishes may be appreciated by the young, but experience suggests that the elderly do not take to lasagne, pizza and spaghetti bolognaise. Changes of this nature may actually reduce the opportunity to provide varied and balanced diets to elderly hospitalised patients. Fresh fruit is frequently difficult to obtain and modern methods of food preparation have been criticised. It is usually not appreciated that less mobile elderly people have a fast protein flux that requires a higher not lower daily intake of protein (Lehman, 1991). The time allowed for meals is normally less in institutions that at home (Hu, et al., 1986), meal times are inflexible and there is little consideration for personal preference. There is a good case to be made for the inclusion of nutritional and dietetic advice in psychogeriatric multidisciplinary teams.

A great deal needs to be known about the specific interactions between nutrition and mental health in old age. The inseparable nature of physical and mental ill health in the aged makes the evidence connecting nutrition and physical problems important to psychogeriatricians and suggestive that nutrition will be highly relevant to the management of mental disorder. Established roles for nutritional supplementation offer the prospect of simple and economic measures that may improve the treatment and prognosis of mental disorders in old age, enhance clinical recovery, improve well being and prevent morbidity. At the very least greater attention to diet may relieve common problems like constipation induced by psychotropic drugs, complicating depression or causing behaviour disturbance in the demented. A reduction in the risk of physical complications of illness, hospitalisation and aging alone cannot be ignored.

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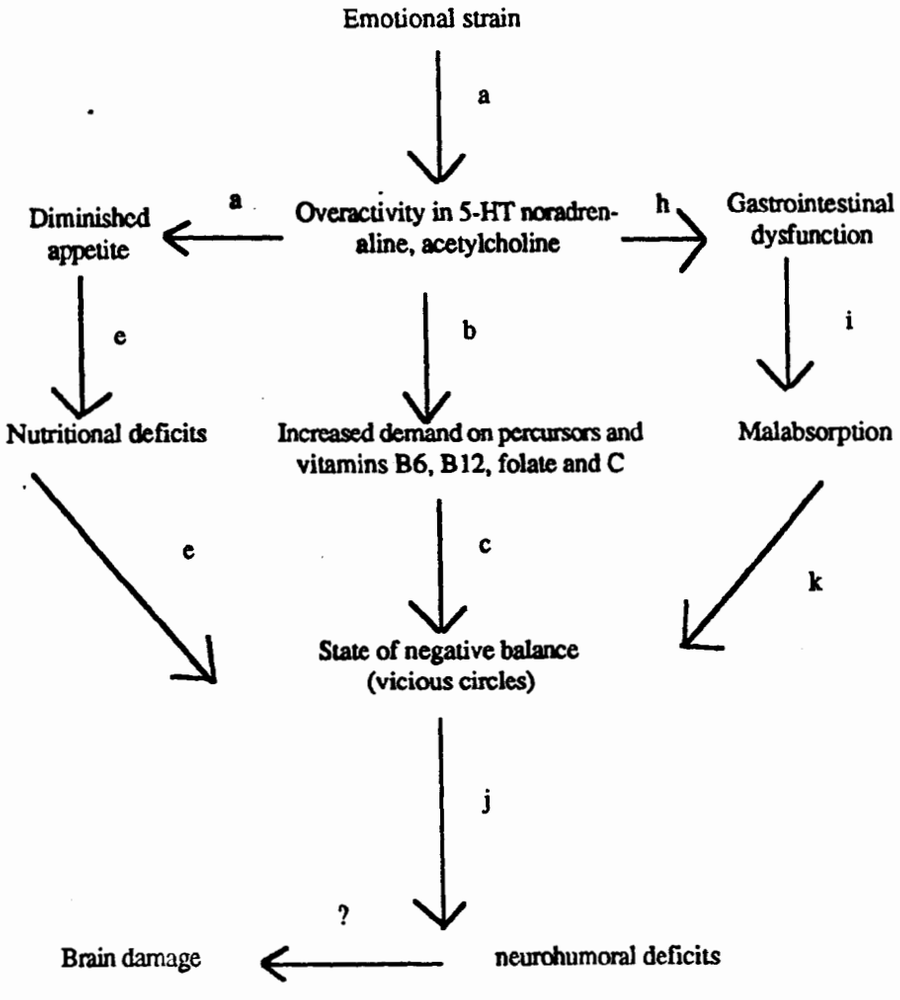
Figure 1
Nutritional Deficiency Model for the Psychoses



- 5-HT, noradrenaline in depression
- dopamine in schizophrenia

La Défaillance en Vitamines et la Santé Mentale

Léxistence d'un rapport entre une défaillance nutritionnelle et des désordres psychiatriques est evident. Les défaillances sous clinicales dans les membres du groupe de vitamine B et ceux du vitamine C sont des occurences fréquentes parmis les differentes variétés de désordres psychiatriques.

En particuliers, la défaillance en Folate, a été impliquée dans les désordres psychiatriques en association avec l'epilepsy, dementia, et la depression. Dans cette derniere condition la défaillance en Folate a été considérée comme étant une cause, et en même temps a été associeé avec les cas sevéres et ceux resistants au traitement.

On a aboutit á de pareilles conclusions en connection avec l'alcholism.

Le traitement avec l'acide folic a été lié aux améliorations des troubles psychiatriques, les symptomes de depression, les symptomes neurastheniques, et dans l'amélioration totale de schizophrenie les depressions indogenes, et les psychoses organiques.

نقص الڤيتامينات وعلاقته بالصحة النفسية

تشير العديد من الأبحاث إلى وجود ارتباط وثيق بين نقص التغذية والاضطرابات النفسية، فقد وجد مثلاً أن نقصاً غير ملموس اكلينيكياً فى مجموعة ڤيتامين ب وڤيتامين ج ينتشر فى مجموعة من الاضطرابات النفسية، كما ارتبط نقص حامض الفوليك بالصرع والعته والاكتئاب بوجه خاص وفيما يتعلق بالاكتئاب فقد يكون هذا النقص عاملاً مسبباً للمرض أو مؤثراً على حدته أو معوقاً للعلاج، هذا وقد أجريت بعض الدراسات التى وصلت إلى استنتاجات مشابهة فى حالات الإدمان الكحولى.

ومن ناحية أخرى فقد تبين أن العلاج بواسطة جرعات كافية من حامض الفوليك تؤدى إلى تحسن الاضطرابات النفسية التى تنشأ أثناء العلاج بمضادات الصرع، وأعراض الاكتئاب والإنهاك العصبى كما تساهم فى التحسن العام فى الفصام والاكتئاب غير التفاعلى والذهانات العضوية.

ومع أنه لم يمكن حتى الآن تحديد الآليات التى تربط بين نقص حامض الفوليك والاكتئاب فإن إضافة مجموعات المثيل والهيدروكسيل للأحماض الأمينية المرتبطة بالاكتئاب تعد من العمليات الكميائية التى تتأثر بحامض الفوليك، ولكنه على الرغم من هذه الاحتمالات فإنه من الممكن أن يكون نقص حامض الفوليك ناتج من حالة الاكتئاب ومصاحباتها وليس سبباً لها.

يطرح هذا المقال نموذجاً لنقص التغذية فى الذهان: فقد تكون التغيرات الناشئة فى مستويات الموصلات العصبية فى الذهان سبباً أو نتيجة لاستنفاذ الڤيتامينات الضرورية كعوامل مساعده فى عملية بناء هذه الموصلات العصبية وبدورها تؤدى هذه التغيرات إلى زيادة النقص فى مواد غذائية معينة حدة وتبدأ حلقة مفرغة فى الحدوث.

أخيراً فإن هناك العديد من الأسباب التى تجعل المرضى المسنين أكثر عرضة للمعاناة من نقص التغذية، وهو احتمال لا يجب تجاهله من بين مسببات الاضطرابات النفسية فى المسنين كما يجب عدم تجاهله عند النظر فى علاجها.