

# PELLAGRINOUS PSYCHOSIS

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## PELLAGRINOUS PSYCHOSIS

**Primary Disciplinary Field(s):** Psychiatry, Neurology, Nutritional Science

### 1. Core Definition and Etiology

Pellagrinous Psychosis refers to the severe neurological and psychiatric manifestations resulting from **pellagra**, a systemic metabolic disorder caused primarily by a profound deficiency of **niacin** (vitamin B3, nicotinic acid). This condition is categorized as a deficiency disease that critically impacts the central nervous system, leading to psychopathology ranging from mild cognitive impairment to acute delirium and chronic dementia. While niacin deficiency is the fundamental cause, the severity of the syndrome can often be compounded by coexisting deficiencies of other B vitamins, particularly **thiamine** and **riboflavin**, as well as **ascorbic acid**. Pellagra represents a classic example of how a singular, severe nutritional deficit can rapidly undermine neuronal function and integrity, causing systemic disease known historically by the triad of symptoms: dermatitis, diarrhea, and dementia.

### 2. Etymology and Historical Context

The name **pellagra** itself offers insight into the condition's most distinctive physical sign, deriving from the Italian phrase *pelle agra*, which translates literally to "rough skin." This refers to the characteristic severe, symmetrical skin lesions often developing on sun-exposed areas. Historically, pellagra was widespread in regions where diet consisted predominantly of untreated maize (corn), which contains niacin in a bound, non-bioavailable form. Although the incidence of full-blown pellagrinous psychosis has sharply declined in developed nations due to **enriched bread** programs and a robust emphasis on a balanced, fortified dietary intake, the disease remains a significant clinical problem. It persists in specific vulnerable populations, including economically deprived or backward communities globally, where nutritional security is tenuous.

### 3. Pathophysiology and Mechanisms

Niacin is essential as a precursor to the coenzymes NAD<sup>+</sup> and NADP<sup>+</sup>, which are vital for numerous metabolic processes, including cellular energy production and DNA repair. A deficiency fundamentally impairs the metabolism of high-turnover cells, including those in the skin, the gastrointestinal tract, and, most critically, the brain. The inadequate availability of these coenzymes produces demonstrable, detrimental changes in the **nerve and blood systems of the brain**. These systemic impairments compromise neurotransmitter synthesis and overall cerebral energy status, leading directly to the variety of psychological symptoms observed. The resultant damage can manifest as acute neurological crises or progressive encephalopathy, demonstrating that the psychological disorder is a direct consequence of severe nutrient-induced **brain disorders**.

## 4. Psychological Symptomatology (Pellagrinous Psychosis)

The psychological manifestations of pellagra often precede the physical signs. Early symptoms are non-specific and subtle, including **irritability**, **forgetfulness**, generalized **restlessness**, vague cephalalgia (headaches), and overall **sluggishness**. As the metabolic deficit deepens, approximately five to ten percent of affected individuals transition to more severe psychiatric symptoms indicative of acute psychosis. These severe symptoms encompass profound **confusion**, **delirium**, and acute **hallucinations**. Furthermore, advanced cases frequently present with symptomatology resembling Korsakoff's syndrome, characterized by severe amnesia and confabulation, highlighting the vulnerability of specific cerebral structures to chronic B-vitamin deficiency.

The presentation of the acute psychosis is highly dependent on the patient's underlying personality structure. Some patients become markedly **overexcited**, demonstrating agitation, mania, or anxiety, while others develop profound **depression** and withdrawal. If the underlying deficiency is not arrested and corrected, the extensive cerebral damage progresses, leading to irreversible consequences such as profound **stupor**, recurrent **convulsions**, generalized **paralysis**, and ultimately, permanent **mental deterioration**.

## 5. Populations at High Risk

Despite broad improvements in public health and nutritional awareness, pellagrinous psychosis persists within defined, vulnerable subgroups. The highest risk populations are those suffering from chronic malnutrition and severe dietary restrictions. These include **aged**, **destitute individuals** who live alone and lack access to adequate, balanced meals. Additionally, chronic substance abuse acts as a powerful predisposing factor; **alcoholics** often suffer from extreme dietary neglect and impaired intestinal absorption, while **drug addicts** typically maintain severely impoverished diets. In these populations, the combination of extremely poor nutritional intake and compromised systemic health accelerates the onset and severity of the vitamin B deficiency, making them highly susceptible to the neurological and psychiatric sequelae of pellagra.

## 6. Prognosis and Treatment

The prognosis for pellagrinous psychosis is generally positive, provided that therapeutic intervention is instituted swiftly, before irreversible structural **brain damage** occurs. The primary treatment protocol involves the immediate administration of **large doses of niacin** (nicotinic acid or nicotinamide) and the co-administration of other essential dietary components and vitamins, particularly those of the B complex, to address probable multiple deficiencies (see also BERI BERI, another deficiency-related disorder). Prompt and adequate nutritional supplementation can lead to a complete reversal of the acute psychological and physical symptoms. However, treatment delay

allows for the progression of neuronal loss and white matter changes, which results in permanent intellectual deficits and chronic mental deterioration.

## 7. Illustrative Case Study (Rosanoff, 1938)

A case documented by Rosanoff in 1938 details M.G., a white female patient who had suffered from **pellagra** for three years. She reported pre-admission symptoms including diffuse bodily pains, vertigo, muscular stiffness, and generalized weakness. Upon hospital admission, she displayed the classic **pellagrous rash** and severe gastrointestinal issues. Mentally, she was acutely **confused**, unable to recall recent events or the journey to the hospital, and was often unresponsive to questions. Her affect was dominated by terror, expressed through repetitive pleas: "I'm scared, I'm scared. What are you going to do to me? Am I going to be killed? I don't like to be left by myself."

Cognitive testing revealed severe defects in retention and concentration; she struggled with simple counting and refused mental calculations. Although she denied **hallucinations**, her behavior strongly suggested their presence. She experienced intermittent periods of relative clarity, during which she demonstrated a degree of insight, acknowledging that there was "something wrong with her mind" and that her environment felt "strange" and unfamiliar. Following successful treatment, the patient fully recovered from the acute psychotic state, retaining a well-marked **amnesia** for the duration of the confusion, with only the brief, clearer periods being readily recalled.

## 8. Further Reading

[Pellagra \(Wikipedia\)](#)

[Niacin \(Vitamin B3\)](#)

[Korsakoff's Syndrome](#)

Rosanoff, A. J. (1938). Manual of Psychiatry and Mental Hygiene.