History

Contribution of Georg Rajka in the relationship between itch and atopic dermatitis

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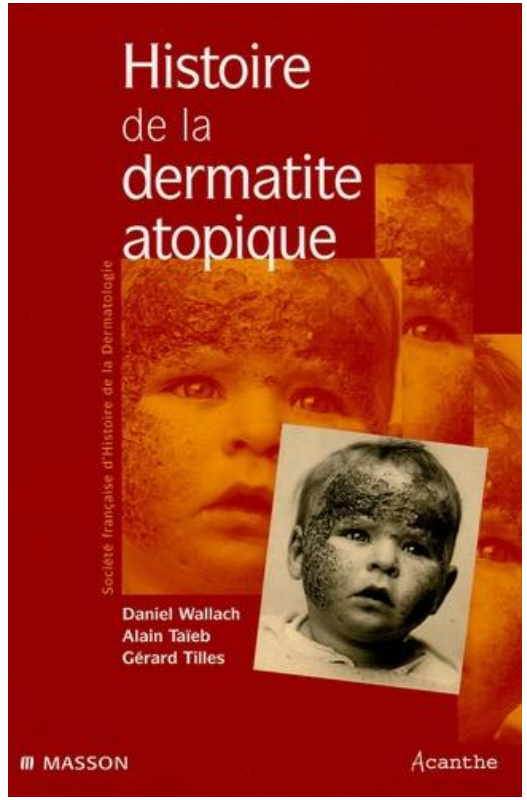
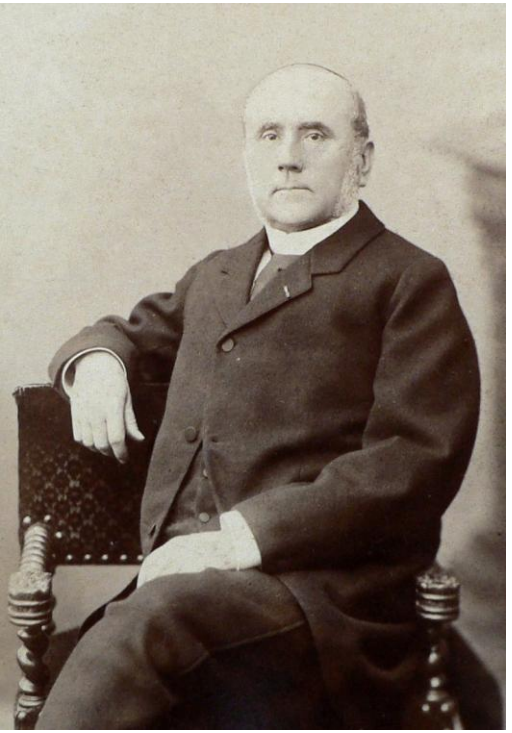
Short history of atopic dermatitis

- 1933: 1st use of this word by Fred Wise (1881-1950) and Marion Sulzberger (1895-1983) in the Year Book of Dermatology and Siphilology
- Numerous precursors describe this disease under other names, including Ernest Besnier (1831-1909), who described diathesis prurigo, which would later be named Besnier prurigo from 1892 to the 1970s, and Fernand von Hebra (1816-1880).



Prurigo of Besnier



- Ernest Besnier (1831-1909), in Paris
- 1892: description of diathesic prurigo, which later bore his name
- Now considered the first description of atopic dermatitis
- Besnier said that it is necessary, however, "to escape the tyranny of the willanic system" by going beyond descriptions of elementary lesions to bring to the fore a complete dermatological picture with the diverse clinical aspects it may take on.
- He concluded that intense, recurrent pruritus is the primary symptom of this diathesic prurigo, but also that the changes in prurigo caused by scratching are not specific and can also occur in other diseases..

Besnier E. Première note et observations préliminaires pour servir d'introduction à l'étude des prurigos diathésiques (dermatites multiformes prurigineuses chroniques exacerbantes et paroxystiques, du type prurigo de Hebra). Ann Dermatol Syphil 1892 ; 3 : 634-48.

Prurigo of Hebra

- Ferdinand von Hebra (1816-1880):
  - considered the founder of the Vienna school
  - described many different sub-forms of prurigo, such as an "ordinary" form and numerous "extraordinary" forms (e.g. prurigo simplex mihi, prurigo mitis and prurigo formicans willani), many being suggestive of the future AD
  - also described prurigo agria, known in the literature as Hebra's prurigo, with the clinical feature of a very pruritic, hard, dry papule deeply embedded in the prefiguration of nodular prurigo

Leitthema

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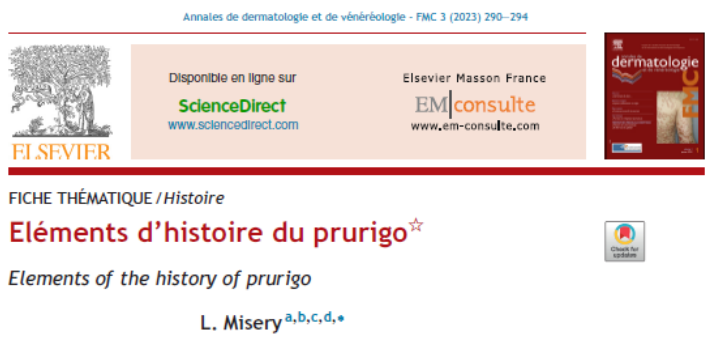
Nihil certum: Historische Entwicklung des Begriffs Prurigo



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FICHE THÉMATIQUE / *Histoire*

Eléments d'histoire du prurigo☆

Elements of the history of prurigo

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Hanifin & Rajka

- 1980, Jon Hanifin (1939-) and Georg Rajka (1925-2013) proposed the first widely used set of diagnostic criteria for AD.
- Hanifin and Rajka also recognized several synonyms for AD such as atopic eczema, Besnier prurigo, and infantile eczema.

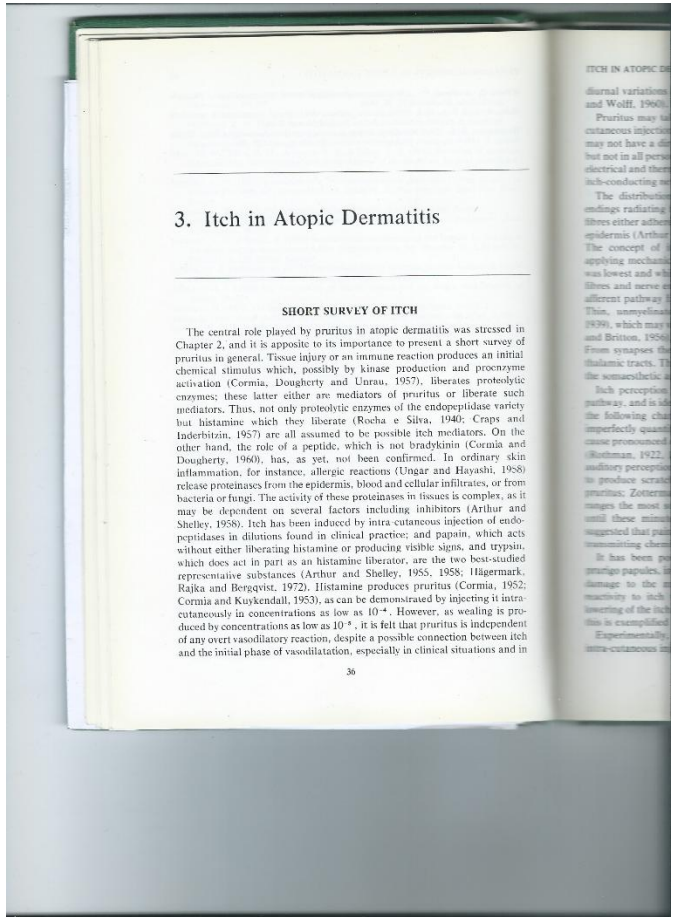
Georg Rajka

- **Born:** March 10, 1925, Budapest, Hungary
- **Died:** March 16, 2013, Oslo, Norway
- **Nationality:** Hungarian-born, later Norwegian
- **Education:** Medical degree from University of Budapest (1949); PhD on Atopic Dermatitis (1963)
- Fled Hungary after the 1956 uprising; settled in Sweden and later Norway
- Worked at **Karolinska University Hospital** and **University Hospital of Umeå**
- Became **Professor of Dermatology** at Rikshospitalet, Oslo (1971–1995)
- Authored **150+ articles** and **2 bestselling books** on Atopic Dermatitis
- Co-developed the **Diagnostic Criteria for Atopic Dermatitis** with Jon Hanifin (1980)
- Organized the **International Symposia on Atopic Dermatitis (ISAD)** starting in 1979

Georg Rajka: an impressive precursor

- In 1975, Georg Rajka made clear that he understood AD as a skin disease due to an abnormal pruritus threshold with secondary inflammation and barrier defects.
- His theory, based on experiments using trypsin as itch elicitor is a more elaborated version of Jacquet's theory of 1904 that pruritus precedes inflammation in AD.
- With our current knowledge, these ideas take on a special flavour

Non histaminergic pruritogens and pruriceptors



3. Itch in Atopic Dermatitis

SHORT SURVEY OF ITCH

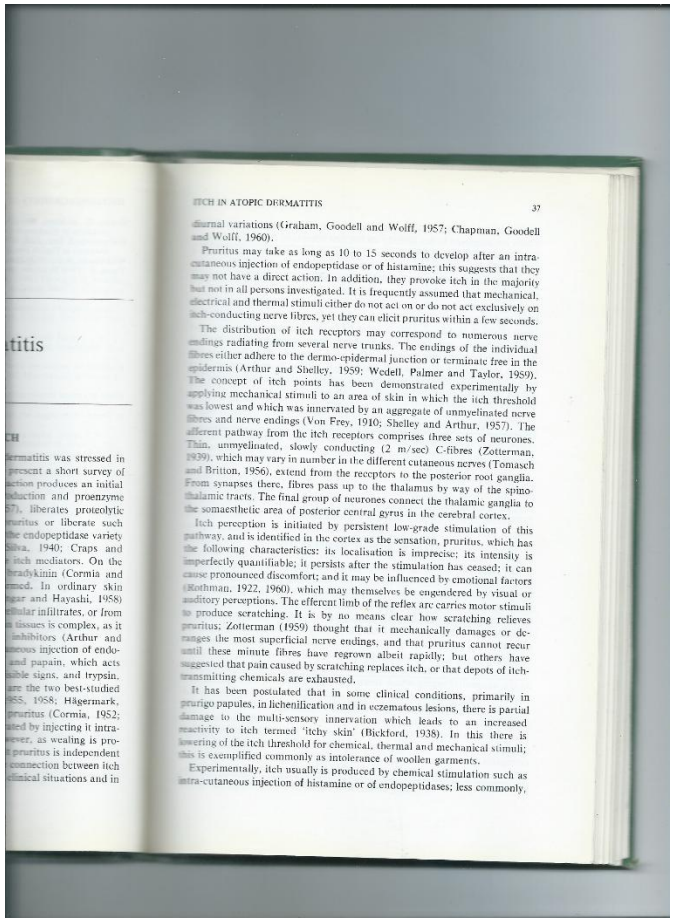
The central role played by pruritus in atopic dermatitis was stressed in Chapter 2, and it is apposite to its importance to present a short survey of pruritus in general. Tissue injury or an immune reaction produces an initial chemical stimulus which, possibly by kinase production and procnzyme activation (Cormia, Dougherty and Unrau, 1957), liberates proteolytic enzymes; these latter either are mediators of pruritus or liberate such mediators. Thus, not only proteolytic enzymes of the endopeptidase variety but histamine which they liberate (Rocha e Silva, 1940; Craps and Inderbitzin, 1957) are all assumed to be possible itch mediators. On the other hand, the role of a peptide, which is not bradykinin (Cormia and Dougherty, 1960), has, as yet, not been confirmed. In ordinary skin inflammation, for instance, allergic reactions (Ungar and Hayashi, 1958) release proteinases from the epidermis, blood and cellular infiltrates, or from bacteria or fungi. The activity of these proteinases in tissues is complex, as it may be dependent on several factors including inhibitors (Arthur and Shelley, 1958). Itch has been induced by intra-cutaneous injection of endopeptidases in dilutions found in clinical practice; and papain, which acts without either liberating histamine or producing visible signs, and trypsin, which does act in part as an histamine liberator, are the two best-studied representative substances (Arthur and Shelley, 1955, 1958; Hägermark, Rajka and Berggvist, 1972). Histamine produces pruritus (Cormia, 1952; Cormia and Kuykendall, 1953), as can be demonstrated by injecting it intracutaneously in concentrations as low as 10^{-4}. However, as wealing is produced by concentrations as low as 10^{-5}, it is felt that pruritus is indepenent of any overt vasodilatory reaction, despite a possible connection between itch and the initial phase of vasodilatation, especially in clinical situations and in

36

- Itch (pruritus) is a central and often the most bothersome symptom of atopic dermatitis.
- It can be triggered by various stimuli, including substances like histamine, but also **other pruritogenic agents** such as:
  - Proteolytic enzymes
  - Bradykinin
  - Prostaglandins
- The text emphasizes that histamine is not the only cause of itch, and that the mechanisms involved are complex.
- **Specific nerve fibers** called C-fibers and the central nervous system play a key role in transmitting and perceiving itch.

In: Rajka G, Major problems in Dermatology, Vol 3, 36-41, Saunders, 1975

Neuro-immune disease and itch sensitization



ITCH IN ATOPIC DERMATITIS 37

diurnal variations (Graham, Goodell and Wolff, 1957; Chapman, Goodell and Wolff, 1960).

Pruritus may take as long as 10 to 15 seconds to develop after an intracutaneous injection of endopeptidase or of histamine; this suggests that they may not have a direct action. In addition, they provoke itch in the majority but not in all persons investigated. It is frequently assumed that mechanical, electrical and thermal stimuli either do not act on or do not act exclusively on itch-conducting nerve fibres, yet they can elicit pruritus within a few seconds.

The distribution of itch receptors may correspond to numerous nerve endings radiating from several nerve trunks. The endings of the individual fibres either adhere to the dermo-epidermal junction or terminate free in the epidermis (Arthur and Shelley, 1959; Wedell, Palmer and Taylor, 1959). The concept of itch points has been demonstrated experimentally by applying mechanical stimuli to an area of skin in which the itch threshold was lowest and which was innervated by an aggregate of unmyelinated nerve fibres and nerve endings (Von Frey, 1910; Shelley and Arthur, 1957). The afferent pathway from the itch receptors comprises three sets of neurones. Thin, unmyelinated, slowly conducting (2 m/sec) C-fibres (Zotterman, 1939), which may vary in number in the different cutaneous nerves (Tomasch and Britton, 1956), extend from the receptors to the posterior root ganglia. From synapses there, fibres pass up to the thalamus by way of the spinothalamic tracts. The final group of neurones connect the thalamic ganglia to the somaesthetic area of posterior central gyrus in the cerebral cortex.

Itch perception is initiated by persistent low-grade stimulation of this pathway, and is identified in the cortex as the sensation, pruritus, which has the following characteristics: its localisation is imprecise; its intensity is imperfectly quantifiable; it persists after the stimulation has ceased; it can cause pronounced discomfort; and it may be influenced by emotional factors (Rothman, 1922, 1960), which may themselves be engendered by visual or auditory perceptions. The efferent limb of the reflex arc carries motor stimuli to produce scratching. It is by no means clear how scratching relieves pruritus; Zotterman (1959) thought that it mechanically damages or deranges the most superficial nerve endings, and that pruritus cannot recur until these minute fibres have regrown albeit rapidly; but others have suggested that pain caused by scratching replaces itch, or that depots of itch-transmitting chemicals are exhausted.

It has been postulated that in some clinical conditions, primarily in prurigo papules, in lichenification and in eczematous lesions, there is partial damage to the multi-sensory innervation which leads to an increased reactivity to itch termed 'itchy skin' (Bickford, 1938). In this there is lowering of the itch threshold for chemical, thermal and mechanical stimuli; thus is exemplified commonly as intolerance of woollen garments.

Experimentally, itch usually is produced by chemical stimulation such as intra-cutaneous injection of histamine or of endopeptidases; less commonly,

- Itch in atopic dermatitis is **multifactorial** and **complex**, involving:
  - **Inflammatory mediators** that sensitize skin nerves
  - **Skin barrier dysfunction**, allowing irritants to penetrate
  - **Hyperreactivity of sensory nerves**, especially C-fibers
  - **Central nervous system involvement**, which can amplify itch even without external stimuli
- Some itches persist without visible inflammation, suggesting deeper neurological involvement.
- Itch threshold is decreased.

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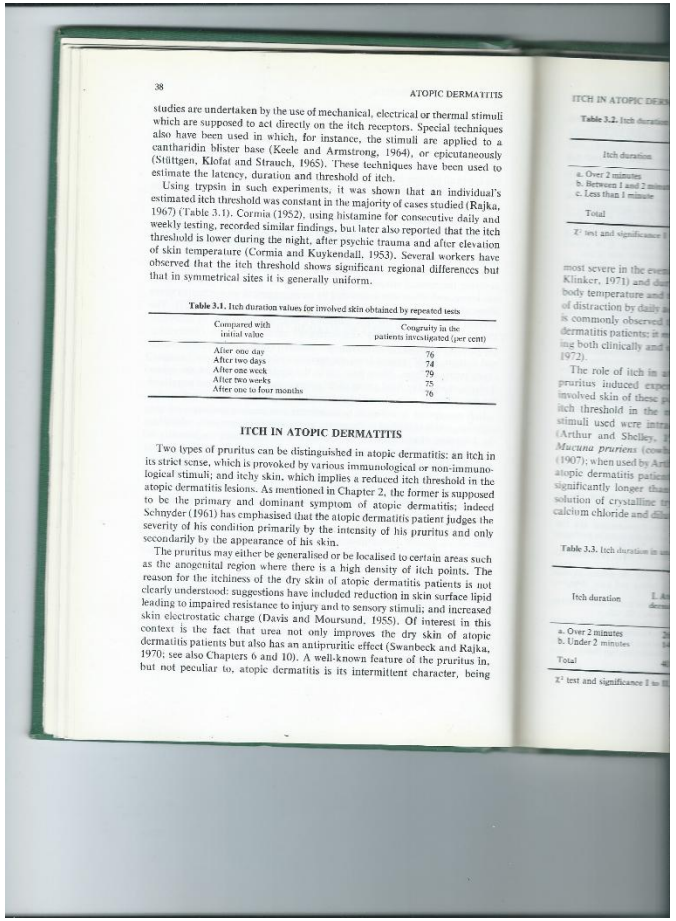
Itch–Inflammation Cycle and sensitization



38 ATOPIC DERMATITIS

studies are undertaken by the use of mechanical, electrical or thermal stimuli which are supposed to act directly on the itch receptors. Special techniques also have been used in which, for instance, the stimuli are applied to a cantharidin blister base (Keele and Armstrong, 1964), or epicutaneously (Stüttgen, Klofat and Strauch, 1965). These techniques have been used to estimate the latency, duration and threshold of itch.

Using trypsin in such experiments, it was shown that an individual's estimated itch threshold was constant in the majority of cases studied (Rajka, 1967) (Table 3.1). Cormia (1952), using histamine for consecutive daily and weekly testing, recorded similar findings, but later also reported that the itch threshold is lower during the night, after psychic trauma and after elevation of skin temperature (Cormia and Kuykendall, 1953). Several workers have observed that the itch threshold shows significant regional differences but that in symmetrical sites it is generally uniform.

Table 3.1. Itch duration values for involved skin obtained by repeated tests

Compared with initial value	Congruity in the patients investigated (per cent)
After one day	76
After two days	74
After one week	79
After two weeks	75
After one to four months	76

ITCH IN ATOPIC DERMATITIS

Two types of pruritus can be distinguished in atopic dermatitis: an itch in its strict sense, which is provoked by various immunological or non-immunological stimuli; and itchy skin, which implies a reduced itch threshold in the atopic dermatitis lesions. As mentioned in Chapter 2, the former is supposed to be the primary and dominant symptom of atopic dermatitis; indeed Schnyder (1961) has emphasised that the atopic dermatitis patient judges the severity of his condition primarily by the intensity of his pruritus and only secondarily by the appearance of his skin.

The pruritus may either be generalised or be localised to certain areas such as the anogenital region where there is a high density of itch points. The reason for the itchiness of the dry skin of atopic dermatitis patients is not clearly understood: suggestions have included reduction in skin surface lipid leading to impaired resistance to injury and to sensory stimuli; and increased skin electrostatic charge (Davis and Moursund, 1955). Of interest in this context is the fact that urea not only improves the dry skin of atopic dermatitis patients but also has an antipruritic effect (Swanbeck and Rajka, 1970; see also Chapters 6 and 10). A well-known feature of the pruritus in, but not peculiar to, atopic dermatitis is its intermittent character, being

- **Scratching** caused by itch damages the skin
- This damage releases **inflammatory mediators**
- These mediators worsen inflammation and **further sensitize nerves**, intensifying itch
- The process becomes **self-sustaining**, making the disease chronic and hard to control
- Numerous patients show **skin hypersensitivity** (Itchy skin) even without visible lesions, pointing to dysfunction in both the peripheral and central nervous systems: itch is the primary symptom.

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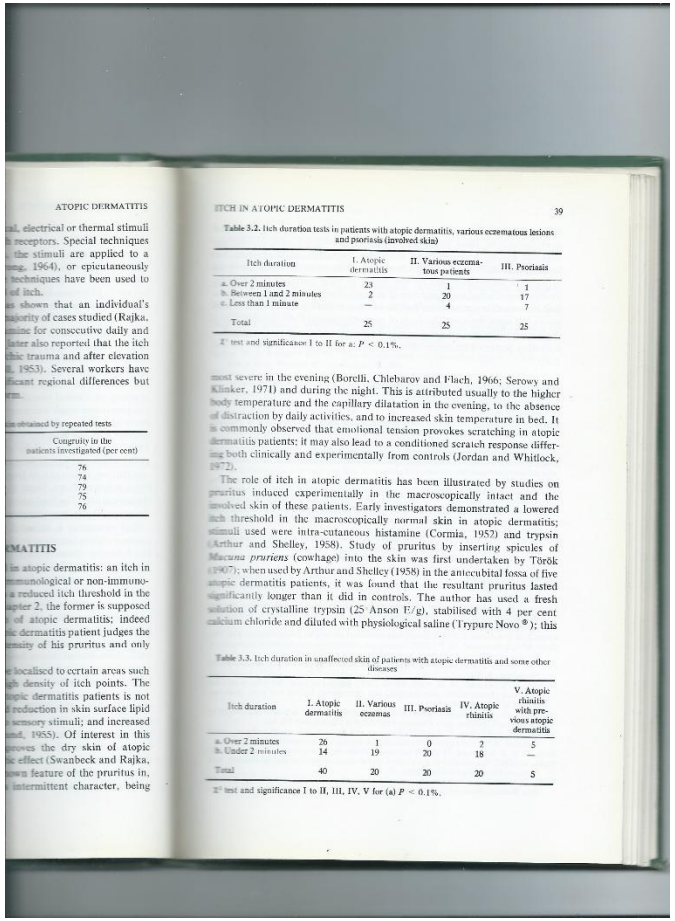
Aggravating Factors and Clinical Impact



ATOPIC DERMATITIS

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...RMATITIS

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ITCH IN ATOPIC DERMATITIS 39

Table 3.2. Itch duration tests in patients with atopic dermatitis, various eczematous lesions and psoriasis (involved skin)

Itch duration	I. Atopic dermatitis	II. Various eczematous patients	III. Psoriasis
a. Over 2 minutes	23	1	1
b. Between 1 and 2 minutes	2	20	17
c. Less than 1 minute	—	4	7
Total	25	25	25

χ^2 test and significance I to II for a: $P < 0.1\%$.

...most severe in the evening (Borelli, Chlebarov and Flach, 1966; Serowy and Klinker, 1971) and during the night. This is attributed usually to the higher body temperature and the capillary dilatation in the evening, to the absence of distraction by daily activities, and to increased skin temperature in bed. It is commonly observed that emotional tension provokes scratching in atopic dermatitis patients: it may also lead to a conditioned scratch response differing both clinically and experimentally from controls (Jordan and Whitlock, 1972).

The role of itch in atopic dermatitis has been illustrated by studies on pruritus induced experimentally in the macroscopically intact and the involved skin of these patients. Early investigators demonstrated a lowered itch threshold in the macroscopically normal skin in atopic dermatitis; stimuli used were intra-cutaneous histamine (Cormia, 1952) and trypsin (Arthur and Shelley, 1958). Study of pruritus by inserting spicules of *Mucuna pruriens* (cowhage) into the skin was first undertaken by Török (1907); when used by Arthur and Shelley (1958) in the antecubital fossa of five atopic dermatitis patients, it was found that the resultant pruritus lasted significantly longer than it did in controls. The author has used a fresh solution of crystalline trypsin (25 Anson E/g), stabilised with 4 per cent calcium chloride and diluted with physiological saline (Trypure Novo ®); this

Table 3.3. Itch duration in unaffected skin of patients with atopic dermatitis and some other diseases

Itch duration	I. Atopic dermatitis	II. Various eczemas	III. Psoriasis	IV. Atopic rhinitis	V. Atopic rhinitis with previous atopic dermatitis
a. Over 2 minutes	26	1	0	2	5
b. Under 2 minutes	14	19	20	18	—
Total	40	20	20	20	5

χ^2 test and significance I to II, III, IV, V for (a) $P < 0.1\%$.

- Several **aggravating factors** of itch are highlighted:
- **Psychological stress**, which intensifies itch via neuroimmune pathways
- **Environmental triggers** like heat, sweat, allergens, and chemicals
- **Lowered perception threshold**, making patients react to normally harmless stimuli
- Chronic itch can lead to:
  - Sleep disturbances
  - Reduced quality of life
  - Significant psychological distress
- Itch is not just a symptom—it's a **central and disabling feature** of the disease that requires comprehensive care.

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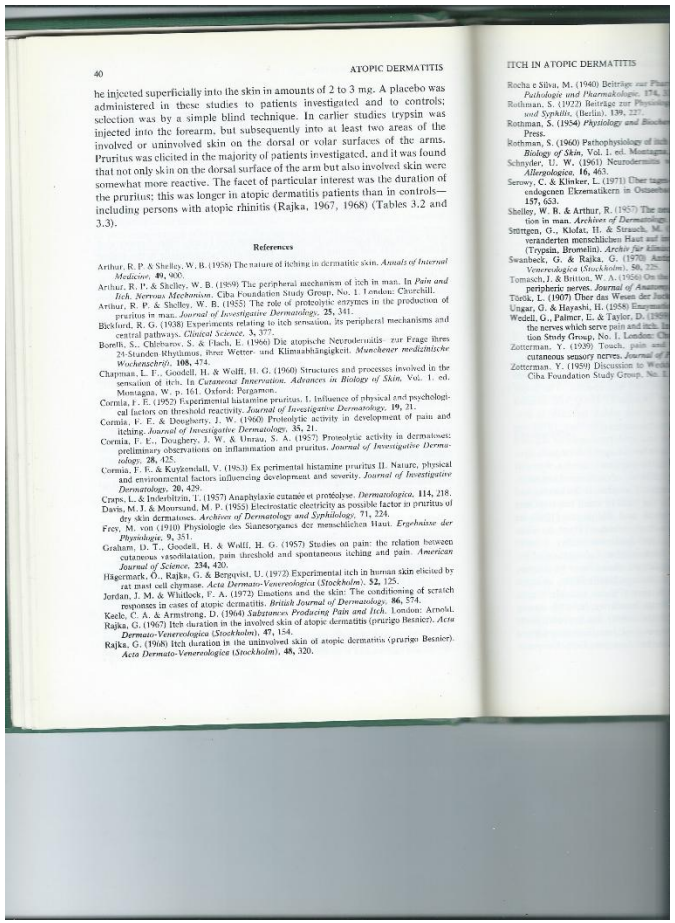
Therapeutic Perspectives



40 ATOPIC DERMATITIS

he injected superficially into the skin in amounts of 2 to 3 mg. A placebo was administered in these studies to patients investigated and to controls; selection was by a simple blind technique. In earlier studies trypsin was injected into the forearm, but subsequently into at least two areas of the involved or uninvolved skin on the dorsal or volar surfaces of the arms. Pruritus was elicited in the majority of patients investigated, and it was found that not only skin on the dorsal surface of the arm but also involved skin were somewhat more reactive. The facet of particular interest was the duration of the pruritus; this was longer in atopic dermatitis patients than in controls—including persons with atopic rhinitis (Rajka, 1967, 1968) (Tables 3.2 and 3.3).

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- The text concludes with **treatment approaches** for itch in atopic dermatitis:
  - **Traditional antihistamines** are often ineffective, as histamine is not the sole mediator
  - New therapeutic targets include **cytokines** and **specific neural receptors**
- Treatments should aim to **reduce inflammation**, **restore the skin barrier**, and **modulate nerve transmission**
- A **multidisciplinary approach** is recommended, involving dermatology, allergology, and psychological support when needed
- Itch is now recognized as a **complex, central, and disabling symptom**, requiring personalized and holistic management.

In: Rajka G, Major problems in Dermatology, Vol 3, 36-41, Saunders, 1975

Conclusions

- Georg Rajka had guessed some concepts, which have been m
  demonstrated nowadays:
  - Non histaminergic pruritus
  - Role of proteases
  - Pruriceptors
  - Neurogenic inflammation
  - Itch-inflammation cycle
  - Itch sensitization
  - Role of both central and peripheral nervous system

I would like to thank Prof. Alain Taieb for bringing this matter to my attention and for providing me with historical documents.

30 janvier 2026

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30 janvier 2026

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Pour s'inscrire:

PROGRAMME

9h-9h45 : Prurit urémique - *Guy Rostoker*
9h45-10h30 : Prurit cholestatique - *Jérôme Gournay*
10h30-11h : *Pause*
11h-11h45 : Physiopathologie - *Matthieu Talagas*
11h45-12h30 : Prurigo nodulaire - *Emilie Brenaut*
12h30-13h30 : *Déjeuner*
13h30-14h15 : Prurit hématologique - *Christelle Le Gall*
14h15-15h : Prurit psychogène et conséquences psychologiques du prurit - *Barbara Ferreira*
15h-15h30 : *Pause*
15h30-16h15 : Quel bilan ? Quel parcours de soin ? - *Serge Boulinguez*
16h15-17h : Panorama thérapeutique - *Laurent Misery*