Global Journal of Translational Medicine

Research Article

Inflammation and Neuroinflammation Caused by Environmental Factors

Kurt Müller[1]* and Martina Wagner[2]

[1]Dresden International University (DIU), Department Functional Medicine, Germany.

[2]Cooperating practitionar, specialist in Functional and Occupational Medicine, Waidhofen, Austria.

ABSTRACT

A wide variety of non-physiological chemical and physical influences affect humans in all areas of modern life. These factors can trigger diseases and, through epigenetic effects, may also affect subsequent generations. Chronic exposure to environmental stressors induces inflammatory reactions that are associated, among others, with allergies, mast cell activation syndrome, and depression.

TRPV1 is a non-selective cation channel whose activation by chemicals is considered a key pathomechanism of MCS. Both endogenous stress responses and chronic inflammation can lead to increased oxidative stress, resulting in severe performance decline up to burnout or chronic fatigue syndrome.

The most important therapeutic principle and prerequisite for further therapeutic interventions is the interruption or elimination of disease-triggering mechanisms.

Keywords

Inflammation, Neuroinflammation, Environmental toxins, Clinical Environmental Medicine, COVID-19, Depression, Transient receptor potential vanilloid 1, Multiple Chemical Sensitivity, Stress response

Corresponding Author Information

Dr. Kurt E Müller
Leutkircher Strasse 27b, D-87439 Kempten/Germany.

Received: Januaryr 09, 2026; **Accepted:** February 18 2026; **Published:** February 27, 2026

Citation: Kurt Müller, Martina Wagner. Inflammation and Neuroinflammation Caused by Environmental Factors. Trends Global J Transl Med. 2026;2(1):1-9.

Abbriviations

ATP: adenosine triphosphate, CBD: cannabidiol, CGRP: calcitonin gene-related peptide, CFS: chronic fatigue syndrome, COVID-19: corona virus disease 2019, COMT: catechol-O-methyltransferase, EMF: electromagnetic fields, GABA: gamma-aminobutric acid, HMGB1: high-mobility group box proein, IFN-y: interferon-y, IDO: indoleamine-2,3-dioxygenase, IL-1ß: interleukin-1ß, IL-2: interleukin-2, IL-6: interleukin-6, IL-8: Interleukin-8, IL-4: interleukin 10, LTB4: leukotriene B4, MAO: monoamine oxidase, MCAS: mast cell activation syndrome, MCS: multiple chemival sensitivity, mod-mRNA: modified messanger ribunuceic acid, NEIS: neuroendocrine-immune system, NF-κB: nuclear factor-kappa B, NMDA: N-methyl-D-aspartate, NSAIDs: non-steroidal anti-inflammatory drugs, PCBs: polychlorinated biphenyls, PCP: pentachlorphenol, PKA: protein kinases A, PKC: protein kinases C, REACH: registration, evaluation and authorisation of chemicals, ROS: reactive oxygen species, SAM: S-adenosylmethionine, SP: substance P, TGF-ß: transforming growth factor-ß, TH1: T helper cells 1, TH2: T helper cells 2, TNF-α: tumor necrosis factor-α, Treg: regulatory T cells, TRP: transient receptor potential, TRPV1: transient receptor potential vanilloid 1.

Introduction

From embryonic development until death, humans are exposed to complex physiologically necessary as well as harmful environmental influences. The extent of non-physiological chemical and physical influences originating from geographical, occupational, and personal environments, as well as changes in food production and preparation, has increased to a degree that individuals can no longer reliably assess.

In addition, spaces that are essential for human development—air, water, and soil—are now used as repositories for pollutants, and naturally occurring electromagnetic fields have been expanded by non-physiological sources. The acceptance of so-called "background exposures" was not preceded by sufficiently long and thorough scientific investigations to demonstrate their harmlessness.

Examples include the former use and late prohibition of γ-hexachlorocyclohexane (lindane), PCP, PCBs, as well as emissions of dioxins and furans from incineration plants. It is therefore not surprising that a study conducted in six European Union countries found that 7% of all diseases are caused by a limited number of nine environmental factors (benzene, formaldehyde, dioxins/furans/PCBs, lead, ozone, particulate matter, radon, noise, and passive smoking) [1].

The prevalence of environmentally associated diseases thus corresponds to that of the most common widespread diseases. More recent data are not available, and consequences derived from these findings are still lacking.

This demonstrates how unrealistic the concept of primary prevention remains in this field. Current official assessments of glyphosate, hydraulic fracturing, or risks posed by non-natural EMF clearly show that a paradigm shift is still far away. Under current healthcare strategies, at best secondary or tertiary prevention can be achieved—if a risk is even recognized as such.

A major obstacle to identifying environmental medical factors as causes of disease lies in the fact that various established disciplines each claim authority in determining what constitutes a risk. These disciplines rely on the limited explanatory power of their preferred methodologies or focus on exposure within restricted life environments (toxicology, occupational medicine, hygiene, allergology).

Only Clinical Environmental Medicine has begun to integrate established methods and to assess the interaction of various chemical and physical influences across all areas of daily life, taking into account nutrition, stimulants, drugs, electromagnetic fields, and chronic infections. Furthermore, research into the epigenetic effects of environmental factors has expanded understanding of their interaction with the NEIS [2-4].

A decisive breakthrough was achieved when it became clear that epigenetic effects of environmental influences not only alter the susceptibility of the affected generation but also that of the subsequent two to three generations. It was further demonstrated that not only classical allergic reactions represent dose-independent responses of the organism to substances, but that chronic inflammation induced by various immunological mechanisms leads to clinical symptoms depending on the levels of expressed cytokines [5].

Finally, inflammatory reactions triggered by xenobiotics were described as being independent of the dose–response principle [6].

Exposure Cessation

The cessation of exposure to disease-triggering noxae represents the primary therapeutic principle in the treatment of environmental medical disorders. In recent years, substantial progress has been made in identifying triggers and their individually variable pathological mechanisms. Avoidance of such environmental stressors is generally successful within individually controllable settings, owing to the typically good compliance of affected patients. This applies particularly to the various forms of food intolerances and their resulting consequences.

The relevance of medically used alloplastic materials has been recognized and is now accessible to standardized diagnostic procedures [7]. The development of therapeutic solutions is therefore possible. In dentistry, significant progress has recently been achieved through the establishment of the field of environmental dentistry. Cooperation with this discipline is indispensable. Comparable advances have not yet been achieved in other medical specialties (e.g., orthopedics, surgery, internal medicine/cardiology).

To this day, authorities and institutions continue to follow a principle of trivialization and leave the clarification of causal relationships to a medical community that is not supported by research funding or third-party grants, or to the affected individuals themselves. The opportunity to use the REACH regulatory framework for a fundamental strategic shift was missed [8].

Inflammation

As early as 2001, an immunology textbook pointed out that the susceptibility of cells to intracellular pathogens is significantly increased in the presence of IFN-γ: *"IFN-γ makes cells abnormally susceptible to intracellular pathogens"* [9]. The relevance of this mechanism has been demonstrated in patients suffering from MCS [6].

The facilitated penetration of noxae into the intracellular compartment through exposure to combinations of lipophilic and hydrophilic pollutants, as commonly encountered in everyday life, has been described [10]. IFN-γ expression is associated with lymphocytic inflammation and thus with activation of TH1 cells, as is the pro-inflammatory interleukin-17. Regulatory counterbalances include IL-4 and TH2 cells as well as IL-10 and

Treg cells. Increased proliferation of sensitized T lymphocytes is regulated by IL-2.

In myelomonocytic inflammation, organ-specific macrophages—derived from circulating monocytes—play a central role. TNF-α, IL-1, IL-6, and IL-8 are the dominant cytokines in this context. Mast cell activation is generally associated with TH2 cell activation. Clinically, this results in type I allergies, pseudoallergies, mast cell disorders, and histamine intolerance.MCAS is increasingly diagnosed following COVID-19 infection and mod-mRNA gene therapy [11]. Histamine, leukotrienes, TGF-β, and serotonin are key mediators and cytokines [7]. The TH2 system is particularly stimulated under certain stress conditions, which will be discussed later.

The immune system preferentially employs the immunological strategy best suited to the task at hand. Given the relatively short evolutionary exposure to environmental noxae since the industrial revolution, the development of entirely new immunological mechanisms is unlikely. Instead, the immune system applies long-established strategies to new challenges, selecting the mechanism with the highest likelihood of success. This has also been demonstrated in studies of immunological effects induced by 5G exposure. Within 30 minutes, an increase in IL-1β was observed, and after 90 minutes, TNF-α levels were pathologically elevated [12] (Figures 1 and 2).

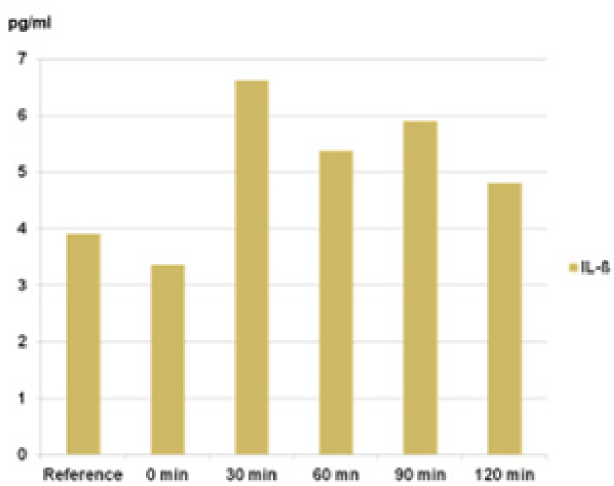

Figure 1: Operation of an iPhone 12 at a distanceof 10 cm from the ear. Expression of IL-1ß at 30-minute intervals. The rear comumns indicate the upperlimit of the reference value.

Inflammation characterized by moderate but chronic elevations of cytokines and mediators is commonly referred to as *silent* or *smouldering inflammation*.

Relationship to Depression

The functional coupling of inflammation and depression was an evolutionary necessity [13], aimed at improving survival during illness by economizing energy expenditure through reduced drive and passivity, as inflammation increases energy consumption by approximately 20–25% [14]. This elevated energy demand must be compensated by energy savings elsewhere. This is achieved through the release of pro-inflammatory cytokines, which induce IDO activity, thereby reducing serotonin production to 1–10% of baseline levels [15,16].

This leads to diminished motivation, activity, initiative, and physical as well as cognitive agility. Simultaneously, cortisol production is activated to control inflammation. This is accompanied by reduced synthesis of sex hormones [14], resulting in decreased libido and fertility. The organism focuses on what is essential: control of inflammation. From an environmental medicine perspective, the increase in cortisol is not the cause of depression—as suggested in psychiatric literature [17]—but rather serves an anti-inflammatory function [14].

Depression thus results from inflammation-dependent enhancement of kynurenine synthesis at the expense of serotonin production [15,18-21]. It is therefore not surprising that anti-inflammatory approaches are increasingly discussed in psychiatric literature [22]. Chronic infections are receiving growing attention in this context. However, the fact that chronically acting environmental factors may trigger comparable mechanisms has not yet been adequately considered. Consequently, the identification of such influences and strategies for exposure reduction remain largely neglected within psychiatry.

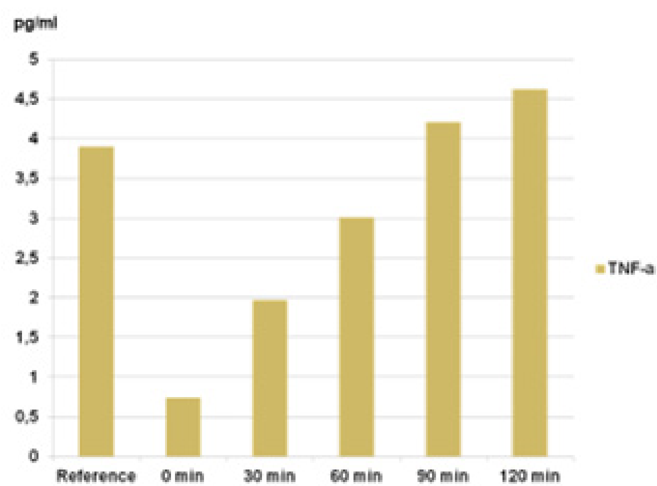

Figure 2: Operation of an iPhone 12 at a distanceof 10 cm from the ear. Expression of TNF-ɑ at 30-minute intervals. The rear comumns indicate the upperlimit of the reference value.

Therapeutic Implications

It becomes evident that therapeutic control of inflammation is as complex as its initiation and is highly individual. Broad anti-inflammatory therapies using corticosteroids, NSAIDs, and immunosuppressive agents will need to be replaced by treatments with lower risk and greater specificity. Current research on so-called *biologics* targets individual immunological pathways, often accepting substantial side-effect risks.

In environmental medicine, the evaluation of natural anti-inflammatory agents and the assessment of their effects in individual patients are favored and have shown good success. Responses vary significantly between individuals, meaning that different substances may be required to achieve the same therapeutic goal. The TNF-α inhibition test allows identification of the most suitable substance for this purpose. In our own applications of this method, SAM has proven to be the most effective agent. It can also be used to modulate catecholamine-induced stress responses [23].

Following the wave of gene-based therapies related to COVID-19 infections, it is now particularly advisable to evaluate the effects of natural anti-inflammatory substances such as omega-3 fatty acids, curcumin, garlic, epigallocatechin gallate, quercetin, and ginger, and to align their use with therapeutic goals, as paradoxical effects may occur.

Transient Receptor Potential Vanilloid (TRPV1) – the Link to Neuroinflammation

TRPV receptors belong to the superfamily of transient receptor potential (TRP) ion channels, which comprises 30 subtypes [24]. TRPV1 was the first cationic ion channel identified to be activated by capsaicin and was therefore initially referred to as the vanilloid receptor. It is often used *pars pro toto* when referring to this receptor family. TRPV1 is a non-selective cation channel whose activation induces calcium and sodium influx and stimulates nociceptive neurons. Accordingly, its significance has primarily been associated with pain perception.

TRPV1 exhibits thermo-, mechano-, and chemosensory properties, the latter being a key factor in the development of MCS [25,26]. Its role in susceptibility to infections and infection-related symptoms has also been described [27]. The TRPV1 protein is encoded by the TRPV1 gene located on chromosome 17p13 and has a molecular weight of 95 kDa. It is a tetrameric protein composed of four monomers, each containing six transmembrane segments (S1–S6). The cation-permeable pore is located between segments S5 and S6. Each transmembrane segment possesses both N-terminal and C-terminal domains. Capsaicin binds to the N-terminal domain (Figure 3).

A large and diverse number of chemical substances are capable of activating these domains, thereby inducing calcium and sodium influx into the cell [27].

In MCS, TRPV1 activation is likely the decisive pathomechanism [25,26]. Patients exhibit increased sensitivity to noise and light and intolerance to specific temperature ranges, which differ among TRPV receptors (TRPV1–4) (Figure 4). The immunological role of TRPV1 in regulating inflammation, its relevance for neuroimmune interactions, mitochondrial biogenesis, energy metabolism, and autophagy remains underestimated [28].

Its role in susceptibility to and symptoms of COVID-19 infection has been described [27]. Inactivation of TRPV1 improves pulmonary function and accelerates viral clearance in COVID-19. TRPV1 has been detected in numerous cells, tissues, and organs, including membranes, epithelia, muscle tissue, skin, gastrointestinal tract, lungs, and urinary bladder.

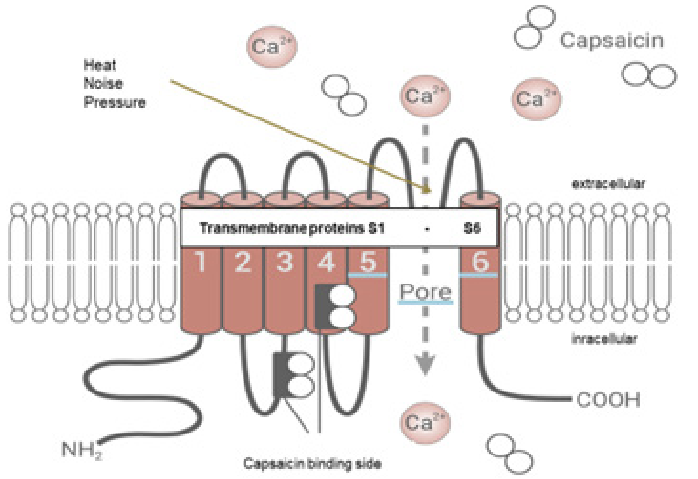

Figure 3: Structure of TRPV1. S1-S6 transmembrane proteins. Pore between S5 and S& for calcium influx upon activation. Capsaicin binds to the N-domain, while others also bind to the C-domain.

Activators of TRPV1	Activators of TRPV4
43°C Capsaicin Endocannabinoids Endovanilloids Phosphatidylglycerol (Phospholipid) Nerve Growth Factor low pH Alcohol Allicin (garlic) Venom (natural poison)	28 – 35°C Anantamid (Cannabinoid) Serotonin Histamine Endothelin (Peptid hormon) Lipids Phorbol ester (natural carcinogen) Osmotic pressure Mechanical pressure Andrographis paniculata: extract of Kalmengh
Causing	
Neuropathic Visceral Inflammatory pain Headache Itching Burning of the skin Stinging of the skin	Neuropathic Visceral Inflammatory Mechanical pain Headache Itching Burning of the skin Stinging of the skin

Figure 4: Differences in activators and effects between TRPV1 und TRPV4.

Within the nervous system, TRPV1 is expressed in astrocytes, microglia, neurons, olfactory nerves, spinal cord, basal ganglia, thalamus, hippocampus, amygdala, cerebellum, and neocortex. This widespread distribution explains the diverse neuropsychiatric symptoms observed upon receptor activation. In the immune system, TRPV1 expression has been demonstrated in T lymphocytes, macrophages, natural killer cells, dendritic cells, and neutrophils.

TRPV1 activation induces the release of IL-6 and activates NF-κB. Interactions occur with neuropeptides such as CGRP, SP, somatostatin, and other neurotransmitters. Noradrenaline enhances the effect of SP in neurons (Figure 5).

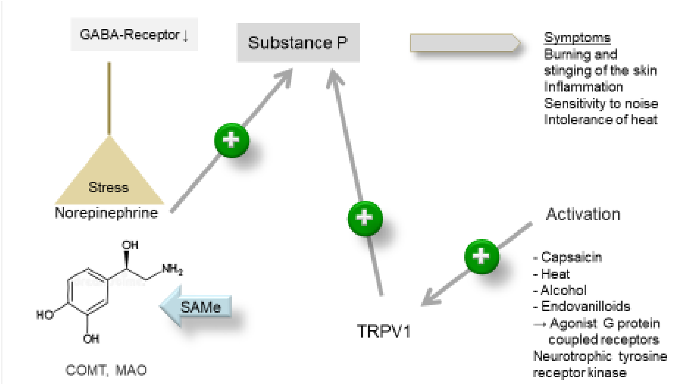

Figure 5: TRPV1 is activated by physical, chical, and thermal stimuli. This leads to the release of substance P. This response is enhanced by norepinephrine, which increases during stress reactions. In presence of slow COMT and/or slow MAO, this reaction is more intens.

Antigen receptors on immune cells—such as the T-cell receptor, B-cell receptor, and chemokine receptors—can trigger calcium influx from the extracellular space, thereby increasing mitochondrial ROS. TRPV1 activation exerts pro-inflammatory effects, including the induction of IL-1β, IL-6, TNF-α, and HMGB. LTB4, which is elevated in salicylate intolerance, also activates TRPV1 [29].

The extent to which chronic TRPV1 activation—with sustained calcium influx and associated oxidative stress—may induce apoptosis or autophagy and promote calcium deposition leading to neurodegenerative disorders (e.g., Fahr's disease) remains poorly investigated.

There is evidence that during midlife a shift toward anti-inflammatory effects (IL-4, IL-10, IL-13, TGF-β) may occur (Figure 6). Our own investigations in post-COVID/post-vaccination patients revealed that in cases of pronounced TRPV1 symptomatology (hypersensitivity to noise, light, and temperature; intolerance to frequencies <30 Hz; burning and stinging skin sensations), both TH1 and TH2 axes as well as Treg cells may be downregulated (Figures 7-10). In the presence of CD39++ Treg cells, ATP dephosphorylation leads to significant energy loss.

Additionally, TRPV1 activation itself consumes ATP via phosphorylation of PKA, PKC. This process is associated with vascular inflammation such as vasculitis racemosa/livedo reticularis and may progress to polyarteritis nodosa.

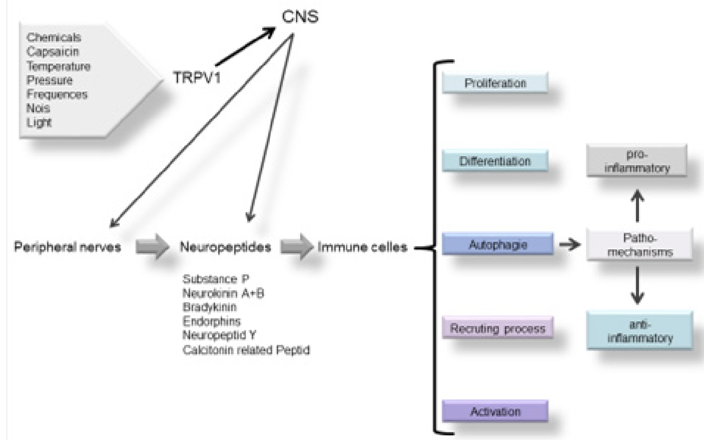

Figure 6: After activation of TRPV1, the brain takes on the function of a relay system.

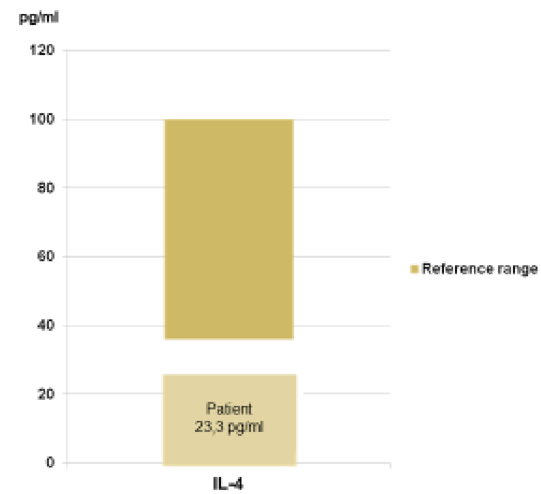

Figure 7: Expression of IL-4 (23,3 pg/ml) by TH2 cells in a patient with post-vaccination-syndrome and activated TRPV1. Reference range: 30-100 pg/ml.

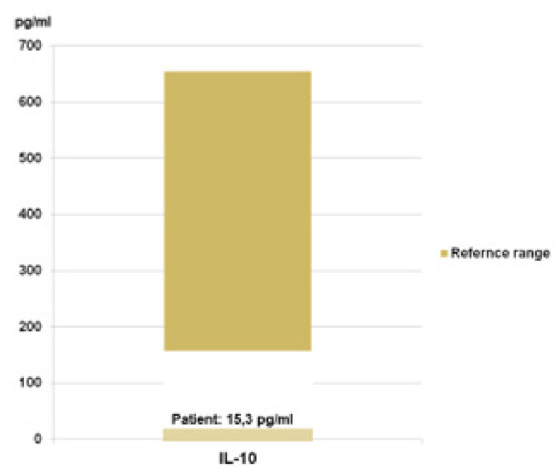

Figure 8: Expression of IL-10 (15,3 pg/ml) by Treg cells in a patient with post-vaccination-syndrome and activated TRPV1. Reference range: 168-654 pg/ml.

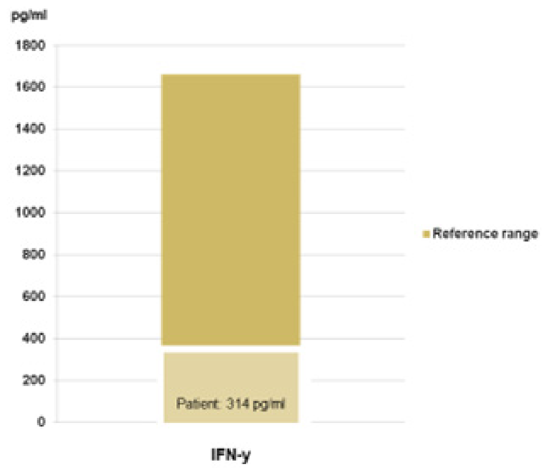

Figure 9: Expression of IFN-y (15,3 pg/ml, reference range 374 – 1660 pg/ml) in a patient with post-vaccination-syndrome and activated TRPV1.

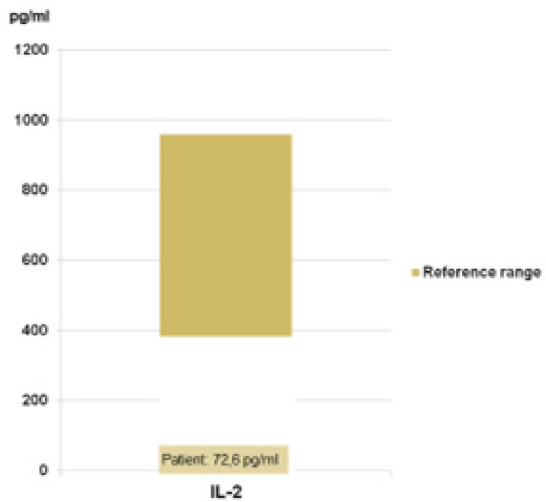

Figure 10: Expression of IL-2 (72,6 pg/ml, reference range 384 – 960 pg/ml) in a patient with post-vaccination-syndrome and activated TRPV1.

Therapeutic Considerations

Therapy remains challenging. Physically induced effects may be cautiously desensitized through showering or brush massage (the English term *desensitization* is more appropriate). Capsaicin activates TRPV1 but may induce desensitization similar to bee or wasp venom. Case observations indicate that following TRPV1 stabilization, reactions to venoms become markedly less severe.

Menthol and CBD inhibit TRPV1, whereas heat and acidic solutions activate it. Caffeine stimulates calcium influx and may paradoxically induce desensitization, as patients frequently report calming effects. Because TRPV1 activation is often associated with activation of the NMDA receptor [25,26,30], magnesium taurate, liposomal zinc citrate, and selenium are beneficial. In severe cases, injectable preparations may be required.

Vitamin B6 promotes the conversion of glutamate to GABA, providing additional therapeutic support. Pregabalin has proven particularly useful, as it inhibits calcium influx during TRPV1 activation.

Experimental research predominantly focuses on lipophilic molecules such as N-arachidonoyl dopamine, N-oleoyldopamine, oleoylethanolamide, and 12- and 15(S)-hydroperoxyeicosatetraenoic acid. Agonists include rimocidine A, evodiamine, butacaine, piperine, nicotine, and unsaturated terpenes. Several cannabis species also act as agonists [31,32].

TRPV1 antagonists are categorized into those that induce hyperthermia, those that do not, and those for which no information is available. The latter group includes grifolin and neogrifolin (isolates from *Peperomia galioides*, *Rhododendron dauricum*, *Albatrellus* spp.), as well as alkaloids such as yohimbine, voacangine, pellitorine, monoanchomycalin B, and pulchranin.

Ginsenosides exert inhibitory effects but increase body temperature. Thapsigargin also inhibits TRPV1 but exhibits significant toxicity. Flavonoids such as naringenin, cochinchinenins A and B, loureirin B, gomisin A, eriodictyol, vitexin, and quercetin display antagonistic effects without inducing hyperthermia, as do eriodictyol and naringin from citrus fruits [31].

Pregabalin is easy to administer, with a broad dosage range of up to 300 mg daily. Cytochrome enzymes are not required for its metabolism. Approximately 80% is excreted unchanged via the urine, necessitating dose reduction in renal insufficiency. The remaining 20% is eliminated through hydrolysis or conjugation. QTc prolongation has been reported. Respiratory depression may occur in association with gabapentinoids, particularly in cases of substance abuse [33]. Addictive potential is suspected, although withdrawal symptoms have not been observed.

Stress Response

The term *stress*, borrowed from physics, was first used by Hans Selye to describe the "nonspecific response of the body to any demand" [34]. It has become customary to attribute stress primarily to psychosocial factors such as excessive demands and conflicts at work, school, or in personal relationships, as well as time pressure. However, following Selye's broad conceptualization developed during his teaching in the United States, all factors that lead to persistent disruption of homeostatic stress-compensation mechanisms must be considered.

Stress represents a network of immunological, neurogenic, and hormonal factors that is temporarily highly efficient [35,36]. From this perspective, unphysiological chemical and physical environmental exposures qualify as stressors to the same extent as chronic infections, intense mental or physical strain—including excessive athletic training as in overtrained athlete syndrome—or malignant diseases.

Healthy organisms can compensate short-term catecholamine-driven stress responses of diverse origins remarkably well, provided that enzymatic functions of COMT and MAO are not genetically impaired [23]. Limits are exceeded when acute stress surpasses the compensatory capacity of a normally or genetically compromised NEIS, particularly in the presence of preexisting disease [37].

The ubiquitous presence of speed, complexity, chronic exposure, and the inability to influence unwanted exposures (e.g., EMF) is of underestimated importance. For a long time, stress research focused primarily on cortisol synthesis by the adrenal cortex, which follows a hippocampus-regulated circadian rhythm. Cortisol peaks in the early morning and declines steadily throughout the day, with a midday nadir and lower evening levels. Stress superimposes transient peaks onto this curve.

Physiological cortisol rhythms are modulated by genetic factors, sex, training status, and age, flattening with advancing age. Inflammatory intolerance reactions to environmental exposures increase cortisol secretion to suppress inflammation, thereby simultaneously intensifying the stress response. Chronic inflammatory activation exhausts adrenal function over time, resulting in reduced morning cortisol associated with morning inertia and frequently elevated evening cortisol. Consequently, nocturnal recovery of the cortisol axis is delayed or absent.

For rapid adaptation to stress and regulation of complex demands, the cortisol axis is too slow. Speed and complexity can only be managed through catecholamines, particularly noradrenaline [23,37,38]. This leads to chronic utilization of emergency response mechanisms for routine activities. Catecholamine activation requires energy comparable to that of peak athletic performance. Regulation of this response itself demands substantial energy expenditure [39], as synthesis of SAM from methionine fully dephosphorylates ATP [40] and requires folate conversion to tetrahydrofolate as well as vitamin B12 methylation [37].

This methylation process competes with the methylation of lipophilic chemicals ubiquitous in background environmental exposure, such as dioxins, furans, and PCB. When chronic inflammation due to environmental noxae coexists with chronically elevated energy expenditure, three critical problems emerge simultaneously in a deleterious manner:

Consequences of Chronic Stress and Energy Deficiency

1. Stress control and downregulation of energy expenditure fail precisely when they are most needed, due to the underlying energy deficit, as the catabolism of noradrenaline is ATP-dependent [37,40].
2. A shift toward activation of the TH2 axis occurs, associated with an increased risk of atopic manifestations, including mast cell–related disorders [13,41].
3. Catecholamine-induced activation of the TH2 axis and Treg [13] is accompanied by increased production of IL-4 and IL-10. This reduces immune tolerance and increases susceptibility to infections, particularly intracellular pathogens, through inhibition of TH1-mediated immune responses.

Oxidative Stress and Inflammatory Amplification

This situation is further aggravated by stress-induced oxidative stress [26]. While this response is protective in the short term, it increases membrane permeability. Under chronic conditions, nitric oxide production is elevated and reacts with superoxide—preferentially generated within mitochondria—to form peroxynitrite. No endogenous compound induces oxidative stress as intensely as peroxynitrite.

This process induces activation of NF-κB, which in turn increases the release of pro-inflammatory cytokines [26]. The use of antioxidants such as vitamins A, C, and E, ubiquinone/ubiquinol, selenium, and flavonoids can limit these effects. Otherwise, a cascade of declining performance develops, to which even young individuals may succumb.

The marked increase in prevalence of CFS (syptoms > 6 months) and burnout (transient) observed over the past decade is a consequence of the psychosomatic interpretation of fundamentally somatic dysfunctions within these regulatory systems [19,21,42]. In the future, it will be crucial to distinguish to what extent oxidative stress represents a physiological necessity and when it assumes a pathophysiological role that triggers or perpetuates disease [43,44].

Conclusion

Elimination of exposure and avoidance of triggering stimuli constitute the first therapeutic step in clinical environmental medicine and represent a *conditio sine qua non*. While a substantial proportion of the population continues to behave uncritically and unsuspectingly as consumers despite the escalating prevalence of environmentally induced diseases [1], even the vigilant and proactive segment increasingly fails to implement avoidance strategies to the extent desired or required.

In the post-COVID and post-vaccination era, the design of therapeutic concepts must become even more individualized and closely monitored for intended effects than previously.

Although the mechanisms underlying environmentally induced inflammatory responses triggered by chronic exposure to stressors—and their interactions with the NEIS—have been recognized and can now be well investigated in affected individuals, the various medical disciplines and stakeholders within the healthcare system have thus far failed to adequately respond to this development.

If healthcare systems are to remain financially viable without socially questionable restrictions, a fundamental strategic shift is unavoidable. The treatment of presumed health risks and the symptomatic management of largely preventable diseases have become an expanding economic market that has yet to demonstrate effective prevention or sustained therapeutic success. Current morbidity data do not suggest meaningful progress. Moreover, the mandate of preserving individual health has increasingly become a political instrument, strategically and systematically exploited through the generation of unfounded fear.

References

1. Fajloun Z, Ziad AK, Jean MS. SARS-CoV-2 or Vaccinal Spike Protein can Induce Mast Cell Activation Syndrome (MCAS). Infect Disord Drug Target. 2025; 25.
2. Ding X, Chenyu Z, Qi L, Yuhao Z, Binghong G. TRPV1 signaling in skelatal muscle. A mini review of physilological and pathological roles. Cell Calcium. 2025; 131: 103057.
3. Dörner G. Die Ontogenese des neuroendokrinen Systems als kinetischer Prozess. Nova Acta Leopoldina. 1980; 237: 279-291.
4. Müller KE. Störung der immunoneurogenen Kommunikation. Zeitsch Orthomol Med. 2008; 3: 9-13.
5. Abbas AK, Lichtman AH, Pillai S. Cellular and Molecular Immunology. 6th Edition. 2007.
6. Maes M, Frank N M Twisk. Chronic fatigue syndrome: Harvey´s and Wessely´s (bio) psychosocial model versus a bio (psychosocial) model based on oxidative and nitrosative stress pathways. BMC Med. 2010; 8: 1203-1217.
7. Andrei C, Anca Z, George MN, Octavian TO, Simona N. Natural Active Ingredients and TRPV1 Modulation: Focus on Key Chemical Moities Involved in Ligand-Target Interaction. Plants. 2023; 12: 339.
8. Morilak D, Gabe Barrera, David J Echevarria, April S Garcia, Angelica Hernandez, et al. Role of brain norepinephrine in the behavioral response to stress. Prog Neuropsychopharmacol Biol Psychatry. 2005; 29: 1214-1224.
9. Rassow J. Biochemie. Thieme Verlag, Stuttgart. 2008: 162.
10. Müller KE. Stressregulation und Mitochondrienfunktion. Zeitsch Orthomol Med. 2013; 1: 1-6.
11. Dürr F. Fatigue und Depression: Genetische und biochemische Faktoren können die Entwicklung und Ausprägung der Symptomatik beeinflussen. umwelt-medizin-gesellschaft. 2009; 22: 148-150.
12. Hänninen O, Knol A. European Perspectives on Environmental Diseases. Estimates for Nine Stressors in Six European Countries. University Printing, Helsinki. 2011.
13. Roskosky R. Biochemistry WB. Saunders Company, Philadelphia. 1996: 83-86.
14. Sternberg E, Philip W. Gold. Psyche, Stress und Krankheitsabwehr. In Güntürkün O. Biopsychology. Spektrum Akademischer Verlag, Heidelberg. 1988: 174-181.
15. Müller KE. Genetische Polymorphismen der Catechol-O-Methyltransferase (COMT). umwelt-medizin-gesellschaft. 2007; 20: 282-288.
16. Müller KE. Epigenetik und funktionelle Teratologie. umwelt-medizin-gesellschaft. 2009; 22: 9-13.
17. HillHU. Multiple Chemikalien Sensitivität. Shaker. 2020.
18. Chen J, Wenqian S, Youjia Z, Feng Z, Shuixiang D, et al. TRPV1: The key bridge in neuroimmune interactions. J Intensive Med. 2024; 4: 442-452.
19. Dantzer R, Jason CO, Marcus AL, Keith WK. Inflammation-associated depression: From serotonin to kynurenine. Psychoneuroendocrinology. 2011; 36: 426-436.
20. Dörner G. Neuroendocrinology and neuroendocrine prophylaxis. Neuroendocrin Letter. 1988; 10: 1-4.
21. Liviereo F, Manuela Campisi, Paola Mason, Sofia Pavanello. Transient Receptor Potential Vanilloid Subtyp: Potential Role in Infection, Susceptibility, Symptoms and Treatment of COVID-19. Front Med. 2021; 8: 753819.
22. Schedlowski M, Schmidt RE. Stress, Hormone und zelluläre Immunfunktion. Spektrum Akademischer Verlag. 1994; 9-30.
23. Müller KE. REACH Die neue Chemikalienbewertung in der EU. umwelt-medizin-gesellschaft. 2006; 18: 181-185.
24. Bansal M, Kaushal N. Oxidative Stress, Mechanisms and their Modulation. Springer. 2014.
25. Henz D, Kurt EM. Einflüsse von 5G auf die Immunfunktion und Veränderungen des Elektroenzephalogramm. umwelt-medizin-Geselschaft. 2022; 35: 24-27.
26. Padgett D, Ronald Glaser. How stress influences the immune response. Trends in Immunology. 2003; 24: 444-448.
27. Köberle U, Stammschulte T, Acquarone D, Bonnet U. Pregabalin. Arzeneiverordnung in der Praxis. 2020; 47: 62-65.
28. Dantzer R, Capuron L. Inflammation Associated Depression: Evidence, Mechanisms and Implications. Springer. 2017.
29. Mayer WR. MCS - eine chronische Entzündung? Zeitsch f Umweltmed. 2002; 10: 141-149.
30. Müller KE. Schädigung von Strukturen und Funktionen der Membranen durch Umwelteinflüsse. umwelt-medizin-gesellschaft. 2014; 27: 11-16.
31. https://www.federalregister.gov/documents/1994/06/28/94-15613/philadelphia-electric-company
32. Pall M. Explainig Unexplained Illnesses. Harrington Park Press; New York. 2007.
33. Holsboer F, Gerken A, Steiger A, Benkert O, Müller OA, et al. Corticotropin-releasing factor induced pituitary-adrenal responses in depression and after recovery. Lancet. 1984; 1: 55.
34. Schwarz MJ. Schizophrenie und Depression. Immunsystem im therapeutischen Visier. NeuroTransmitter. 2017; 4: 51-56.
35. Schauenstein K, Liebmann P. Regulation von Immunfunktionen durch Katecholamine. In Straub RH: Lehrbuch der klinischen Pathophysiology komplexer chronischer Erkrankungen. Vandenhoeck&Ruprecht, Göttingen. 2006: 91-102.
36. Selye H. Stress in Health and Disease. Butterworth, Boston. 1976; 3-34.
37. Müller KE. Depression bei umweltmedizinischen Erkrankungen. umwelt-medizin-gesellschaft. 2010; 23: 294-308.
38. Michalik L, Wolfgang M Kuebler. TRPV4 - A Missing Link between Mechanosensation and Immunity. Front. Immunol. 2020, 11: 413.
39. Roitt I, Brostoff J, Male D. Immunology 6th ed Edinburgh: Mosby Elsevier. 2001.

40. Rahman M, Youn-Yi Jo, Yong Ho Kim, Chul-Kyu Park. Current Insights and therapeutic strategies for targeting TRPV1 in neuropathic pain management. Life Science. 2024; 355: 122954.
41. Naviaux RK. Oxidative Shielding or Oxidative Stress. J Pharmacol Exp Ther. 2012; 342: 608-618.
42. Müller KE. Umweltstress in Wolf A, Calabrese P: Stressmedizin & Stresspsychologie. Schattauer Verlag. 2020; 280-305.
43. Baehr von V. Möglichkeiten und Grenzen der Laboranalytik – Blutdiagnostik bei Darmerkrankungen und chronischer Entzündung. umwelt-medizin-gesellschaft. 2016; 29: 19-23.
44. Myhill S, Norman E Booth, John McLaren-Howard. Chronic fatigue syndrome and mitochondrial dysfunction. Int J Clin Exp Med. 2009; 2: 1-16.