News

Not just in your head: Here's how stress triggers flare-ups of eczema



Chronic stress may cause flare-ups of eczema, a common , according to research.
It was previously unknown how stress triggers flare-ups of eczema, but a study from Shanghai, China's Fudan University has now thoroughly examined the biochemical relationships.

It discovered a particular network of neurons that, when under stress, cause an immunological response in the skin that results in flare-ups of eczema.
An estimated 16.5 million persons in the US alone suffer with atopic dermatitis, commonly referred to as eczema. It probably affects up to 101.27 million adults worldwide, if not more.

The development of roughened, scaly, or discolored skin patches, as well as persistent, occasionally severe itching, are common symptoms. Excessive or frequent scratching can

worsen symptoms and result in additional skin infections.
Using barrier-repair moisturizers and using medicine, such as antibiotics or antihistamines, as directed on a case-by-case basis, are two methods of managing eczema.

Nevertheless, there is presently no treatment for this skin ailment, and its symptoms might fluctuate in intensity.

"Flare-ups" are symptom recurrences that, depending on their intensity, might seriously interfere with day-to-day activities and cause distress.

Flare-ups can be caused by a variety of reasons, but a National Eczema Society study revealed that many people with this skin condition believed that "stress was the single biggest trigger of their eczema flare-ups," which is in line with results from focus-group-based research.

Lawread

Researchers from Shanghai, China's Fudan University recently conducted a study that explored the possible processes behind the connection between flare-ups and stress.

The study found a brain network that, when triggered by stress, further activates an immunological response that results in flare-ups of eczema. The findings are published in the journal Science.

The study's "whys"
Shenbin Liu, PhD, a neurobiologist at Fudan University and co-author of the study, told Medical News Today that after learning from dermatologists about the substantial effects eczema has on its sufferers, he and his colleagues made the decision to investigate the connection between stress and eczema further.

Understanding how the brain interacts with peripheral organs is one of our lab's goals. The skin, which serves as the body's main barrier organ and is rich in immune cells and nerve fibers, is extremely vulnerable to the effects of our emotions, according to Liu.

For this study, Liu and his colleagues recruited 51 participants who had been diagnosed with atopic dermatitis. They measured the participants' stress levels using the Perceived Stress Scale (PSS), the severity of skin inflammation using the SCORing Atopic Dermatitis (SCORAD) method, and the Numeric Rating Scale (NRS).

Additionally, they examined blood and skin samples from every research subject.

Researchers discovered that during eczema flare-ups, those with high stress levels also had more severe skin inflammation than those with low stress levels.

Eosinophils, a type of white blood cell that is crucial to immunological responses, were also more prevalent in those who had high stress levels.

How skin irritation is exacerbated by stress

The researchers verified that these symptoms significantly exacerbated under stress using a mouse model of eczema that acquired symptoms such skin redness, itching, and inflammation.

The researchers discovered that skin samples from mice who had been under a lot of stress had four times as many eosinophils as samples from mice that had not.

The research team then examined cutaneous nerve cells and found a particular subset that was activated in response to psychological stress.

The central nervous system (CNS) sent stress signals to these neural cells, which caused the production of certain inflammatory proteins. This quadrupled the number of eosinophils in the skin, causing inflammation.

Liu used the following to demonstrate this mechanism:

The researcher claims that "it requires repeated stress challenges" to see this process in action, implying that the main cause of eczema flare-ups may be chronic stress, or stress that is continuously experienced over an extended period of time.

What we now know about stress and eczema

"One of [its] most striking findings is the specificity of the pathway involved," said Susan Mayou, MBBS, FRCP, a consultant dermatologist at the Cadogan Clinic in London, UK, who was not involved in this study.

Mayou found it noteworthy that "the traditional stress pathway was not the primary driver" of eczema flare-ups and that "the study shows that only a particular subset of sympathetic neurons (Pdyn+) drives stress-related inflammation, rather than a broad, generalized stress response."

Lawread

Peripheral nerves, on the other hand, were more important. This calls into question long-held beliefs regarding the relationship between inflammatory disorders and stress, she informed us.

"In addition, the fact that eosinophils by themselves do not cause eczema but become extremely problematic in the context of stress helps explain why some immune-targeting treatments have shown inconsistent results in clinical practice," Mayou added.

Additionally not participating in this study, Tanya Evans, MD, a board-certified dermatologist and medical director of the Skin Cancer Program at the Melanoma Clinic at MemorialCare Saddleback Medical Center in Laguna Hills, CA, concurred.

According to Evans, "previously, stress was thought to worsen eczema through broad systemic pathways like the hypothalamic-pituitary-adrenal (HPA) axis." "This study challenges that perspective," she stated.

She was surprised that "blocking the adrenal (HPA) pathway worsened inflammation, suggesting it may be protective rather than harmful in this context," in addition to the route's specificity.

Like Mayou, she mentioned the "eosinophil paradox," which seems to explain why some eczema therapies haven't worked.

Evans pointed out that the results also imply that "stress is not just a trigger — it is biologically wired into the skin's immune response."

What do these findings mean?

Liu states that "the significance of [the current] findings lies in their transformative impact on our understanding of the mind-skin connection, moving the explanation for stress-induced flare-ups of atopic dermatitis from a vague psychological concept to a concrete, physical pathway that delineates exactly how a feeling, such as psychological stress, can translate into a biological event, namely inflamed skin, thereby revealing a direct mind-body circuit."

Additionally, he stated, "our work points to a potential new biomarker, as the strong link between stress and elevated eosinophils suggests that clinicians might one day measure a patient's eosinophil levels not only to assess disease severity but also to gauge the specific contribution of stress to a current flare-up."

Lawread

However, the researcher stressed that the primary objective is to "open the door for novel, targeted therapeutic strategies, including interventions designed to block the stress signal in specific nerves, inhibit the release of the CCL11 chemokine, or prevent eosinophil recruitment and activation."

The study "significantly advances our understanding of eczema by identifying a direct biological pathway linking psychological stress to skin inflammation," Mayou added.

The study "moves the narrative beyond a vague 'mind–skin connection' to a precise neuro-immune mechanism," she said.

Additionally, the dermatologist was optimistic that the recently discovered data could guide future studies on more effective, "more targeted" eczema therapies.

Where should eczema research go next?

Liu told MNT, "I believe that future research should proceed on multiple fronts, encompassing both translational and basic science investigations." "Confirming that the same Pdyn+ sympathetic neuron-CCL11-eosinophil axis functions in human skin would be the most immediate next step."

He went on, "Studies should look into whether stress-induced eosinophilia or elevated CCL11 levels in blood or skin could serve as useful biomarkers to identify patients whose disease is particularly stress-responsive, thereby enabling personalized treatment approaches."

Liu added that there might be room to repurpose current drugs for eczema therapy with different indications.

Researchers should investigate the possibility of reformulating or repurposing current medications that reduce hyperactivity, like gabapentinoids or local anesthetics, to precisely target Pdyn+ sympathetic neurons.

Shenbin Liu, Ph.D.

To completely comprehend the mechanisms at play and find new therapeutic avenues, the researcher further underlined that "a deeper understanding of Pdyn+ neurons themselves is also essential."

Does stress have a comparable effect on rosacea and psoriasis?

Lawread

"More research is necessary to determine the wider significance of this pathway [involved in eczema activation under stress]," Liu continued. "Does the same mechanism operate in other stress-sensitive dermatological conditions like psoriasis, rosacea, or urticaria?" he pondered."

Additionally, Evans expressed interest in "whether similar neuroimmune pathways exist in psoriasis, chronic urticaria, [or] prurigo nodularis," the latter of which frequently co-occurs with eczema.

Liu concluded by emphasizing that the research team's present findings from studies in mice models of eczema need be confirmed by more human research, a point that Evans and Mayou also underlined.

"Translating these results from animal models into human clinical studies is the crucial next step." Mayou informed us, "We need to confirm whether this same neuro-immune pathway functions in eczema patients and how strongly it influences disease severity."

Including psychological interventions in treatment paradigms is equally crucial. Future studies should look into whether stress-reduction techniques like mindfulness, cognitive behavioral therapy, or sleep optimization can quantitatively lower inflammation through this pathway.

"Eczema is not just skin-deep": Professional management advice

For the time being, individuals with eczema must continue to investigate the various treatment options that are most effective for them individually, with the assistance of their reliable medical specialists.

Mayou said the study's findings "reinforce the importance of managing both the skin and the nervous system" in this skin condition.

She claims that some useful tactics in line with this strategy are as follows:

putting skin barrier restoration first by avoiding harsh soaps and using emollient creams on a daily basis, which "helps reduce baseline inflammation and vulnerability to flare-ups."

recognizing and reducing "common factors such as allergens, temperature changes, and [skin] irritants" as eczema triggers.

adopting a proactive approach to stress management, where "improving sleep quality, breathing exercises, and mindfulness can help reduce flare frequency,"

Simple actions like "keeping nails short, using anti-itch treatments, and applying cold

Lawread

compresses" can disrupt "the itch–scratch cycle."
Lastly, it is important to treat symptoms as soon as possible since "prompt use of prescribed topical therapies during early flare signs can prevent escalation."
Evans concurred with Mayou's conclusion that "eczema management should be holistic, addressing both physical and psychological drivers, as they are more interconnected than previously understood."

Evans emphasized that dermatologists have a responsibility to "help patients understand [that] your eczema is not just skin-deep — your nervous system and immune system are working together," adding that "this study reinforces a multidimensional approach, not just skin-directed therapy." The researcher stated that "it requires repeated stress challenges" to observe this mechanism in action, implying that chronic stress—stress experienced consistently over a long period of time—may be the biggest culprit in eczema flare-ups.

What we now know about stress and eczema

"One of [its] most striking findings is the specificity of the pathway involved," said Susan Mayou, MBBS, FRCP, a consultant dermatologist at the Cadogan Clinic in London, UK, who was not involved in this study.

Mayou found it noteworthy that "the traditional stress pathway was not the primary driver" of eczema flare-ups and that "the study shows that only a particular subset of sympathetic neurons (Pdyn+) drives stress-related inflammation, rather than a broad, generalized stress response."

Peripheral nerves, on the other hand, were more important. This calls into question long-held beliefs regarding the relationship between inflammatory disorders and stress, she informed us.

"In addition, the fact that eosinophils by themselves do not cause eczema but become extremely problematic in the context of stress helps explain why some immune-targeting treatments have shown inconsistent results in clinical practice," Mayou added.

Additionally not participating in this study, Tanya Evans, MD, a board-certified dermatologist and medical director of the Skin Cancer Program at the Melanoma Clinic at MemorialCare Saddleback Medical Center in Laguna Hills, CA, concurred.

According to Evans, "previously, stress was thought to worsen eczema through broad

Lawread

systemic pathways like the hypothalamic-pituitary-adrenal (HPA) axis." "This study challenges that perspective," she stated.

She was surprised that "blocking the adrenal (HPA) pathway worsened inflammation, suggesting it may be protective rather than harmful in this context," in addition to the route's specificity.

Like Mayou, she mentioned the "eosinophil paradox," which seems to explain why some eczema therapies haven't worked.

Evans pointed out that the results also imply that "stress is not just a trigger — it is biologically wired into the skin's immune response."

What do these findings mean?

Liu states that "the significance of [the current] findings lies in their transformative impact on our understanding of the mind-skin connection, moving the explanation for stress-induced flare-ups of atopic dermatitis from a vague psychological concept to a concrete, physical pathway that delineates exactly how a feeling, such as psychological stress, can translate into a biological event, namely inflamed skin, thereby revealing a direct mind-body circuit."

Additionally, he stated, "our work points to a potential new biomarker, as the strong link between stress and elevated eosinophils suggests that clinicians might one day measure a patient's eosinophil levels not only to assess disease severity but also to gauge the specific contribution of stress to a current flare-up."

However, the researcher stressed that the primary objective is to "open the door for novel, targeted therapeutic strategies, including interventions designed to block the stress signal in specific nerves, inhibit the release of the CCL11 chemokine, or prevent eosinophil recruitment and activation."

The study "significantly advances our understanding of eczema by identifying a direct biological pathway linking psychological stress to skin inflammation," Mayou added.

The study "moves the narrative beyond a vague 'mind–skin connection' to a precise neuro-immune mechanism," she said.

Lawread

Additionally, the dermatologist was optimistic that the recently discovered data could guide future studies on more effective, "more targeted" eczema therapies.

Where should eczema research go next?

Liu told MNT, "I believe that future research should proceed on multiple fronts, encompassing both translational and basic science investigations." "Confirming that the same Pdyn+ sympathetic neuron-CCL11-eosinophil axis functions in human skin would be the most immediate next step."

He went on, "Studies should look into whether stress-induced eosinophilia or elevated CCL11 levels in blood or skin could serve as useful biomarkers to identify patients whose disease is particularly stress-responsive, thereby enabling personalized treatment approaches."

Liu added that there might be room to repurpose current drugs for eczema therapy with different indications.

Lawread

Researchers should investigate the possibility of reformulating or repurposing current medications that reduce hyperactivity, like gabapentinoids or local anesthetics, to precisely target Pdyn+ sympathetic neurons.

To completely comprehend the mechanisms at play and find new therapeutic avenues, the researcher further underlined that "a deeper understanding of Pdyn+ neurons themselves is also essential."

Does stress have a comparable effect on rosacea and psoriasis?

"More research is necessary to determine the wider significance of this pathway [involved in eczema activation under stress]," Liu continued. "Does the same mechanism operate in other stress-sensitive dermatological conditions like psoriasis, rosacea, or urticaria?" he pondered."

Additionally, Evans expressed interest in "whether similar neuroimmune pathways exist in psoriasis, chronic urticaria, [or] prurigo nodularis," the latter of which frequently co-occurs with eczema.

Liu concluded by emphasizing that the research team's present findings from studies in mice models of eczema need be confirmed by more human research, a point that Evans and Mayou also underlined.

"Translating these results from animal models into human clinical studies is the crucial next step." Mayou informed us, "We need to confirm whether this same neuro-immune pathway functions in eczema patients and how strongly it influences disease severity."

Including psychological interventions in treatment paradigms is equally crucial. Future studies should look into whether stress-reduction techniques like mindfulness, cognitive behavioral therapy, or sleep optimization can quantitatively lower inflammation through this pathway.

"Eczema is not just skin-deep": Professional management advice

For the time being, individuals with eczema must continue to investigate the various treatment options that are most effective for them individually, with the assistance of their reliable medical specialists.

Mayou said the study's findings "reinforce the importance of managing both the skin and the nervous system" in this skin condition.

She claims that some useful tactics in line with this strategy include

putting skin barrier restoration first by avoiding harsh soaps and using emollient creams on a daily basis, which "helps reduce baseline inflammation and vulnerability to flare-ups."

recognizing and reducing "common factors such as allergens, temperature changes, and [skin] irritants" as eczema triggers.

Lawread