Turn Therapeutics Announces New Peer-Reviewed Research Showing GX-03 Selectively Reduces Key Drivers of Skin Inflammation and Itch

August 20, 2026

In-vivo study showed GX-03 reduced IL-36, a key early driver of the inflammatory cascade, by approximately 50%, IL-31, a major driver of itch, by nearly 68%, and IL-4, an important inflammatory cytokine, by 16.7%

Findings support Turn's approach of acting earlier in the inflammatory process, before inflammation becomes fully established

WESTLAKE VILLAGE, Calif.--(BUSINESS WIRE)--Aug. 20, 2026-- Turn Therapeutics Inc. (Nasdaq: TTRX), a clinical-stage biotechnology company developing localized therapies for inflammatory and infectious skin diseases, today announced the publication of new peer-reviewed research providing in-vivo evidence that its lead investigational therapy, GX-03, selectively reduces several important inflammatory signals associated with inflammatory skin disease.

The study, published in *Life*, showed that pretreatment with GX-03 reduced IL-36α and IL-36γ release by approximately 50%. IL-36 is an important early signal in the inflammatory cascade and helps amplify inflammation in the skin. GX-03 also reduced IL-31 by nearly 68%. IL-31 is closely associated with itch in inflammatory skin diseases such as atopic dermatitis. In addition, IL-4, another important inflammatory cytokine involved in the immune response, was reduced by 16.7%. Importantly, IL-13 was essentially unchanged, suggesting that GX-03 did not suppress every inflammatory signal measured, but instead produced a selective pattern of cytokine reduction. These biological changes were accompanied by a 66% reduction in clinical dermatitis severity in GX-03-pretreated animals compared with untreated controls.

"This publication provides important new biological evidence about GX-03," said Bradley Burnam, Chief Executive Officer of Turn Therapeutics. "In-vivo, GX-03 strongly reduced IL-36, a key early driver of the inflammatory cascade, as well as IL-31, a major driver of itch. It also reduced IL-4, an important inflammatory signal. What is particularly interesting is when this occurred. Rather than targeting inflammation only after it is already established, GX-03 was present before the challenge and substantially reduced the inflammatory response that followed skin challenge. We believe this supports a fundamentally different approach: modifying the local skin environment upstream, before the inflammatory cascade takes hold."

The timing of the intervention is an important part of the study. Many current therapies for atopic dermatitis are designed to block inflammatory pathways after disease and inflammation are already established. In this study, researchers applied GX-03 before exposing the skin to a standardized bacterial challenge known to trigger atopic dermatitis-like inflammation. They then measured the inflammatory response that developed afterward. Animals pretreated with GX-03 developed substantially less severe dermatitis and showed significant reductions in IL-36α, IL-36γ, IL-31 and IL-4. The study therefore provides a different experimental perspective on inflammatory skin disease by examining whether modifying the skin environment can change the intensity and pattern of the inflammatory response.

The study evaluated 40 mice that received either no pretreatment or four days of topical GX-03 before standardized *Staphylococcus aureus* exposure. Following predefined exclusions, 18 animals in each group were included in the final analysis. GX-03 pretreatment reduced mean clinical disease severity from a vIGA-AD score of 2.44 in untreated animals to 0.83, representing a 66.0% reduction. IL-36α expression decreased by 49.9%, IL-36γ by 50.9%, IL-31 by 67.7%, and IL-4 by 16.7%. IL-13 did not differ significantly between the groups. No local irritation, dryness, redness, barrier disruption or other adverse skin findings were observed during the GX-03 pretreatment period.

The publication, titled "Effects of Extended-Release Polyhexanide on Inflammatory Cytokine Expression and Disease Severity Following *Staphylococcus aureus* Exposure in a Murine Dermatitis Model," was co-authored by Bradley Burnam, Chief Executive Officer of Turn Therapeutics, and Stephen D. Bresnick, M.D., an independent researcher. The study was conducted at Altogen Labs under an IACUC-approved protocol and builds upon Turn Therapeutics' previously published research in the *Journal of Dermatological Treatment*.

About GX-03

GX-03 is Turn Therapeutics' proprietary, non-systemic topical investigational therapy currently in clinical development for atopic dermatitis. GX-03 is designed to act locally in the skin, where Turn is investigating whether modifying the local skin environment can reduce important inflammatory signals associated with disease while avoiding broad systemic immune suppression. The newly published findings provide in-vivo evidence that GX-03 is associated with selective reductions in IL-36, IL-31 and IL-4 following a standardized skin challenge. GX-03 is currently being evaluated in a randomized, double-blind, vehicle-controlled clinical trial in atopic dermatitis.

About Turn Therapeutics

Turn Therapeutics is a clinical-stage biotechnology company focused on developing targeted, localized therapies for inflammatory and infectious skin diseases. GX-03 is Turn Therapeutics' lead investigational topical candidate and is being developed as a targeted, non-systemic treatment for atopic dermatitis.

Citation

Burnam B, Bresnick S. Effects of Extended-Release Polyhexanide on Inflammatory Cytokine Expression and Disease Severity Following *Staphylococcus aureus* Exposure in a Murine Dermatitis Model. *Life*. 2026. DOI: 10.3390/life16081369

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements, other than statements of historical fact, contained in this press release are forward-looking statements, including statements regarding clinical development plans, optimization of enrollment criteria and endpoints, interpretation of interim clinical observations, expected trial timing, regulatory interactions, and the therapeutic potential of GX-03. Forward-looking statements contained in this press release may be identified by the use of words such as "anticipate," "believe," "contemplate," "could," "estimate," "expect," "intend," "seek," "may," "might," "plan," "potential," "predict," "project," "suggest," "target," "aim," "should," "will," "would," or the negative of these words or other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements are based on Turn's current expectations and are subject to inherent uncertainties, risks, and assumptions that are difficult to predict, including risks related to the success of development programs, the availability of additional financing, and the Company's ability to execute its strategic plan. Further, certain forward-looking statements are based on assumptions as to future events that may not prove to be accurate. For a further discussion of risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to the business of Turn Therapeutics in general, see the risk disclosures in the Company's filings with the SEC. All such forward-looking statements speak only as of the date they are made, and Turn undertakes no obligation to update or revise these statements, whether as a result of new information, future events, or otherwise.

View source version on businesswire.com: https://www.businesswire.com/news/home/20260820905760/en/

Turn Therapeutics
Investor & Media Contact
Sasha Damouni
The Damouni Group
Sasha@turntherapeutics.com

Source: Turn Therapeutics Inc.