First Results from the 2025 FPRF Scientific Symposium

Multiple Research Projects in TN and Other Facial Pain Were Reviewed

The foundation will be distributing summaries of many studies presented at the 2025 symposium. This email will highlight the presentation by Dr Kim Burchiel reviewing the main findings from the Foundation's study on the:

Genetics of Trigeminal Neuralgia.

The genetic study had two components. In the larger analysis, we compared 781 people with Trigeminal Neuralgia type 1 (TN1)—most of whom had paroxysmal (sudden, brief) pain—to 1,015 control participants. Within the TN1 group, we also looked at people with and without neurovascular compression (NVC), where a blood vessel presses on the trigeminal nerve.

What we found

We identified eight genetic variants that were significantly more common in people with TN1 than in controls. The strongest association was with a gene called LRP1B, which is well studied and **has functions that make biological sense for TN**:

- **Myelin support and repair**: LRP1B is involved in the development and maintenance of myelin, the insulating layer that helps nerves transmit signals properly. It also has roles linked to Schwann cells, the cells that produce myelin in the peripheral nervous system. Myelin disruption has long been considered a contributor to TN, so a variant in a myelin-related gene provides a clear, intuitive connection.
- **Pain modulation**: LRP1B is also implicated in dampening neuropathic pain, which further increases its potential relevance to TN.

We also found associations involving CHL1, LINC00290, CUX1, SGMS1, ZNF804B, and KCNK10. These genes are all linked in various ways to nervous system biology, but how—or whether—they contribute directly to TN is not yet clear. More work is needed to understand the proteins these genes encode (or regulate) and what those proteins do in trigeminal nerve pathways.

What this suggests about the causes of TN

Overall, the results support a view many clinicians already suspect: trigeminal neuralgia is not caused by one single mechanism.

- Genetic variation appears to contribute meaningfully to TN risk.
- Neurovascular compression is important, but not required—people can develop TN even without NVC.
- What we call "TN" is likely a shared symptom pattern that can arise from more than one underlying condition.

Possible TN subtypes

The genetic data point to different TN subgroups that share similar symptoms but may differ biologically, including:

- Older patients with NVC

- Younger female patients
- Patients without NVC
- Patients with multiple sclerosis (MS) who develop TN

One illustrative example: TN1 patients with NVC showed strong associations with five variants, while TN1 patients without NVC had associations with two additional variants—suggesting partially distinct genetic drivers depending on whether compression is present.

Big picture takeaway

These findings imply that TN1 risk may be strongly shaped by rare variants in key neural genes, creating a genetic predisposition that interacts with other factors—such as age, sex, and NVC status—to determine who develops TN and what subtype they have.

If you would like to support future research in facial pain therapeutic development, feel free to visit our donation page on our website!

Click to Donate

The Facial Pain Research Foundation | 2653 SW 87th Drive, Suite A | Gainesville, FL 32608-9313 US

Unsubscribe | Update Profile | Constant Contact Data Notice