ASX Announcement **11 November 2026**

BlinkLab Reports Positive Results in Genetic Autism Study, Ahead of Schedule

Highlights

- **Strong multi-domain biomarker signal in SHANK2-related autism:** Individuals with a SHANK2 mutation (n = 8) showed significant differences in BlinkLab smartphone-tests on sensorimotor gating, startle habituation and spontaneous sensorimotor behaviour compared with matched community participants (n = 99), unaffected siblings (n = 10), or both.
- **Biological validation of BlinkLab's platform:** SHANK2-related disorder is a genetically defined condition associated with autism. BlinkLab Dx1 measured differences across objective neurophysiological domains, supporting the view that our technology captures underlying brain biology rather than behavioural variation alone.
- **Expansion into therapeutic development:** Objective, scalable measures of sensory gating and response dynamics may have future relevance as digital biomarkers and exploratory clinical trial endpoints in genetically defined neurodevelopmental disorders.
- **Dx1 regulatory program on track:** The study used the same core smartphone-based neurometric technology that underpins BlinkLab Dx1 and does not change the Company's FDA 510(k) pivotal program.
- **More study readouts later this year:** The current SHANK2 announcement marks the first in a series of upcoming readouts from the Company's exploratory research programs. BlinkLab continues to make progress across all these programs and remains on track to announce further study results before the end of the year.

BlinkLab Limited (ASX:BB1) ("BlinkLab" or the "Company") is pleased to report findings from its exploratory study of SHANK2-related neurodevelopmental disorder, conducted in collaboration with the SHANK2 Foundation.

The collaboration was announced on 13 March 2026 in (BlinkLab Forms Partnership with SHANK2 Autism Foundation in the United States)[1]. The study was subsequently included in BlinkLab's Company Update on Clinical Development Programs released on 14 July 2026, which identified Q1 2027 as the expected readout[2]. Completion of the analysis enables BlinkLab to report these findings earlier than anticipated.

[1] ASX Announcement (13 March 2026) - "BlinkLab Forms Partnership with SHANK2 Autism Foundation in the United States"

[2] ASX Announcement (14 July 2026) - "Company Update on Clinical Development Programs Beyond Paediatric Autism"

About SHANK2 Related Disorder

SHANK2 is a gene that encodes a scaffolding protein involved in excitatory synapses. Rare pathogenic variants in SHANK2 are associated with neurodevelopmental conditions including autism, intellectual disability, and ADHD[3]. Because the condition is rare and affected families are geographically dispersed, coordinated recruitment through patient organisations and specialist research centres is important. The SHANK2 Foundation is a non-profit rare-disease advocacy and research-enablement organisation that supports families, researchers, clinicians and industry partners, including through research infrastructure and patient data initiatives.

Study Design

Twenty participants were recruited through the SHANK2 Foundation: nine individuals with a SHANK2 mutation and eleven unaffected siblings. This represents a unique cohort for a rare and geographically dispersed genetic condition. For these rare conditions, decentralized study designs are particularly important and smartphone-based assessments[4] enabled study participants to be recruited across the US, Australia and Europe. Eight individuals with a SHANK2 mutation and ten siblings completed the 15-minute smartphone assessment; two participants were unable to complete testing because of discomfort with the headphones or auditory stimuli. The completed groups did not differ significantly in age or sex. SHANK2-related disorder is very rare, so cohorts are necessarily small. Nonetheless, the observed group differences reached statistical significance.

The study was further strengthened by a matched community comparison group of 99 participants who completed the same experimental protocol. Participants were matched on sex, race and age within two years where an eligible match was available. This design enabled the rare SHANK2 cohort to be evaluated against both unaffected siblings and a substantial community benchmark.

The assessment measured acoustic startle response, prepulse inhibition, startle habituation and spontaneous sensorimotor behaviour using computer vision analysis of the smartphone camera feed. The study used the same core smartphone-based neurometric technology that underpins BlinkLab Dx1, deployed in a research setting.

Results

Against the sibling group and the matched community comparison group, the study identified a multi-domain neurophysiological and sensorimotor signature in the SHANK2 group. In summary, BlinkLab's smartphone startle test detects a clear, consistent difference between subjects with a SHANK2 mutation and the comparison groups. Matched community participants and siblings both show normal filtering of sound (+27.2% and +27.8%). The SHANK2 group shows the reverse (-26.5%), a gap of about 54 percentage points (group effect $p = 0.020$; $p = 0.0001$ after adjustment for age and sex). The size of the acoustic startle response itself did not differ between groups ($p = 0.55$; $p = 0.61$ after adjustment for age and sex). These new SHANK2 findings emphasize the critical need

[3] Berkel et al. (2010), *Nature Genetics;* Won et al., (2012), *Nature*

[4] Boele at. (2023), *Scientific Reports*

to evaluate how sensorimotor responses adapt over time and are gated, rather than overall responsiveness.[5]

Startle habituation also differed between groups. With sounds at random intervals, the SHANK2 group showed greater habituation than matched community participants ($p < 0.001$) but not siblings ($p = 0.208$). With sounds at rhythmic intervals, the SHANK2 group showed greater habituation than both matched community participants ($p = 0.004$) and siblings ($p = 0.021$). For spontaneous sensorimotor behaviour, the SHANK2 group differed from siblings in postural stability, head rotations and mouth openings (p-Holm = 0.009, 0.010 and 0.010) and showed more mouth openings than matched community participants (p-Holm = 0.033). The main sensory gating finding is illustrated in **Figure 1**, and all results are summarised in **Table 1.**

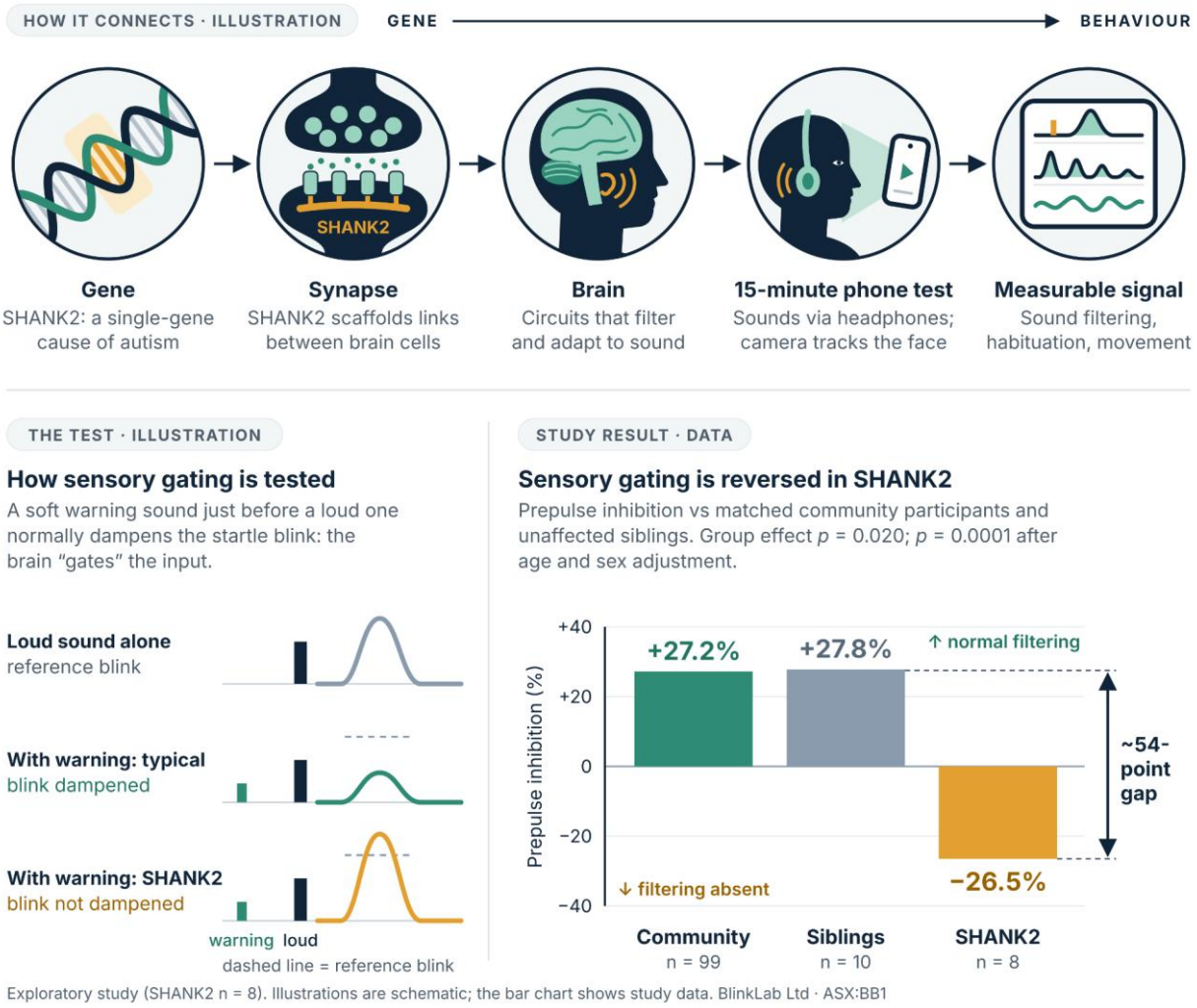


Figure 1 | From gene to smartphone measurement, and the sensory gating result. The top row and the test diagram are illustrations; the bar chart shows study data.

[5] Gultig et al. (2026), *Autism Research*

A Measurable Biomarker Signature in a Monogenic Form of Autism

The SHANK2 result is important because it links a known genetic cause of autism with objective neurophysiological differences measured through BlinkLab's smartphone-based platform. Most autism is biologically heterogeneous, reflecting the combined influence of many genetic and environmental factors[6]. The SHANK2 mutation provides a more biologically defined model that enables us to study the pathways that contribute to autism and related neurodevelopmental conditions.

The findings show measurable differences across sensory gating, response habituation and spontaneous sensorimotor behaviour (**Table 1** below). These domains are consistent with the sensory-processing and movement differences identified in BlinkLab's broader autism research[7], while also illustrating that the most informative biomarker may be a combination of signals rather than any single measurement.

STUDY RESULTS · DATA

Where the SHANK2 group differed on BlinkLab's smartphone test SHANK2 n = 8 · Community n = 99 · Siblings n = 10

Measure	What we found	Overall group effect	vs matched community	vs unaffected siblings
SENSORY GATING				
Prepulse inhibition sound filtering	Reversed: −26.5% in SHANK2 vs +27.2% community, +27.8% siblings	✓ **p = 0.020** p = 0.0001 adj.	—	—
RESPONSE ADAPTATION				
Startle habituation random intervals	Greater habituation than community; not vs siblings	✓ **p < 0.001**	✓ **p < 0.001**	○ p = 0.208
Startle habituation rhythmic intervals	Greater habituation than both comparison groups	✓ **p = 0.014**	✓ **p = 0.004**	✓ **p = 0.021**
SPONTANEOUS BEHAVIOUR				
Postural stability	Differs from siblings	✓ **p = 0.003**	—	✓ **p = 0.009**
Head rotations	Differs from siblings	✓ **p = 0.002**	—	✓ **p = 0.010**
Mouth openings	More than community; differs from siblings	✓ **p = 0.024**	✓ **p = 0.033**	✓ **p = 0.010**
STARTLE REACTIVITY				
Acoustic startle response size of the startle blink	Normal: no group difference	○ p = 0.20 p = 0.24 adj.	—	—

✓ Statistically significant ○ Not significant — Pairwise result not reported adj. = adjusted for age and sex

Spontaneous-behaviour p-values are Holm-corrected and adjusted for age and sex. Habituation showed no significant age or sex effects, so is unadjusted. Exploratory study in a rare genetic condition; group sizes are small. BlinkLab Ltd · ASX:BB1

Table 1 | Summary of results by measure and comparison group. Ticks mark statistically significant differences.

[6] Bölte et al. (2018), *Cellular and Molecular Life Sciences;* Willsey et al. (2022), *Nature Reviews Neuroscience*

[7] Gultig et al. (2026), *Autism Research*

As illustrated in **Figure 1**, the biological pathway can be described from gene to smartphone measurement: SHANK2 contributes to the structure and function of synapses; when its function is altered, sensory-processing circuits may develop and operate differently; and those differences may be expressed through altered sensorimotor gating, response adaptation and movement-related behaviour, which BlinkLab quantifies through a standard smartphone assessment. Rare genetic forms of common conditions can provide particularly informative models because the underlying biological cause is known. In other areas of medicine, genetically defined populations have helped validate disease mechanisms, identify biomarkers and guide the development of targeted therapies. The SHANK2 program applies the same principle to neurodevelopmental research.

BlinkLab Non-Executive Director, Prof. Raphael Bernier, commented:

"The importance of studying Shank2 and other genetic forms of autism, along with their associated neurological effects that BlinkLab captures, should not be understated. This approach has the power to identify treatment biomarkers essential to clinical trials, can inform the discovery of novel therapeutic targets, and holds the promise for the realization of precision medicine for autism."

BlinkLab Co-Founder, Managing Director and CEO, Dr Henk-Jan Boele, commented:

"Based on previous clinical and preclinical studies, including my own research at Erasmus and Princeton, we anticipated seeing a strong biological signal in this population using the BlinkLab platform. More specifically, we predicted a distinct signature across sensorimotor gating, habituation and movement. This is exactly what we now observe in our smartphone-based data in SHANK2.

It is important to understand that this study is not about the immediate market size of SHANK2-related autism (it's a rare condition). It is about a much larger scientific and commercial opportunity: understanding how specific genetic mutations shape behaviour, including behaviours associated with autism, and ultimately translating these insights into new therapeutic approaches.

What makes this particularly exciting is that SHANK mutations are also studied in genetically defined animal models (in many labs, including my own at Erasmus and Princeton), allowing us to investigate similar sensorimotor mechanisms across humans and animals. This creates a unique opportunity to evaluate potential pharmaceutical interventions in preclinical models and subsequently measure treatment responses in humans using comparable objective endpoints. We believe this could be particularly attractive to pharmaceutical companies developing targeted therapies for neurodevelopmental conditions and that we are well positioned to provide the objective measurements needed to support their development and evaluate treatment effects.

We are deeply grateful to the participating individuals and families and look forward to expanding this work together with the SHANK2 Autism Foundation. A special thanks to Ben Hutz for his dedication and support in making this study possible."

BlinkLab Clinical Research Lead on the SHANK2 autism project, Kayleigh Gultig, commented:

"This study is an important step in the search for SHANK2 biomarkers. Reliable outcome measures are prerequisites for future clinical trials of pharmacological treatments. This was an important point of discussion during the SHANK2 Day Conference. The sensorimotor differences we observed in SHANK2 show that BlinkLab could provide such a measure. It was mentioned during the conference that outcome measures are particularly important during Phase 2 clinical trials where the choice of endpoint can determine whether or not a potentially promising treatment progresses to the next phase or is abandoned. By providing objective and reliable measures that families, all over the world, can collect at home, we can help ensure potential treatments are judged on their true effect."

Implications For Diagnosis

Autism is still diagnosed primarily through specialist observation and interviews. BlinkLab Dx1 is being developed to add objective, reflex-based measures to clinical assessment. The SHANK2 findings support the biological relevance of the underlying neurometric technology by showing that it can detect measurable differences in a genetically defined neurodevelopmental condition. The study is exploratory and does not itself establish the diagnostic performance of Dx1.

Patient Stratification and Personalised Medicine

Autism comprises multiple biological subtypes with different genetic and neurodevelopmental causes. A platform capable of measuring several signals at once, including sensory gating, habituation and movement, could help researchers characterise these subtypes and evaluate whether different groups show distinct biomarker profiles. This may support future approaches to patient stratification and more personalised research and care.

Drug Development

Clinical development in autism and rare neurodevelopmental disorders is limited by the availability of objective, scalable endpoints. Trials often rely on caregiver- and clinician-rated measures that can vary between assessments and may be affected by placebo response. Drug developers need measures that are biologically relevant, repeatable and practical to collect across sites or in the home.

The SHANK2 findings support further evaluation of BlinkLab's platform for this purpose. Sensorimotor gating and startle habituation are translational measures that can be studied in both preclinical models and humans, creating potential continuity from laboratory research to clinical trials. Additional prospective work is required before these measures could be used as validated clinical trial endpoints or treatment-response biomarkers.

Investment Significance and Next Steps

- **Supports the Dx1 investment case:** The findings provide additional evidence that BlinkLab's core technology measures biologically relevant neurophysiological features as Dx1 advances through its FDA 510(k) pivotal program, while remaining separate from the regulatory evidence for Dx1.
- **Opens a potential research and pharmacological intervention market:** Beyond diagnostic testing, objective biomarker services may support research collaborations and, subject to further validation, future clinical and pharmacological trials for genetically defined neurodevelopmental disorders.
- **Reinforces BlinkLab as a platform company:** A common smartphone assessment and software platform can be applied across clinical diagnosis, research into biological subtypes and therapeutic development.
- **Capital-efficient rare-disease research model:** The study combined BlinkLab's existing technology, recruitment through a patient advocacy organisation and academic collaborator, and a substantial matched community comparison group. This provides a scalable model for evaluating other rare genetic conditions where patient cohorts are necessarily limited.

This announcement is intended to lift the Company's Trading Halt, applied for and granted on Monday 9 November 2026.

This announcement has been authorised for release by the Board of BlinkLab Limited.

For Further Information:

Dr Henk-Jan Boele
Managing Director & CEO
henkjan@blinklab.org
M: +31 (0) 611 132 247

Brian Leedman
Non-Executive Chairman
brian@blinklab.org
M: +61 (0) 412 281 780

About BlinkLab Limited

BlinkLab Limited was founded by neuroscientists at Princeton University and is developing a smartphone-based diagnostic platform for autism. Its most advanced product, BlinkLab Dx1, is an autism diagnostic aid for clinicians that leverages smartphones, artificial intelligence and machine learning to capture objective, reflex-based measures, supporting earlier and more accurate autism identification. This enables timely intervention during critical periods of brain development. BlinkLab is led by an experienced management team and Board with deep expertise in digital healthcare, computer vision and AI, supported by a Scientific Advisory Board of leading experts in autism and brain development.