REALISE (Real-life Use and Safety of EPIT) Study: 3 Year Results in Peanut-Allergic Children

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Disclosures

- DBV Technologies: principal investigator, consultant, Clinical / Medical Advisory Board
The Burden of Peanut Allergy

**Peanut allergy is the most common food allergy**\(^1\)

2.2% of children in the US are allergic to peanut

**Standard of care for peanut allergy is strict avoidance plus personalized medical intervention plans**\(^4\)

However, despite practicing strict avoidance, accidental exposure often occurs and commonly leads to allergic reactions\(^5-7\)

In some patients, reactions can occur after exposure to low doses of peanut\(^2,3\)

**The management of peanut allergy remains a challenge for patients, families, and healthcare providers due to**\(^8-10\):

- Concerns about unintentional exposure
- Unpredictability of severe reactions
- Relatively high risk of anaphylaxis

Peanut allergy is the most common food allergy\(^1\)
2.2% of children in the US are allergic to peanut

Investigational Epicutaneous Immunotherapy for the Management of Peanut Allergy

**Viaskin™ Peanut 250 µg (DBV712)**\(^1,2\)

- Single, daily-dose patch
  - Applied to the back
- Dose: 250 µg
  - \(\sim1/1000\) of a peanut\(^3\)
- 2-week at-home treatment initiation leading to 24-hour wear time
- No restrictions based on illness or daily activities required

Study Objective

- Efficacy and safety of epicutaneous immunotherapy with Viaskin Peanut has been previously studied in a phase 3 randomized controlled trial in children\textsuperscript{1,2}
- We further examined its safety over 3 years in REALISE, a phase 3 study approximating anticipated real-world use

REALISE Study Design and Methods

- Children aged 4–11 years with physician-diagnosed peanut allergy (well-documented clinical history, SPT $\geq$ 8 mm, and peanut-specific IgE $\geq$ 14 kUA/L) were enrolled
- **Entry food challenges were not required**
- Subjects with a history of severe peanut anaphylaxis were eligible
- Subjects initially randomized to 6 months VP250 or placebo were offered VP250 for a total of 3 years in an open-label extension
- Safety and compliance data were collected

Subject Disposition and Baseline Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Active Treatment Period (ATP) Safety Population N=392</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, n (%)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>229 (58.4%)</td>
</tr>
<tr>
<td>Female</td>
<td>163 (41.6%)</td>
</tr>
<tr>
<td>Median age, years</td>
<td>7.0</td>
</tr>
<tr>
<td>Median peanut-specific IgE, kU/L (range)</td>
<td>95.5 (14.5–1515.0)</td>
</tr>
<tr>
<td>Median SPT wheal size, mm (IQR)</td>
<td>10.5 (9.0–14.0)</td>
</tr>
<tr>
<td>History of severe anaphylaxis, n (%)</td>
<td>14 (3.6%)</td>
</tr>
<tr>
<td>Median treatment exposure to VP250, days</td>
<td>1093.0</td>
</tr>
<tr>
<td>Mean compliance, %</td>
<td>96.4%</td>
</tr>
</tbody>
</table>

DBP=double-blind period; IQR=interquartile range; SPT=skin-prick test; VP250=Viaskin Peanut 250 µg.

The Majority of TEAEs Were Mild or Moderate

- Most subjects (98.7%) treated with VP250 experienced at least 1 TEAE

Severity of TEAEs in subjects who experienced ≥1 TEAE (Total ATP Safety Population [N=392])

- Any:
  - Mild: 98.7%
  - Moderate: 70.4%
  - Severe: 13.8%

- Treatment-related:
  - Mild: 49.0%
  - Moderate: 34.4%
  - Severe: 11.2%

ATP=active treatment period; TEAE=treatment-emergent adverse event; VP250=Viaskin Peanut 250 µg.
Most Treatment-related TEAEs Were Local Application Site Reactions

Most Frequent Treatment-related TEAEs Occurring in ≥10% of Subjects (ATP Safety Population)

<table>
<thead>
<tr>
<th>Preferred Term, n (%)</th>
<th>VP250 N=392</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any Treatment-related TEAE</td>
<td>371 (94.6%)</td>
</tr>
<tr>
<td>Administration Site Conditions</td>
<td>358 (91.3%)</td>
</tr>
<tr>
<td>Application site erythema</td>
<td>297 (75.8%)</td>
</tr>
<tr>
<td>Application site pruritus</td>
<td>259 (66.1%)</td>
</tr>
<tr>
<td>Application site swelling</td>
<td>148 (37.8%)</td>
</tr>
<tr>
<td>Application site papules</td>
<td>57 (14.5%)</td>
</tr>
<tr>
<td>Application site eczema</td>
<td>55 (14.0%)</td>
</tr>
<tr>
<td>Application site urticaria</td>
<td>40 (10.2%)</td>
</tr>
<tr>
<td>Skin and subcutaneous tissue disorders</td>
<td>47 (12.0%)</td>
</tr>
</tbody>
</table>

Incidence and Severity of TEAEs Decreased Over Time

Incidence of TEAEs by year of VP250 treatment (ATP Safety Population)

Severity of TEAEs by year of VP250 treatment (ATP Safety Population)
Rates of Treatment-Related Anaphylactic Reactions Observed

Anaphylactic Reactions

- 16 (4.1%) subjects experienced 17 anaphylactic reactions deemed related to VP250
  - None were severe*
  - In total, 2 serious VP250-related TEAEs (both anaphylaxis):
    - Both were considered medically important events
    - 1 event led to permanent study discontinuation
  - 12 subjects temporarily discontinued and 3 subjects (including the SAE) permanently discontinued treatment due to VP250-related anaphylactic reactions
  - 10 events in 9 subjects (2.3% of total population) required epinephrine administration due to VP250-related anaphylactic reactions
  - 2 additional subjects received epinephrine for non-anaphylaxis VP250-related events

*As assessed by the Investigator based on a protocol-specified staging system for anaphylaxis.

SAE=serious adverse event; TEAE=treatment-emergent adverse event; VP250=Viaskin Peanut 250 µg.

Summary

- In a study designed to mimic potential real-world use, over 36 months, Viaskin Peanut was generally well tolerated by peanut-allergic children aged 4–11 years
- The frequency and intensity of local and systemic treatment-related TEAEs decreased over time
- Compliance was high throughout the duration of the study
- No specific safety concerns arose in subjects with history of severe peanut anaphylaxis
Longitudinal Analysis of Single B Cells, IgE Antibodies, and Plasma in a Peanut Allergic Subject

Jessica Grossman MD*, Derek Croote PhD*, Jonathan Matz, MD†
Distinguished Oral Industry Abstract #8104
IgGenix, Inc.
† Chesapeake Clinical Research

Introduction

• Our understanding of monoclonal, allergen-specific IgE antibodies has been hindered by the scarcity of circulating IgE-producing B cells, which are rare even in allergic individuals.

• The potential to isolate the single B cells producing IgE antibodies and class switch these antibodies to IgG antibodies, like those that increase with allergen-specific immunotherapy, could be of great therapeutic value.
**Background**

- IgE is the antibody class responsible for type I hypersensitivity reactions.
- As IgE is the least abundant antibody class in circulation, it was not discovered until 1967, decades after IgG, IgM, IgD, and IgA.
- IgE is present in plasma at very low levels, in the nanogram per mL range. The serum concentration of IgE in normal individuals only reaches around 50 ng/mL, in contrast to IgG, which is present at concentrations five orders of magnitude higher, or roughly 5–10 mg/mL.
- The IgE half life in blood is only 2 days, as compared to 3 weeks for IgG1. However, IgE bound to mast cell’s will last in tissue for several weeks.


**Allergen Specific Immunotherapy**

**Allergen specific immunotherapy (AIT)**
- AIT induces allergen specific IgE followed by an increase in IgG
- Gave rise to the concept of ‘blocking antibody’ where a few ‘high quality’ IgG antibodies compete with IgE for allergen binding, which correlates with clinical symptom improvement in Peanut OIT

**Passive immunization**
- Transfer of humoral immunity by either serum or purified antibodies provides rapid protection

Adopting this novel approach for treatment of allergy could be of benefit

Methods

- A 28-year-old peanut allergic subject donated 3 blood samples over a 4-month period in an IRB-approved study after qualifying in screening with a peanut-specific IgE titer of >100 kUA/L.

- For each sample, IgE titers were measured by ImmunoCAP and monoclonal IgE antibody sequences from single B cells were identified using our single-cell RNA-sequencing (scRNA-seq) discovery platform.

- We re-engineered these IgE antibodies as monoclonal IgG4 antibodies and characterized their binding specificity to peanut allergens such as Ara h 2.

Single-cell RNA sequencing (scRNA-seq)

Highly sensitive workflow based on cutting-edge single-cell RNA-sequencing to isolate rare B cells and characterize the monoclonal IgE antibodies they produce
Bioinformatic assembly of antibody sequences

Short 150 base pair sequencing reads

Full length cDNA sequences for heavy and light chains are stitched together

Antibody variable regions are cloned into heavy and light chain expression vectors for mammalian cell expression

Results

• Peanut specific IgE and peanut allergen component IgE titers increased over the three separate visits, while total IgE fluctuated. This occurred despite no clinical history of allergen exposure during this time.

• In total, eight hundred IgE-producing B cells were isolated across the three visits.

• Next-generation sequencing analysis followed by antibody gene expression revealed the persistence of clonal families of related B cells that produced high-affinity peanut-specific IgE antibodies.
# Plasma IgE Values

<table>
<thead>
<tr>
<th></th>
<th>External Screen</th>
<th>Visit #1</th>
<th>Visit #2</th>
<th>Visit #3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total IgE</strong></td>
<td>2,415</td>
<td>1,234</td>
<td>1,807</td>
<td>1,688</td>
</tr>
<tr>
<td><strong>Peanut</strong></td>
<td>&gt;100</td>
<td>97.6</td>
<td>486</td>
<td>692</td>
</tr>
<tr>
<td><strong>Ara h 1</strong></td>
<td>&gt;100</td>
<td>53.3</td>
<td>195</td>
<td>342</td>
</tr>
<tr>
<td><strong>Ara h 2</strong></td>
<td>&gt;100</td>
<td>63.7</td>
<td>192</td>
<td>353</td>
</tr>
<tr>
<td><strong>Ara h 3</strong></td>
<td>72.8</td>
<td>30.9</td>
<td>58.8</td>
<td>50.4</td>
</tr>
<tr>
<td><strong>Ara h 6</strong></td>
<td>&gt;100</td>
<td>44.2</td>
<td>124</td>
<td>87.7</td>
</tr>
<tr>
<td><strong>Ara h 8</strong></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Ara h 9</strong></td>
<td>0</td>
<td>0</td>
<td>0.13</td>
<td>0.12</td>
</tr>
</tbody>
</table>

## Clonal Families

- During an immune response, B cells will divide, giving rise to a “clonal family” of related cells that produce antibodies with the same specificity.
- Over these three visits we observed peanut specific antibodies deriving from multiple clonal families.
- This clonal persistence was observed despite the donor having no known exposure to the allergen during this timeframe, suggesting a non-allergen mediated mechanism by which allergy is maintained.
- Further studies are ongoing to understand the extent of clonal persistence and the stability of the allergic phenotype.
Conclusion

- The integration of single-cell transcriptomics, IgE plasma diagnostic measurements, and antibody specificity yields novel insights into the persistence and evolution IgE antibodies.

- These results also highlight the potential for re-engineered antibodies to serve as therapeutics that may avoid long clinical response times and adverse events associated with food-based treatments like oral immunotherapy.
Peripheral Eosinophils, Total IgE, and Atopy in Newly Identified Patients with Gastroduodenal Eosinophilia

Nicholas J. Talley MD PhD, Amol P. Kamboj MD, Mirna Chehade MD MPH, Kathryn A. Peterson MD, Ikuo Hirano MD, Nirmala Gonsalves MD, Marc E. Rothenberg MD PhD, Neal Jain MD, William J. Sandborn MD, Evan S. Dellon MD MPH

1 University of Newcastle, Australia; 2 Allakos, Inc, Redwood City, CA; 3 Icahn School of Medicine at Mount Sinai, New York, NY; 4 University of Utah, Salt Lake City, UT; 5 Northwestern University Feinberg School of Medicine, Chicago, IL; 6 University of Cincinnati College of Medicine, Cincinnati, OH; 7 Arizona Allergy & Immunology Research, LLC, AZ; 8 University of California San Diego, La Jolla, CA; 9 University of North Carolina, Chapel Hill, NC.

Disclosures

- Dr. Neal Jain is an investigator for Allakos Inc.
Background - EGIDs

- Pathologic accumulation and over-activation of eosinophils and mast cells are implicated in chronic inflammatory diseases of the gastrointestinal (GI) tract, including eosinophilic esophagitis (EoE), gastritis (EG), duodenitis (EoD), and colitis—collectively termed eosinophilic gastrointestinal diseases (EGIDs)\(^1\,^2\)

- Patients with EGIDs have decreased quality of life due to chronic and often debilitating symptoms such as dysphagia, abdominal pain, bloating, nausea, vomiting, and diarrhea\(^3\)

- Current treatment options, such as diet restriction and corticosteroids, have limited efficacy and/or are inappropriate for chronic use\(^4\)


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Eosinophilic Gastrointestinal Diseases (EGIDs)

**EG, EoD, EoE**

*Chronic Eosinophilic Inflammation of the Stomach, Duodenum, or Esophagus*

- Eosinophils and mast cells are important drivers of disease

- Symptoms: abdominal pain, nausea, early satiety, loss of appetite, bloating, abdominal cramping, vomiting, diarrhea, and dysphagia (specific to EoE)

- No FDA approved treatment for EG, EoD, or EoE

- Current standard of care: diet and/or steroids

Background – EG/EoD Prevalence

- EG and/or EoD are thought to affect 45,000–50,000 persons in the US; this could be an underestimate\(^1\)
  - Emerging evidence suggest these conditions are highly underdiagnosed\(^2\)
- EG and/or EoD have been described as rare conditions found in atopic individuals with increased peripheral eosinophils and/or total IgE\(^3,4\)
  - However, this conclusion was based on retrospective prevalence and descriptive studies or analyses of claims data, which include patients already diagnosed with EG and/or EoD

ENIGMA: Unexpected High Discovery Rate of EG and/or EoD in Previously Undiagnosed Patients\(^1,2\)

- 51 patients without history of EG and/or EoD entered ENIGMA screening
- 51% (26/51) met symptom criteria for endoscopy and biopsy
- 58% (15/26) EG and/or EoD
- 29% (15/51) received a new diagnosis of EG and/or EoD
- Most patients without a previous diagnosis of EG and/or EoD came from general GI practices
- These patients had a history of chronic nonspecific functional GI symptoms or diagnoses

Suggests significant underdiagnosis of EG and/or EoD

Sources:
Study Objective

- Here, we conducted a prospective study to systematically evaluate symptomatic patients endoscopically to assess the prevalence of EG and/or EoD in patients with unexplained GI symptoms, and to better understand their clinical characteristics.

EG and/or EoD Prevalence Study Design

- **Study Design**
  - Prospective, multi-center study to assess the prevalence of EG and/or EoD in symptomatic patients with chronic functional GI symptoms
  - at least a 6-month history of GI symptoms without identifiable cause and were unresponsive to pharmacologic or dietary interventions, and/or
  - a diagnosis of IBS or functional dyspepsia (FD), indicating a chronicity of symptoms
  - A separate endoscopy study of healthy volunteers (controls) was conducted for comparison

- **Primary Endpoint**
  - Proportion of symptomatic patients who underwent biopsy and met the histologic criteria for EG and/or EoD (≥30 eos/hpf in 5 gastric or 3 duodenal hpf)
GI Symptom Questionnaire

**EG/EoD GI Symptom Questionnaire**

- Developed in accordance with FDA guidance on PRO development
- Captures the GI symptoms of patients on a daily basis
- Measures symptoms each on a scale of 0-10 for the following:
  - Abdominal pain
  - Nausea
  - Vomiting
  - Early satiety
  - Loss of appetite
  - Abdominal cramping
  - Bloating
  - Diarrhea
- Average daily score of ≥3 (on a scale from 0-10) for any individual symptom and a Total Symptom Score (TSS) ≥10
- Same PRO used for asymptomatic healthy volunteers (controls) who had to have an average daily score ≤1 for all symptoms and no daily score ≥3 on any day for any symptom

Systematic Biopsy and Histopathologic Assessment Protocol for Patients and Controls

**Biopsy Protocol**

<table>
<thead>
<tr>
<th>Stomach</th>
<th>Duodenum</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GASTRIC ANTRUM:</strong> 4 biopsies (2-5 cm proximal to the pylorus)</td>
<td><strong>4 biopsies</strong> from the duodenum, 2 each from the descending and horizontal parts</td>
</tr>
<tr>
<td><strong>GASTRIC CORPUS:</strong> 4 biopsies (2 from the proximal lesser curvature and 2 from the greater curvature)</td>
<td></td>
</tr>
</tbody>
</table>

**Assessment Protocol**

- Biopsy samples were collected and sent to the central lab for fixing and staining and then evaluated by an external expert pathologist, who was blinded to all patient demographic, clinical, and endoscopic data
- Eosinophils and mast cells were counted systematically in a minimum of 5 non-overlapping hpfs in at least 12 biopsies to avoid missing areas of infiltration
- Gastric biopsies were graded using the Sydney System on inflammation, metaplasia, atrophy, and reactive gastropathy; the Marsh Scale Classification was used to grade duodenal samples
33% (181/556) of patients with chronic functional GI symptoms and 45% (181/405) of patients with moderate-severe symptoms who underwent biopsy met histologic criteria for EG and/or EoD.

### Features of Patients with EG and/or EoD

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Met Histologic Criteria for EG and/or EoD n=181</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, years (range)</td>
<td>45 (19-78)</td>
</tr>
<tr>
<td>Female sex, %</td>
<td>73%</td>
</tr>
<tr>
<td>White, %</td>
<td>85%</td>
</tr>
<tr>
<td>Weight, median, kg</td>
<td>83</td>
</tr>
<tr>
<td>TSS [0-80], mean ±SD</td>
<td>31.3 ±11.2</td>
</tr>
<tr>
<td>History of:</td>
<td></td>
</tr>
<tr>
<td>GI symptomsb, mean years</td>
<td>11</td>
</tr>
<tr>
<td>GERD, IBS, FD, and/or EoE, %</td>
<td>93%</td>
</tr>
<tr>
<td>GERD, %</td>
<td>65%</td>
</tr>
<tr>
<td>IBS, %</td>
<td>55%</td>
</tr>
<tr>
<td>FD, %</td>
<td>15%</td>
</tr>
<tr>
<td>Atopyc, %</td>
<td>48%</td>
</tr>
<tr>
<td>EoE, %</td>
<td>2%</td>
</tr>
</tbody>
</table>

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a. Patients who met symptom criteria and ≥30 eos/hpf in 5 gastric hpfs and/or ≥30 eos/hpf in 3 duodenal hpfs.
b. Other prior GI diagnoses included other functional GI disorders, such as chronic abdominal pain or functional diarrhea.
c. Asthma, allergic rhinitis, atopic dermatitis and/or food allergy.
Features of Patients with EG and/or EoD

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Met Histologic(^a) Criteria for EG and/or EoD (n=181)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood eosinophils</td>
<td>Cells/µL, median (IQR) 170 (100-250)</td>
</tr>
<tr>
<td></td>
<td>Blood eos ≥500 cells/µL, % 4%</td>
</tr>
<tr>
<td></td>
<td>Blood eos ≥1500 cells/µL, % 0%</td>
</tr>
<tr>
<td>Immunoglobulin E</td>
<td>kU/L, median (IQR) 34 (14-103)</td>
</tr>
</tbody>
</table>

\(^a\) Patients who met symptom criteria and ≥30 eos/hpf in 5 gastric hpf's and/or ≥30 eos/hpf in 3 duodenal hpf's

Blood Eosinophilia and IgE in Patients with EG and/or EoD

Of patients who met histologic criteria for EG and/or EoD, 47% and 4% had ≥250 eosinophils/µL and ≥500 eosinophils/µL, respectively; the median total IgE was 34 kU/L (inter-quartile range, 14–103 kU/L), and 63% of patients had IgE <70 kU/L
45% (181/405) of patients and 6% (2/33) of asymptomatic controls met histologic criteria for EG/EoD (Odds ratio=12.52; 95% CI, 3.0–53.0; \( P < 0.001 \))

<table>
<thead>
<tr>
<th>Controls (n=33)</th>
<th>EG (n=16)</th>
<th>EG+EoD (n=43)</th>
<th>EoD (n=122)</th>
</tr>
</thead>
</table>

Unpaired t-test; * \( P < 0.05 \); ** \( P < 0.01 \); *** \( P < 0.001 \); **** \( P < 0.0001 \)

Patients and controls used the same PRO questionnaire and underwent identical biopsy protocols.

Histologic evaluation for both groups were performed by the same central pathologists.

**Conclusions**

Endoscopy and systematic biopsy of patients with moderate–severe unexplained GI symptoms led to a high discovery rate (45%) of histologic EG and/or EoD

Most newly identified patients did not have peripheral eosinophilia or elevated IgE, indicating that EG/EoD should be considered in symptomatic patients without markers of atopy.

These results suggest that there may be a spectrum or different phenotypes of EG and/or EoD, with different levels of peripheral eosinophils and IgE.

Endoscopy with systematic biopsy and assessment of tissue eosinophils may lead to a precise diagnosis, including EG/EoD.
We thank the patients who participated in this study, the investigators, and all study staff
Rapid Fingerstick based Point-of-Care System for IgE Quantification

*Dr. Bob Geng, M.D., Allergist and Immunologist, UCSD & Rady Children’s Hospital, San Diego
Dr. Collin Terpstra, MD FRCP Allergy and Clinical Immunology Adjunct Professor McMaster University
Dr. Eric Karlin, M.D. Allergy Partners
Dr. Chris Harder, Ph.D., Chief Technology Officer, Kenota Health

Relevant disclosure

Dr. Bob Geng, M.D., Allergist and Immunologist, UCSD & Rady Children's Hospital, San Diego. Advisor
Dr. Collin Terpstra, MD FRCP Allergy and Clinical Immunology Adjunct Professor McMaster University Advisor/Investor
Dr. Eric Karlin, M.D. Allergy Partners Advisor
Dr. Chris Harder, Ph.D., Chief Technology Officer, Kenota Health Employee
Component testing is not readily used due to patient compliance, phlebotomy being difficult and blood test follow-up being cumbersome.

Kenota Health has built a lateral flow-based platform technology* designed to run components in the Allergist office.

*Not currently FDA cleared and not available for sale.

LFA innovations
- Improved accuracy
- Small volume
- Improved sensitivity

Device innovations
- Accurate analysis algorithms
- Precise blood dispense
- Multiple built-in failure alert checks
Kenota aims to complement the immediacy & broad utility of skin prick testing with the precision of component blood-based testing.

FOR THE PHYSICIAN
- Reimbursable
- Single patient visit
- Better diagnostic info

FOR THE PATIENT
- Only mildly invasive
- Single allergist visit
- Actionable info

Finger prick sample collection

Same visit results

In-office blood-based testing

Anonymized results stored in the Cloud

Five components

1. KENOTA INSTRUMENT
   - Three test drawers analyze up to three patients simultaneously

2. TEST CARTRIDGES
   - Each allergy is tested using a cartridge specific to that allergy. Each drawer can process up to 35 cartridges

3. SAMPLE COLLECTION KIT
   - Finger-prick blood sample collection

4. PORTABLE TABLET
   - Instructions and results

5. CLOUD
   - Anonymized patient data is stored on the Kenota database
The device is comparable to ImmunoCAP

Slope = 1  
Intercept = 0  
Within 95% CI

<table>
<thead>
<tr>
<th></th>
<th>EST</th>
<th>LCI</th>
<th>UCI</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-0.035</td>
<td>-2.179</td>
<td>1.372</td>
<td>Pass</td>
</tr>
<tr>
<td>Slope</td>
<td>1.015</td>
<td>0.923</td>
<td>1.089</td>
<td>Pass</td>
</tr>
</tbody>
</table>

\[ y = 1.0147x - 0.0348 \]
\[ R^2 = 0.9794 \]

**Analytical data**

**Limit of quantification**

\[ \text{LOQ} = \text{Limit of quantification} \]
\[ \text{LOQ} < 2 \text{ kU/L} \]
### Precision

Comparable to large lab-based systems

<table>
<thead>
<tr>
<th>Conc.</th>
<th>20</th>
<th>62</th>
<th>107</th>
<th>261</th>
<th>497</th>
<th>691</th>
<th>Avg</th>
</tr>
</thead>
<tbody>
<tr>
<td>%CV</td>
<td>11%</td>
<td>15%</td>
<td>18%</td>
<td>5%</td>
<td>10%</td>
<td>10%</td>
<td>11.5%</td>
</tr>
</tbody>
</table>

### Hook Effect – the test does not under call at higher concentrations
IgE in whole blood stored in the fridge is stable for a short time.

**Whole Blood Stability of 20 kU/L Sample**

The device is not susceptible to clinically significant interferents.
Future panels

- Foods – Peanut, Egg, milk, tree nuts etc…
- Environmental - cat, dog, pollens & molds etc…
- General immunology – IgA, IgM, IgG & IgE, specific Ab titers
- Venoms
- Drug allergies

Conclusion: Kenota is developing a point-of-care system for Total, whole and component IgE quantification. When available, this new system will provide a solution for advanced in-office blood-based IgE diagnostics and treatments.