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Immuron Limited

Developing Therapies that Fundamentally Change the Paradigms of Care

July 2017

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ASX:IMC NASDAQ:IMRN

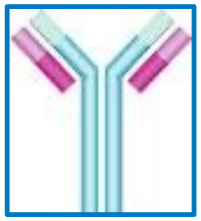
Oral Immunotherapy: Scalable, Disruptive Technology

1

Vaccines Are Developed

2

Antibodies Are Harvested from Colostrum



Antigen Specific Antibodies (IgG and IgG1) + Adjuvants

3

Broad Therapeutic Effect

Induction of regulatory T-cells

+

Clearance of Targeted GUT Pathogens

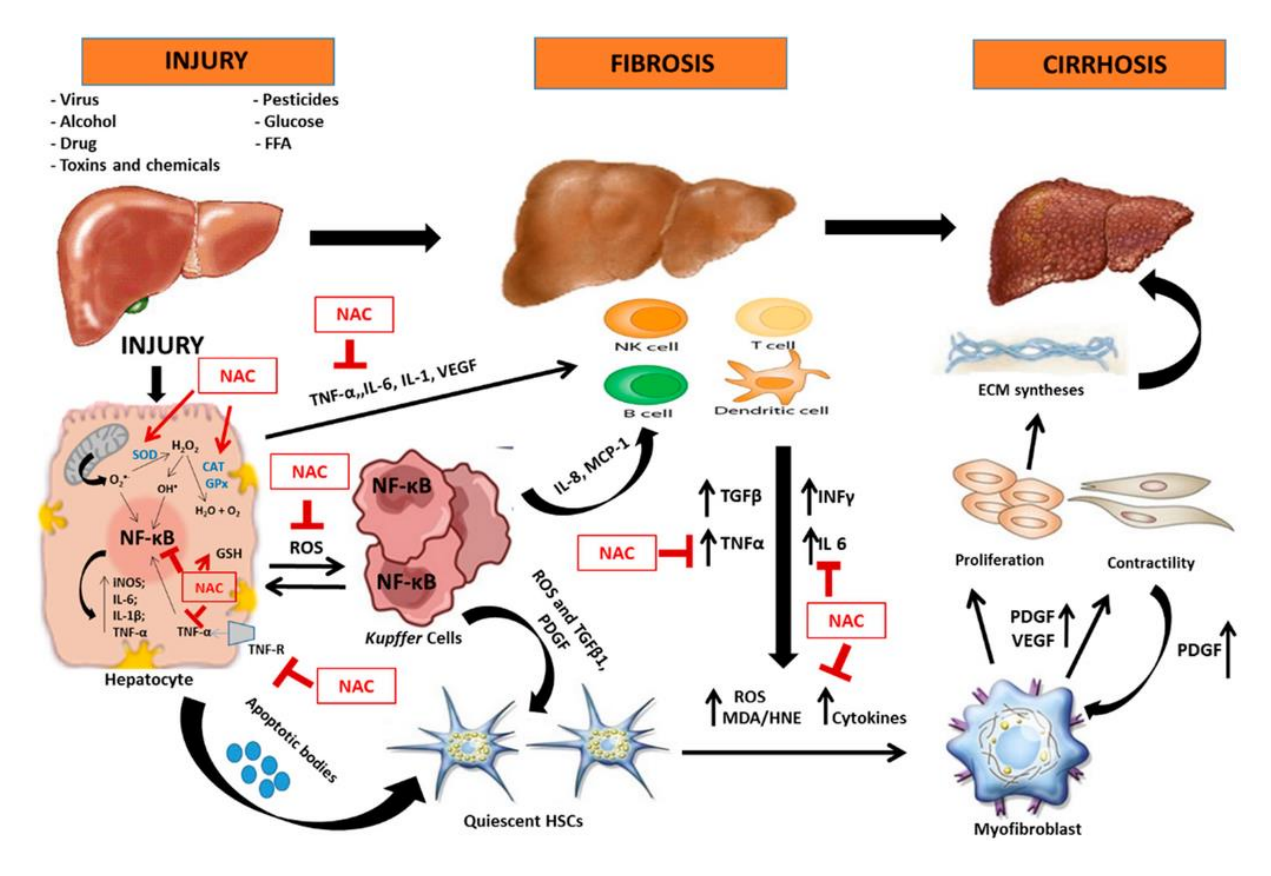
- Reduced gut and blood pathogens responsible for initiating inflammation
- Reduces systemic inflammation
- Lowers organ injury
- Not associated with general immune suppression
- Generally Regarded as Safe (GRAS)

Competitive Advantage

- **Platform capable of spawning multiple drugs** → Long-term value creation
- **Regulated as biologics by the FDA** → 12 years exclusivity in the US for each approval
- **Significant hurdles to generic biosimilar entry** → No pharmacokinetic baseline; Mixture (e.g., Copaxone)
- **Safety established** → Generally Regarded As Safe (GRAS)

NASH (Non-Alcoholic Fatty Liver) Pathophysiology

NASH – Pathophysiology



- Blood derived antigens (including circulating LPS) determines tolerance vs. inflammation
- Kupffer cells play a key role in liver inflammation and fibrosis
- Tregs hold a key role in tolerance (homeostasis)
- Much like hepatic tolerance the gut immune system can promote anti-inflammatory effect

Source: Adapted from Cohen-Naftaly; Scott L. Friedman, 2011

IMM-124E in NASH (Non-Alcoholic Fatty Liver)

- **Targeted antibodies mediate broad anti-inflammatory mechanism of action**
  - Upstream Effect: **LPS-TLR4 pathway**
  - Downstream: **Anti-inflammatory through both innate and adaptive immune systems** (e.g., the induction of regulatory T-cells to control and inhibit excess inflammation)
- Strong **anti-fibrotic effect** demonstrated with CCl4 model
- **Unique competitive profile due to safety/MOA:**
  - Addresses **multi-factorial** nature of NASH
  - Potential for **broad combination use**
  - Safety profile supporting of **long-term chronic use**
  - Potential to **expand to mild/moderate** populations
- **Market exclusivity** (biologics; High barriers to generic biosimilar entry)

IMM-124E – Summary of Data

Prevention of Fibrosis and Improvement in Metabolic & Inflammatory Markers

CCl4 Fibrosis Studies

- **Carbon-Tetrachloride (CCl4) a non-disease related fibrosis model**
- **Aim:** To demonstrate effects of IMM-124E on Fibrosis caused by Intraperitoneal CCl4
- **Results:**
  - Marked **reduction in Liver Fibrosis and Inflammation** on Histology
  - Marked reduction on Liver Damage markers (i.e. ALT, Bilirubin etc.)
  - Marked **reduction in Liver Activated Macrophages (F4/80 high)**

Ob-Ob Mice

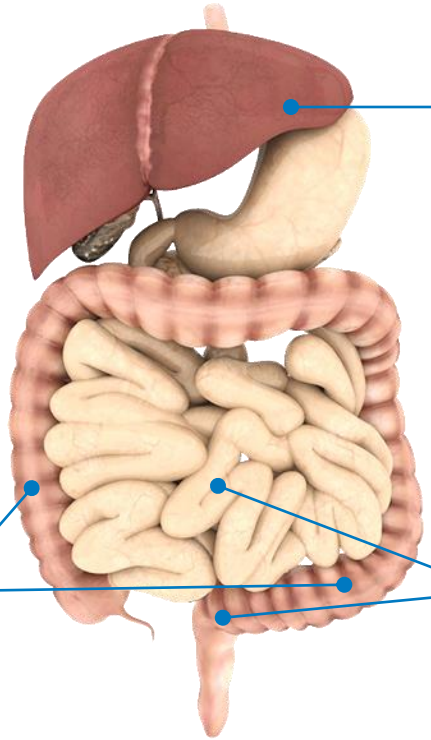
- **Model represents the Metabolic syndrome**
- **Aim:** To demonstrate the effect of IMM-124E or **anti-LPS IgG** (derived from IMM-124E)
- **Results:**
  - Anti-LPS IgG considerable reduces ALT level
  - Improved metabolic status for IG and IMM-124E treated mice (i.e. TG, Fasting Glucose and OGTT)
  - **Anti-inflammatory shift: Decreased TNF-α and increase splenic NKT cells**

Phase 1/2 Clinical Studies

- **Aim: To show safety and efficacy of IMM-124E Biopsy Proven NASH Patients**
- **Population:** 10 subjects with biopsy proven NASH and Type 2 Diabetes
- **Results:**
  - Improved Metabolic status (e.g. HbA1c, HOMA OGTT) GLP1 and Adiponectin
  - Improved Liver status (e.g. ALT)
  - **Proof of concept: increase in Circulatory Regulatory T-Cell**

IMM-124E in NASH (Non-Alcoholic Fatty Liver)

IMM-124E



Liver:

- Activated Kupffer Cells ↓ (F4/80 macrophages)
- Fibrosis and Inflammation ↓

Gut:

- Intestinal LPS ↓
- Intestinal Permeability ↓
- Tolerance – Activation of Innate system to suppress inflammation (NKT, DC, macrophages)

Serum:

- Insulin resistance ↓
- Circulating LPS ↓
- TGF-β ↑
- TNF-α ↓
- IL-2, IL-6, IL-10, IL-12
- Treg ↑ (CD4, CD25, FoxP3)

IMM-124E: Fatty-Liver Portfolio – 3 Phase II Trials

Three Ongoing Phase 2 Programs: NASH, ASH and Pediatric NAFLD

NASH

- Lead Principal Investigator: Arun Sanyal; Former President of AASLD (American Association for the Study of Liver Diseases) and current Chair of the Liver Study Section at the NIH (National Institute of Health)
- Multi-center, double-blinded, placebo controlled trial; 25 sites running in US, Australia and Israel
- Fully recruited: 134 patients with biopsy proven NASH
- Primary endpoint: changes in liver fat content confirmed by MRI; changes in ALT (liver enzymes)
- 3 arms: placebo, high dose and low dose
- Timing: topline results by 4Q 2017

ASH

- NIH funded; sponsored by University of Virginia
- Expected enrollment: 66 patients
- Endpoint: ALT
- Timing: topline results in 2018

Pediatric NAFLD

- NIH funded; sponsored by Emory University
- Expected enrollment: 40 patients
- Endpoint: ALT; 3 months treatment
- Timing: topline results in 1H 2018

IMM-124E: NASH Phase II Trial

IMM-124E-2001 Interim Analysis – No Safety Issues Reported

NASH Study

- The study has 12 scheduled visits over the study duration of 28 weeks (24 weeks treatment and 4 weeks follow-up.
- The interim analysis was triggered when 80 patients (two thirds of the planned study population) had completed the entire 24-week treatment period and had verified Baseline and week 24 MRI data.
- The purpose of the interim analysis was to determine whether any signals exist regarding; safety of the study treatment and to search for signals of efficacy from primary, secondary and exploratory endpoints.

Patient Populations

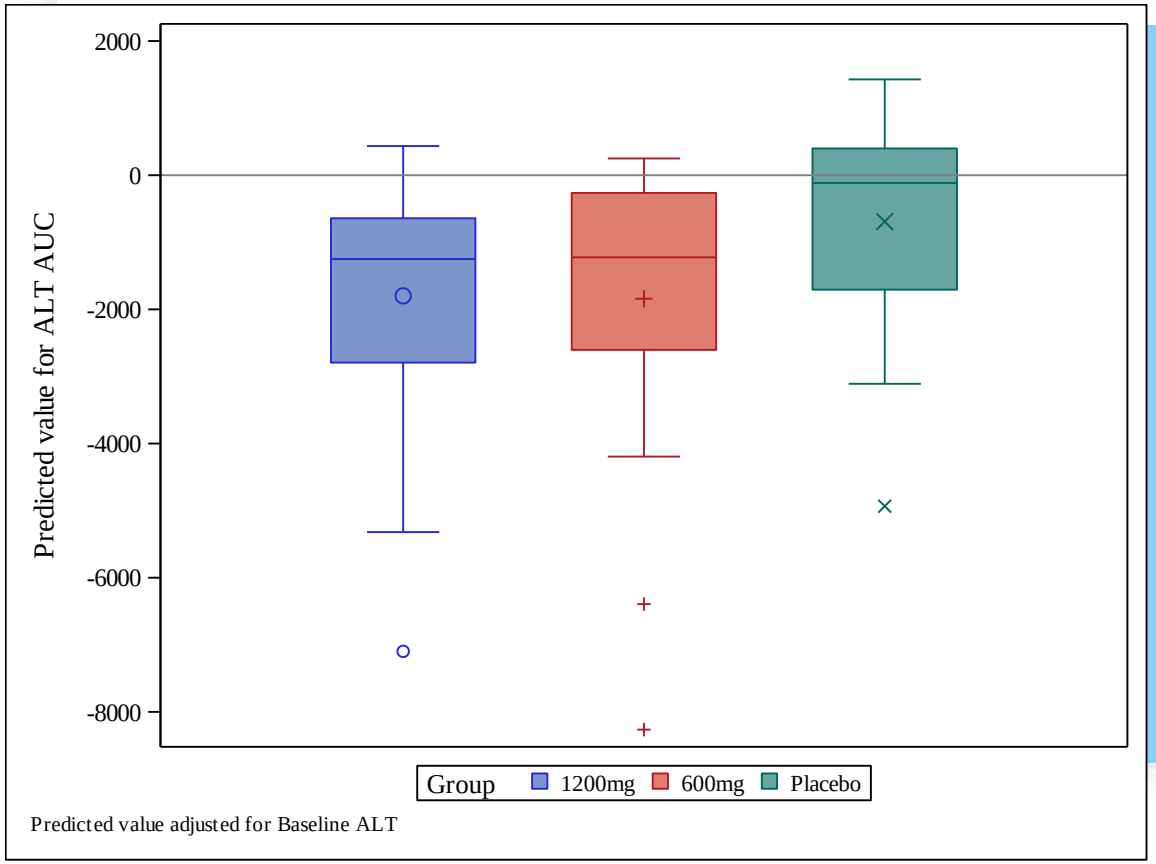
- A total of 133 patients have been randomized into the study.
- To be included in the interim analysis patients were required to have attended one post baseline visit.
- The Full Analysis Set population had 122 patients who met this criterion.
- To be included in the Per Protocol population patients had to complete the 24-week treatment period, have valid Baseline and Week 24 MRI values. A total of 69 patients met this criteria.

Results

- Baseline participant characteristics across the 3 treatment groups are similar
- Baseline LPS 1000 – 10,000 times level reported for healthy blood donors
- Primary endpoint, change in HFF from Baseline to Week 24 did not show any treatment signals, in either FAS or PP populations
- There was a trend for serum ALT to decrease throughout 24 weeks.
- Exploratory analysis of changes in ALT values taking into account all time points by calculating area under the curve and correcting for baseline values demonstrated a dose-related effect.

Phase II: Interim Analysis Report - Improves Liver Function

Box plot for predicted ALT AUC from ANCOVA (FAS population)
Improved Liver Enzymes



Analysis showed that the 1200 mg treatment group approached a significant difference to Placebo and that a dose effect became evident with the 600 mg group

The 1200 mg and 600 mg groups were not different from one another (p=0.3589) but each approached significant difference compared to placebo (p=0.0036 and p=0.0075)

Thank You