Exhibit 99.2

LIPOCINE®

ENHANCING HEALTH

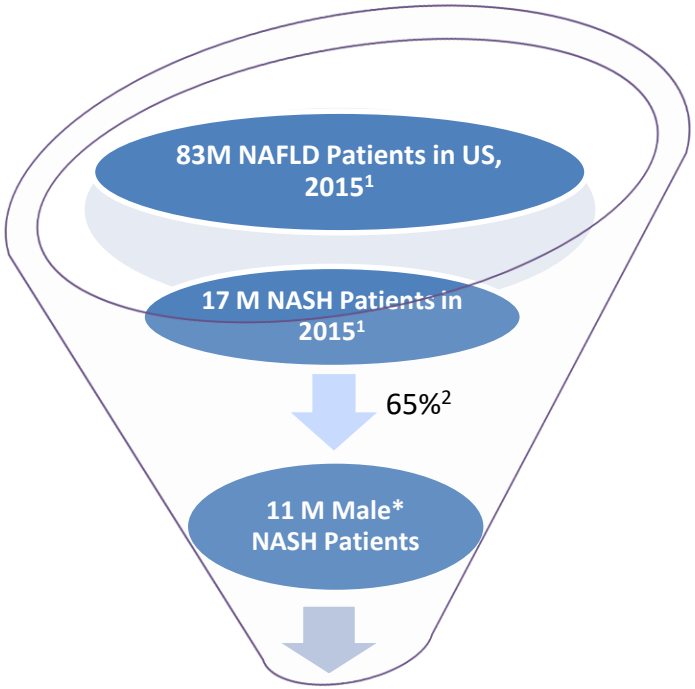
Enabling Oral Drug Delivery to Improve Patient Compliance

LPCN 1144 for Pre-Cirrhotic NASH

LPCN 1144: Rationale to Target Pre-Cirrhotic NASH

Currently No Approved Treatment

Estimated Market



Multi-billion $ Opportunity

Unmet Need

- Efficacy – NASH Resolution and/or Fibrosis Improvement
- Acceptable Tolerability for Chronic Use

Underappreciated conditions

- **Improvement of Sarcopenia[3]**
- **Improvement of Sexual Dysfunction[4]**
- **Improvement of Mental Health/QOL[5,6]**

1. Estes et al., Hepatol 2018;
2. Williams et al., Gastroenterology. 2011.

*near term target

3 Bhanji et al, Hepatol 2017
4. Hawksworth et al., Sex Med Rev 2019
5. Ali et al., Psychosomatics 2011
6. Assimakopoulos et al., J Psychosom Res 2018

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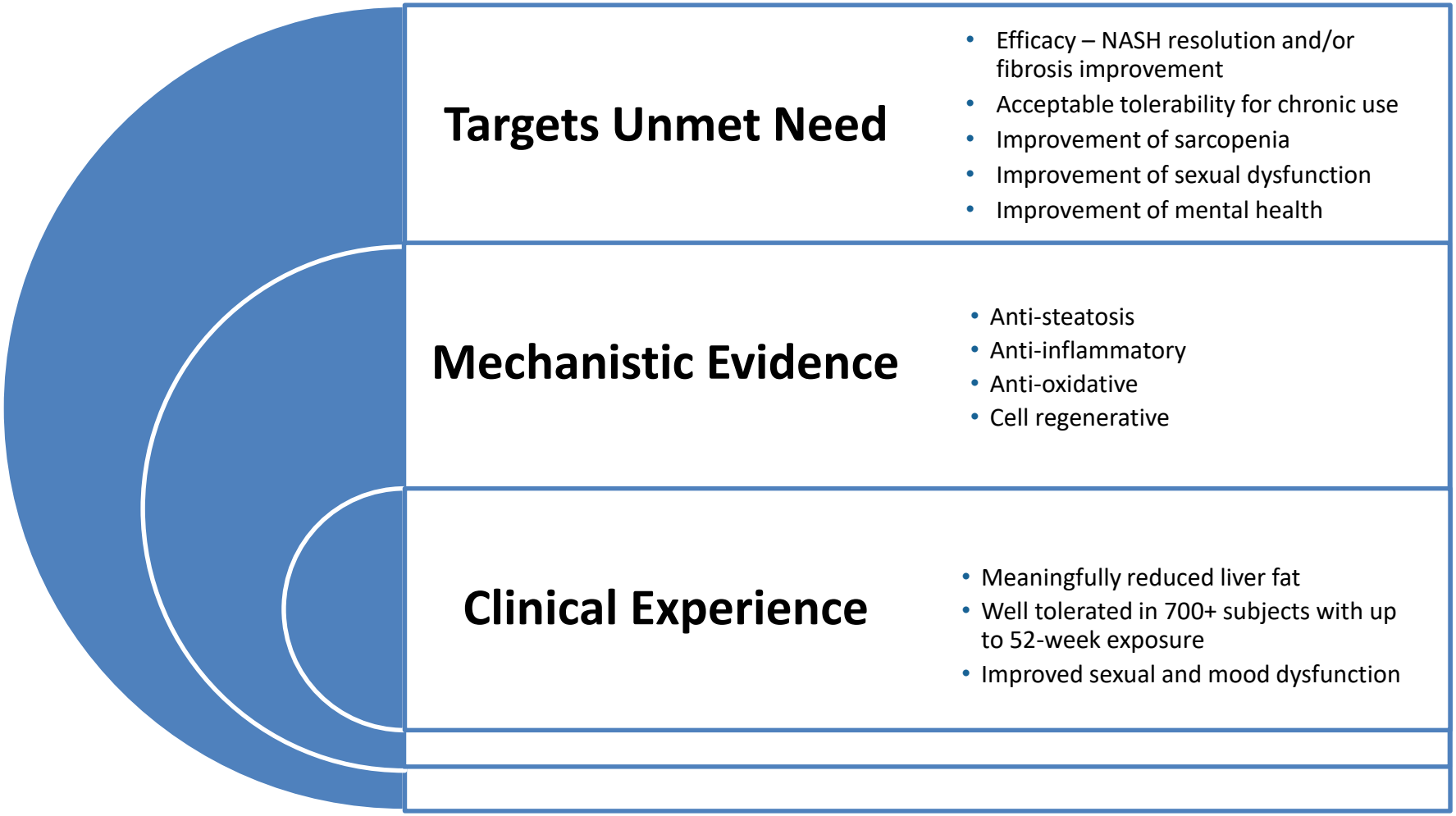
LPCN 1144: Oral Testosterone Therapy

Differentiated NASH Treatment Candidate



LIPOCINE® ENHANCING HEALTH

Association Between T and Liver Disease

Preclinical Evidence

- Low T Induces Liver Disease

	Mouse Model* 
Method	▪ Testicular-feminized
Disease	▪ Hepatic Steatosis

* Kelly et al., Life Sci 2014

- Liver Disease Induction Causes Lower T

Induction Method	Model	T Levels
Gene-modified & Diet	ob/ob (Obese Mouse)[1]	↓ T
	db/db (Diabetic Mouse)[2]	↓ T
Chemical &/or Diet	TAA (Mouse/Rat)[3]	↓ T
	CCL_4 (Rat/Mouse)[4]	↓ T
Diet	High Fat Diet (Rabbit)[5]	↓ T

1. Swerdloff et al., Endocrinol 1976
2. Yabiku et al., BMC Endocr Disord 2018
3. Lipocine TAA rat model study 2019
4. Elsawy et al., PeerJ 2019
5. Vignozzi et al., Mol Cell Endocrinol 2014

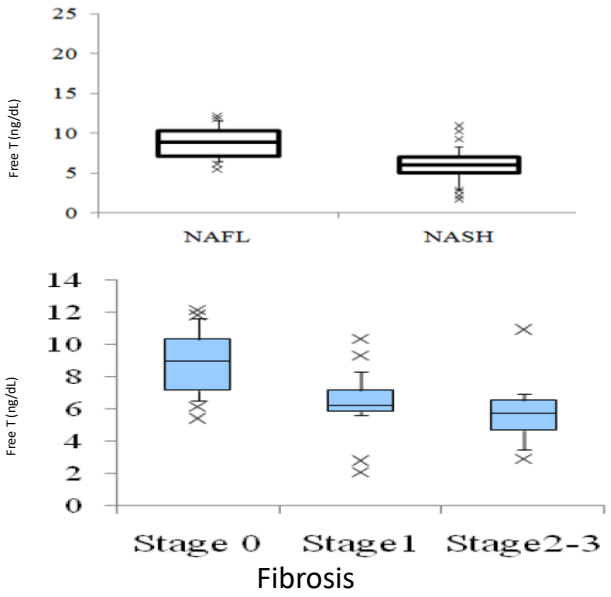
4

LIPOCINE® ENHANCING HEALTH

Association Between T and Liver Disease

Clinical Evidence

- Reportedly 75% of Biopsy-Confirmed NASH Male Patients Have Low T (< 372 ng/dL)[1]



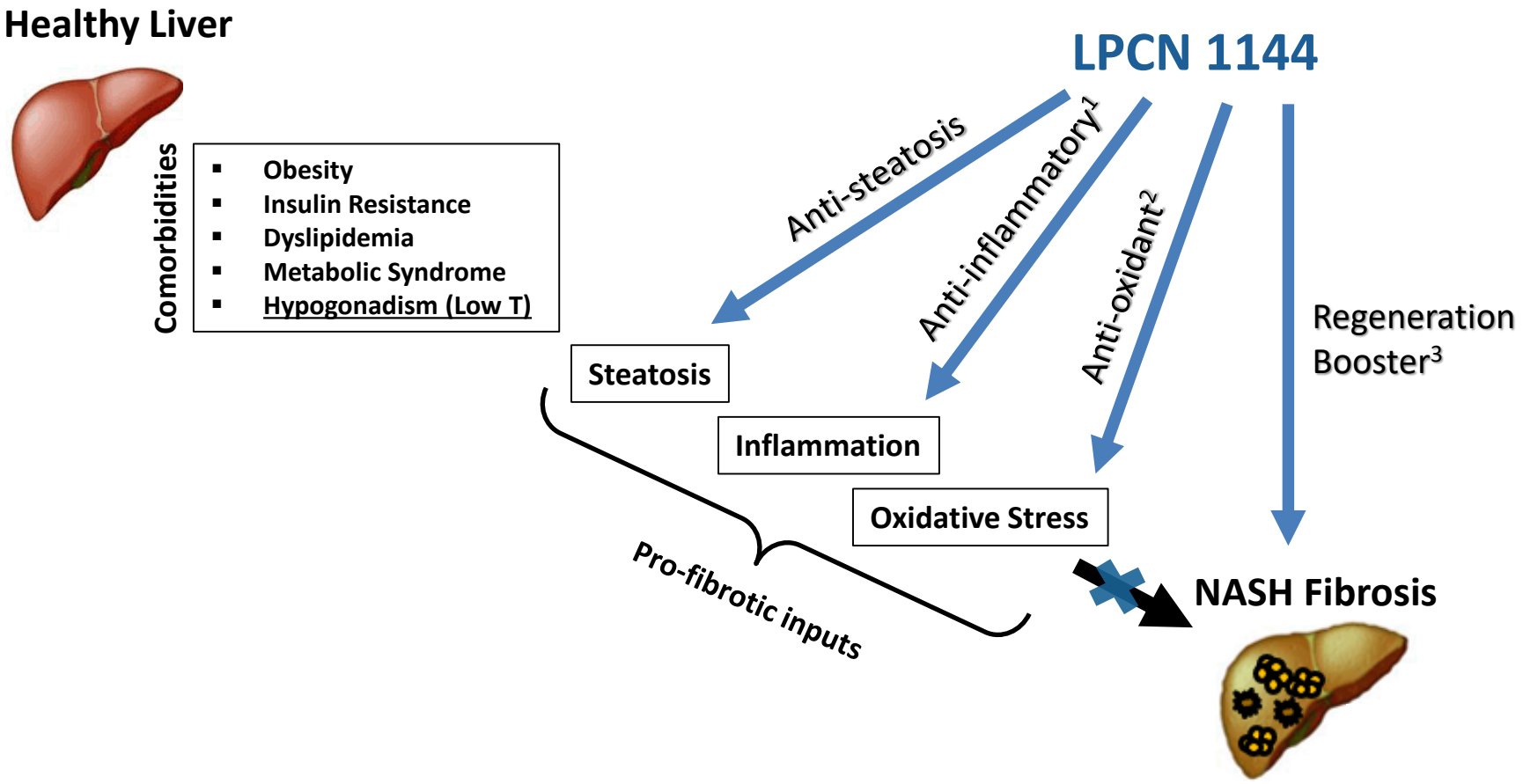
- Levels of **free T** decreased significantly with the increased incidence of lobular inflammation, hepatocyte ballooning, NAFLD activity score, and fibrosis[2].

1. Sarkar et al., Gastroenterology 156(6):S-1258 & Poster Sa1623, Digestive Disease Week 2019
2. Sumida et al., Gastroenterol Hepatol 2015

LIPOCINE® ENHANCING HEALTH

LPCN 1144 Proposed Mechanism

Across the Full Spectrum of NASH Pathogenesis



The removal of pro-fibrotic inputs or the strengthening of anti-fibrotic inputs is expected to stimulate scar resolution[4]

1. Bianchi VE, J Endoc Soc 2019; 2. Vignozzi et al., unpublished clinical data 2018; 3. Vic et al., Hepatol 1982;
4. Lucía Cordero-Espinoza and Meritxell Huch, J Clin Invest 2018

LIPOCINE® ENHANCING HEALTH

Potential of Testosterone Therapy in NAFLD

Preclinical Model Results

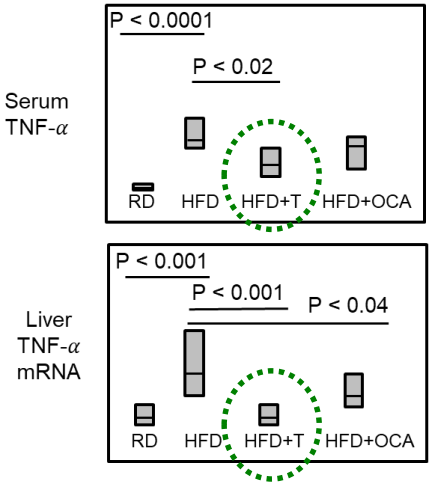
Model	Mouse Model[1] 	Rat Model[2] 	Pig Model[3] 
Methods	▪ Testicular-feminized + High cholesterol diet	▪ Castrated + High fat diet	▪ Castrated + High fat and cholesterol diet
Disease	▪ Hepatic Steatosis	▪ NAFLD	▪ Hepatic steatosis, inflammation, elevated ALT
T Therapy Effect	▪ **Hepatic lipid deposition ↓** ▪ **Lipogenesis ↓**	▪ **Hepatic steatosis ↓** ▪ **Hepatic apoptosis ↓** ▪ **Vesicular inflammation ↓**	▪ **Hepatic lipids ↓** ▪ **Liver injury ↓** ▪ **Hepatic steatosis ↓**

1. Kelly et al., Lif Sci 2014; 2. Nikolaenko et al., BMC Endocrinol 2014; 3. Cai et al., BMC Genomics 2015

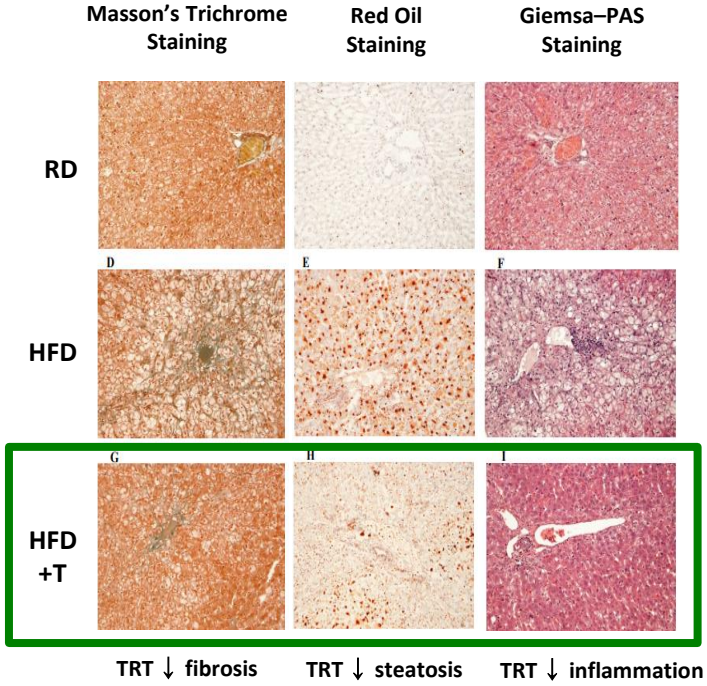
Potential of Testosterone Therapy

Results from high fat diet (HFD) induced rabbit model*

Effects on TNF-α (Inflammatory/Fibrosis Marker)



Effects on Liver Histology



* Vignozzi et al., Mol Cell Endocrinol 2014 T: Testosterone; OCA: Obeticholic Acid

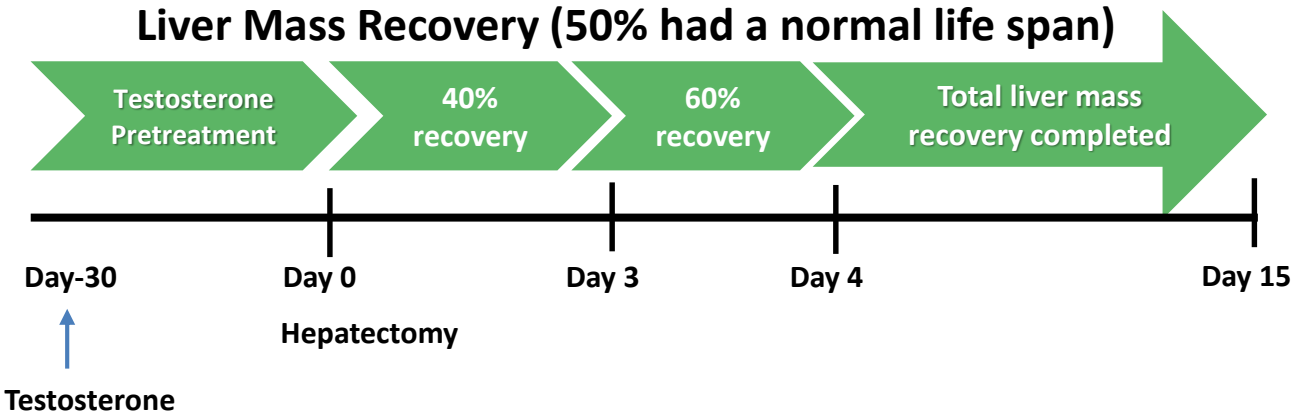
LIPOCINE® ENHANCING HEALTH

T Therapy Effects in Liver Regeneration

Results from Hepatectomized Rat Model*

No Treatment Group 1 (n=50) → **100% died within 40 hours**

T Treatment Group 2 (n=50) → **80% survived beyond 40 hours**



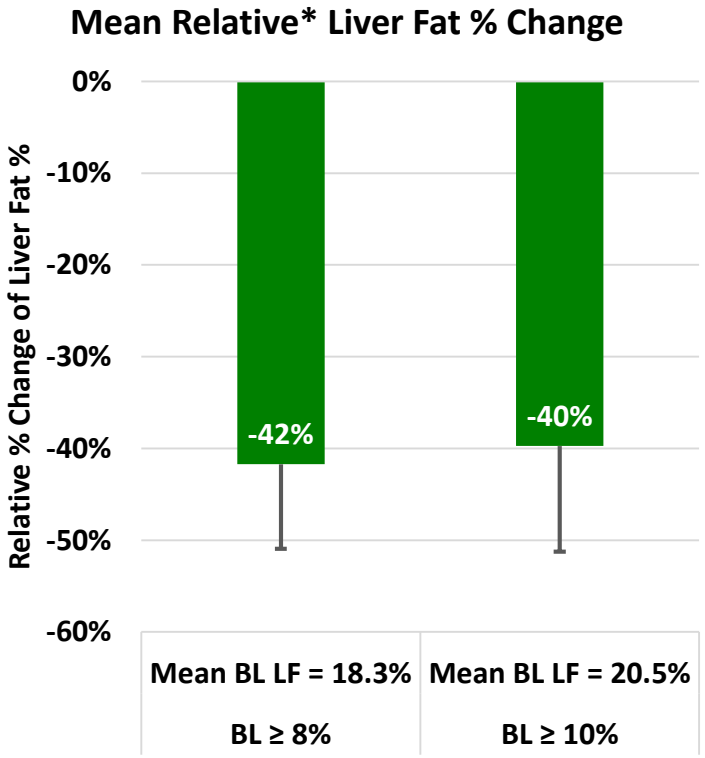
*90% hepatectomized, Vic et al., Hepatol 1982

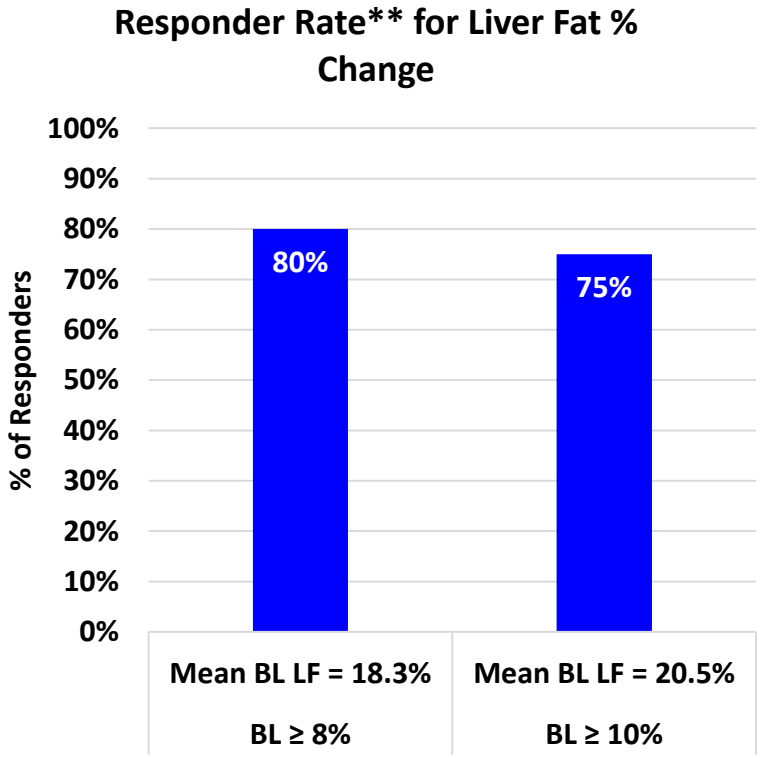
LIPOCINE® ENHANCING HEALTH

LPCN 1144: Liver Fat Imaging Study ("LFS") Results

Meaningful Relative Liver Fat % Change and Responder Rate

LFS was an open-label, multi-center single-arm 16-week study (N=36) with LPCN 1144 in hypogonadal males (NCT03868059)





LF = liver fat

* Mean relative changes of liver fat % were obtained in subjects with BL liver fat ≥ 8% (n=10) and BL ≥ 10% (n=8).

** Responder rate for relative change is % of patients with at least 30% for relative reduction of liver fat % from baseline.

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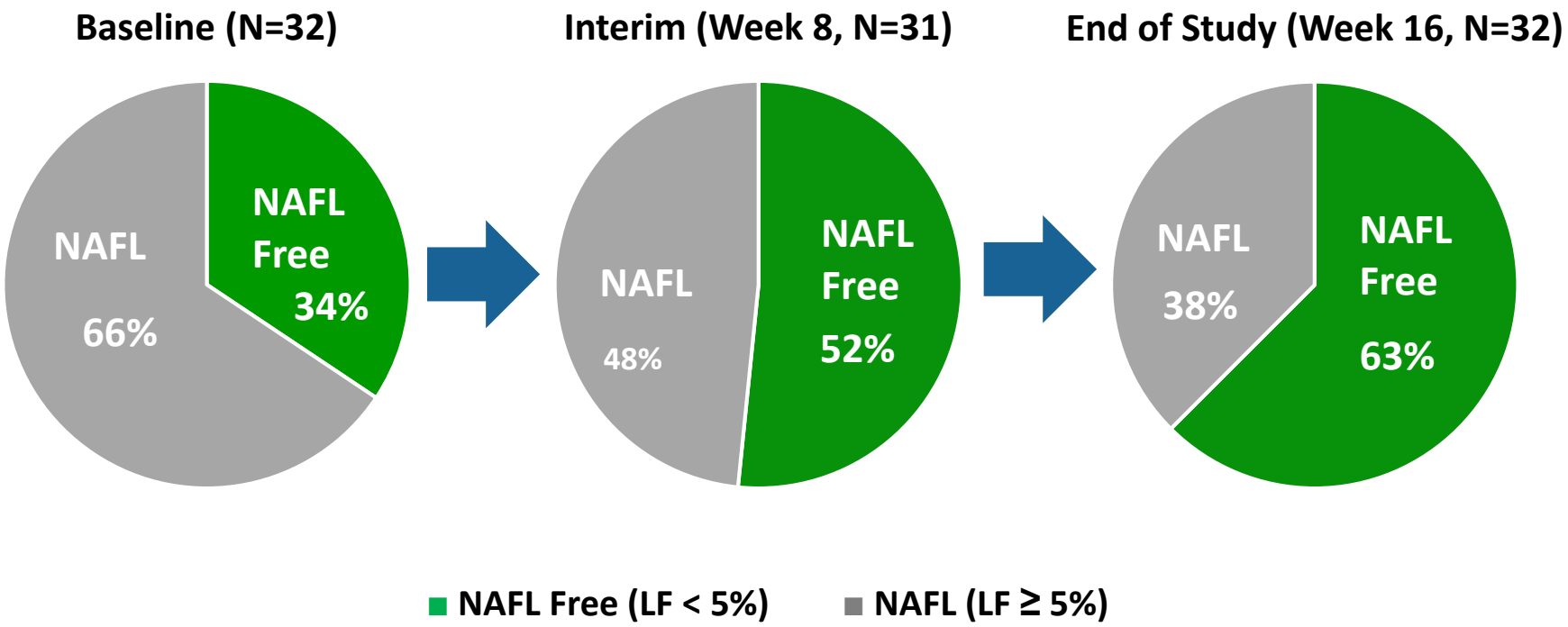
LPCN 1144: Longitudinal Treatment Effect

Improved NAFL Resolution Over Time

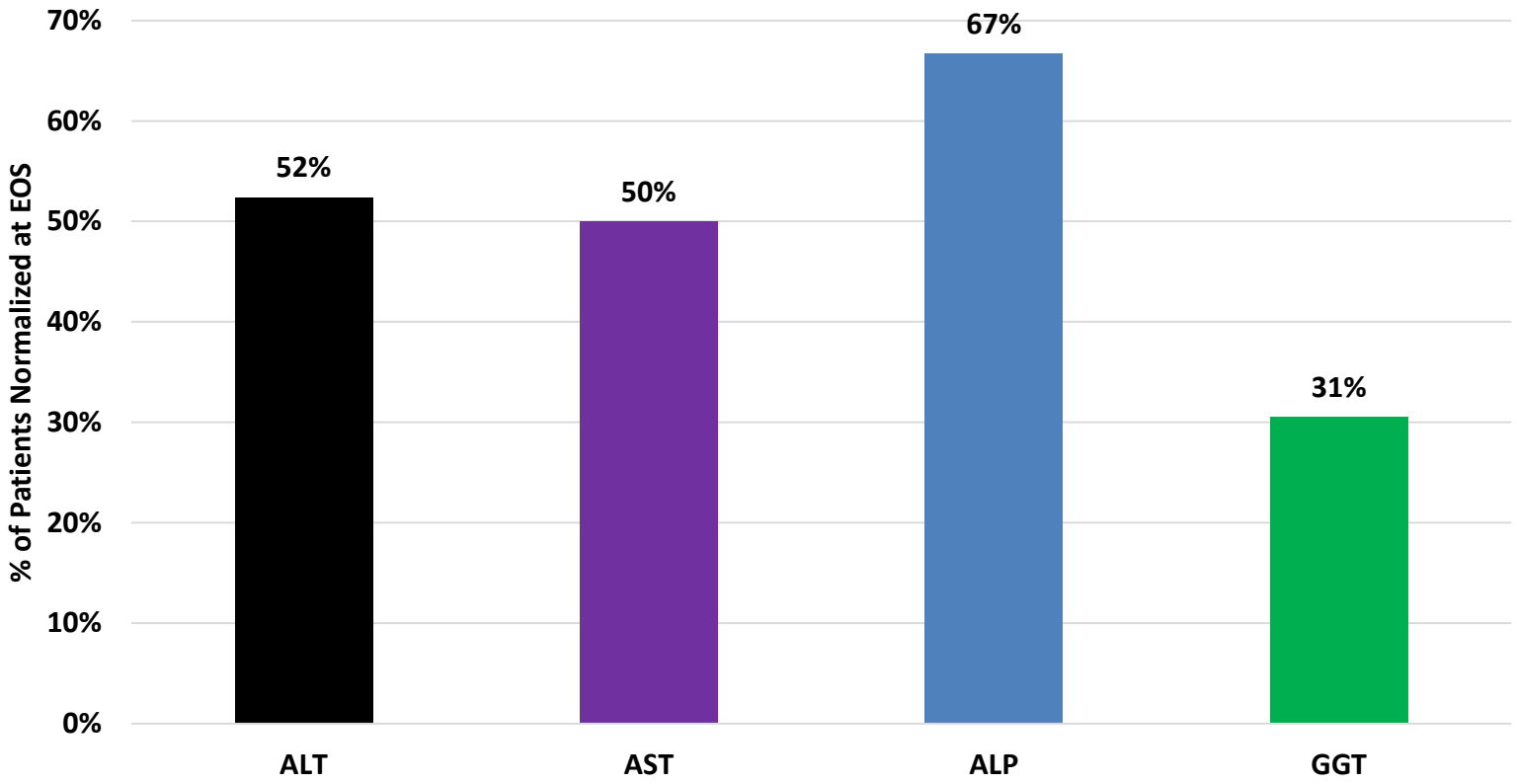


NAFL is non-alcoholic fatty liver

LIPOCINE® ENHANCING HEALTH

LPCN 1144: Resolution of Liver Injury Markers

Significant Normalization of Elevated Liver Enzymes*



N	42	4	6	36
BL	53.6 U/L	59.8 U/L	125.3 U/L	82.2 U/L

*1-Yr SOAR Study (NCT02081300)

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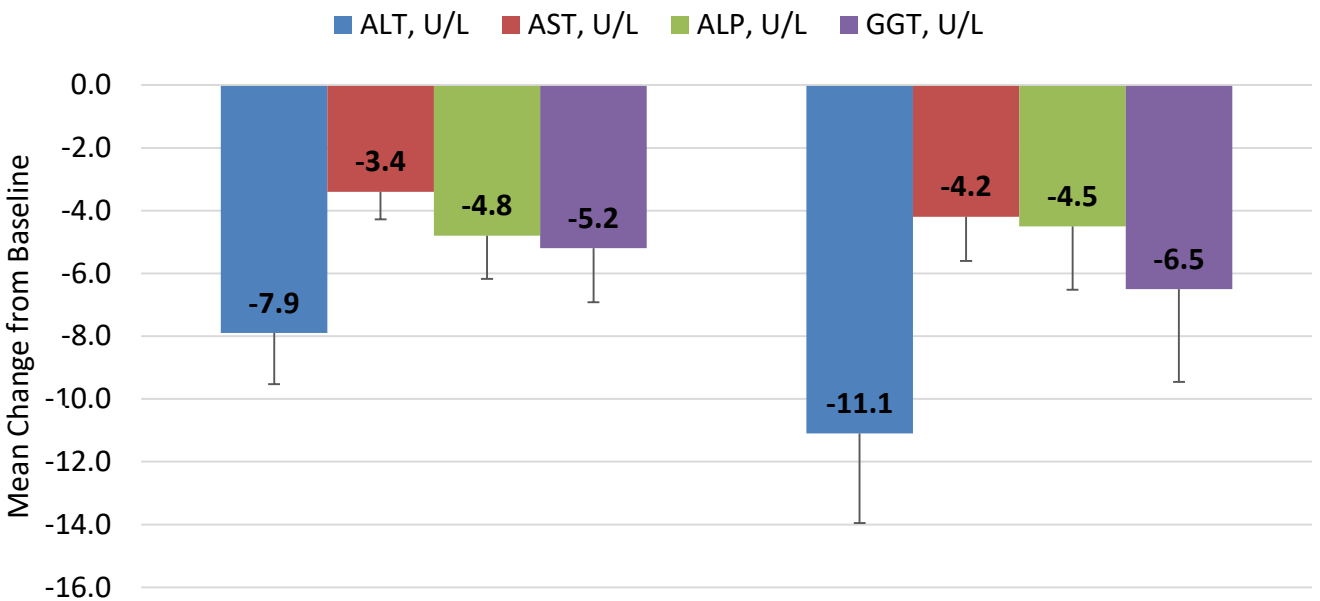
LPCN 1144: Reduction of Liver Injury Markers

In Patients with Elevated ALT at Baseline (BL)

- Liver enzymes mean change from BL to 1-Yr EOS (NCT02081300)



	ALT	AST	ALP	GGT
BL (U/L)	44.5	29.1	68.5	47.1

	ALT	AST	ALP	GGT
BL (U/L)	53.6	32.0	74.0	53.6

* Barritt 4th et al, Contemp Clin Trials, 2017

** Sanyal et al, Hepatol, 2015

Alanine amino transferase (ALT), Aspartate amino transferase (AST), Alkaline phosphatase (ALP), Gamma-glutamyl transpeptidase (GGT)

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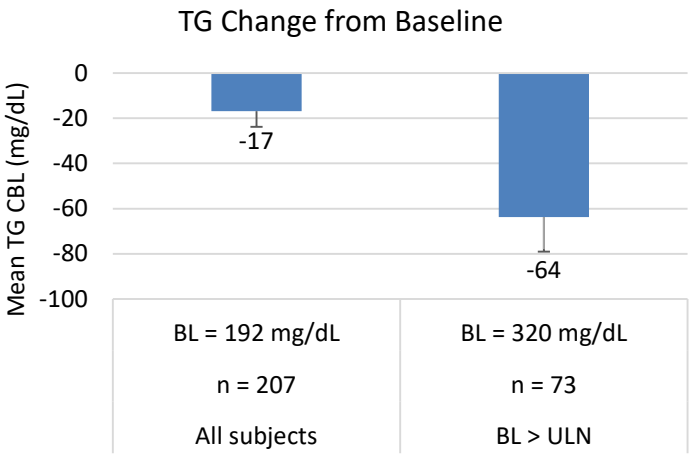
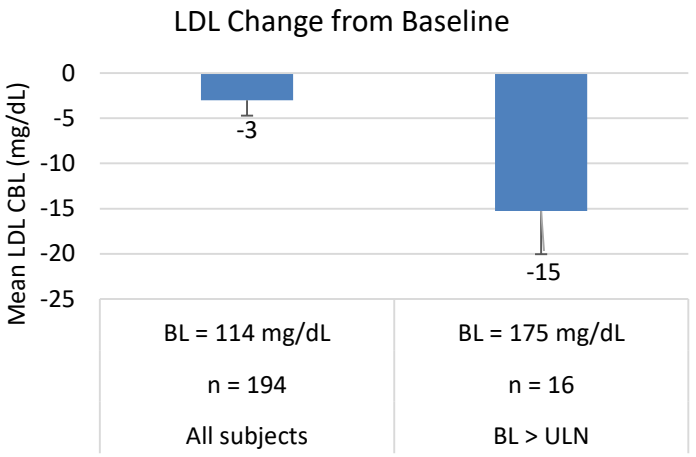
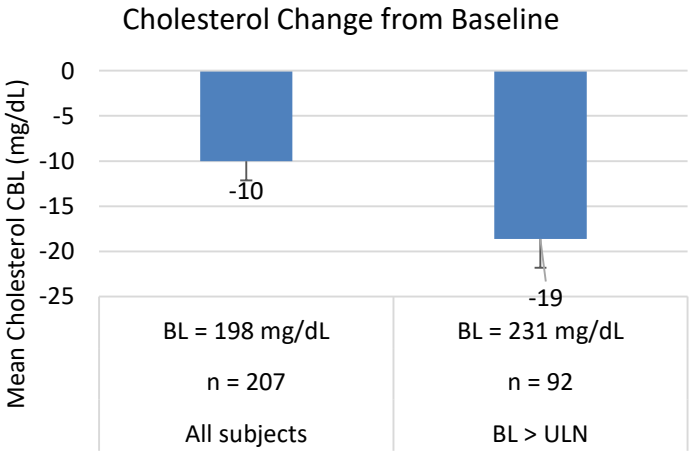
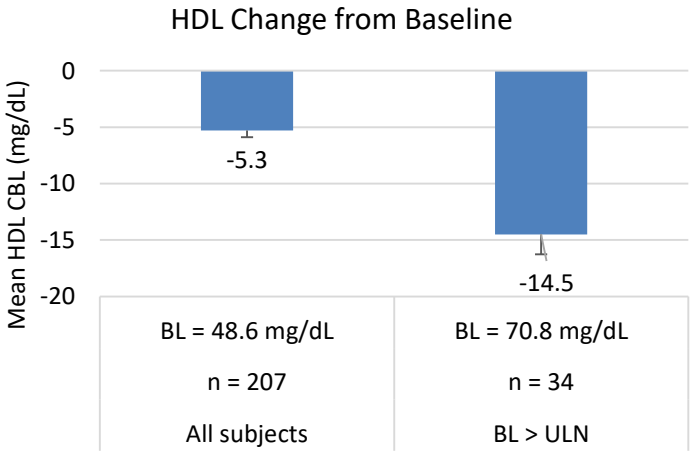
LPCN 1144: Effects on Serum Lipid Markers

In Patients with Elevated Lipids at Baseline (BL)









- TG(triglyceride), LDL, Cholesterol, HDL upper normal limit (UNL) is 200 mg/dL, 160 mg/dL, 200 mg/dL, and 60 mg/dL, respectively.
- 1-Yr SOAR Study (NCT02081300)

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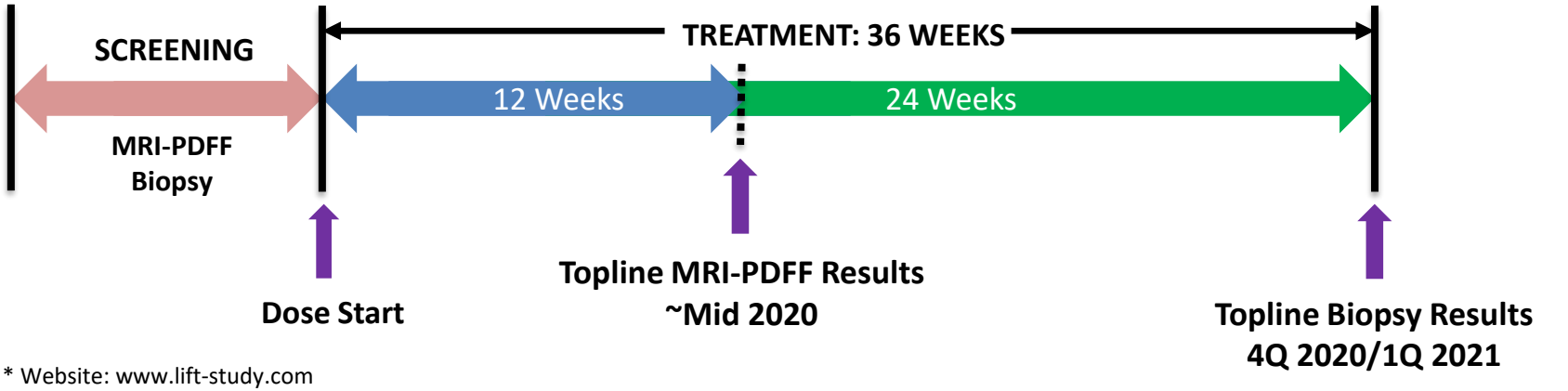
LPCN 1144: *LiFT* Study* Ongoing

Liver Fat Intervention with Oral Testosterone Study

Phase 2 paired-biopsy clinical study in NASH subjects (NCT04134091)

- **Study Design**
  - Three-arm (1:1:1 randomization, two treatments and placebo), multi-center, double-blind
  - 225mg twice daily (450mg Daily)
  - 20-25 biopsy confirmed NASH male subjects per arm with NAS ≥ 4
  - Treatment duration of 36 weeks
- **Primary Endpoint**
  - Change in hepatic fat fraction via MRI-PDFF measure
- **Secondary Endpoints**
  - Change in NASH activity and fibrosis via liver biopsy scoring
  - Change in liver enzymes, anthropometric measure, lipids, insulin resistance, inflammatory/fibrosis markers, and labs
  - Change in quality-of-life degree (SF-36 and PDQ), weight, BMI, waist circumference, waist to hip ratio, and PAQ activity



* Website: www.lift-study.com

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Appendix

LPCN 1144: Multi-dimensional Mechanism of Action

Across the Full Spectrum of NASH Pathogenesis

Homeostasis Modifier[1, 2]	Anti-inflammatory[2]/ Antioxidant/Immuno-modulator[3]	Regeneration Booster[5,6]	Anabolic/Androgenic Agent[9]
• Alter lipid, cholesterol, and glucose metabolism • Reduce visceral abdominal fat • Modify activity of hepatic lipase, and skeletal muscle/ adipose lipoprotein lipase	• Restore mitochondrial turnover and normalizes oxygen consumption[4]	• Stimulate satellite cells and myocyte precursor resulting in cell differentiation and myocyte proliferation[7] • Increases circulating endothelial progenitor cells ("EPC") [8]	• T induces muscle fiber hypertrophy by promoting myogenesis by inhibiting adipogenesis[10]. • Inhibit myostatin[11] • Increase free T (lowering SHBG) • Improve sexual dysfunction[12]

1. Shen and Shi, Int J Endocrinol, 2015
2. Kelly and Jones, J Endocrinol, 2013
3. Sinclair et al., J Gastroenterol Hepatol, 2015
4. Linda Vignozzi et al., University of Florence, IT, unpublished, 2018
5. A. Francavilla et al., Digest Dis Sci, 1989
6. Vic et al., Hepatol 1982
7. Sinha-Hikim et al., J Clin Endocrinol Metab, 2004
8. Liao CH et al., Andrology, 2013
9. Gentile MA et al., J Mol Endocrine, 2010
10. Bhasin S., J Gerontol 2003
11. Dasarathy and Merli, J Hepatol. 2016
12. Rizk et al., Curr Opin Urol 2017

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LPCN 1144: Clinical Tolerability Experience

Oral Prodrug of Endogenous Testosterone

- 654 subjects in multiple completed studies with up to 52-week exposure
  - No death, no drug-related SAEs, no major cardiovascular events, no hepato-toxic events were reported.

Adverse Reaction	N=654 subjects
Headache	1.50%
Acne	0.90%
Hematocrit Increased	1.20%
Blood Pressure Increased	0.30%
Fatigue	0.20%
Hypertension	0.60%

Sarcopenia is Associated with NAFLD/NASH[1]

LPCN 1144 has Potential to Improve Sarcopenia[2,3]

HEPATOLOGY
AASLD
CONCISE REVIEW | HEPATOLOGY, VOL. 66, NO. 6, 2017

Sarcopenia in Hiding: The Risk and Consequence of Underestimating Muscle Dysfunction in Nonalcoholic Steatohepatitis

Rahima A. Bhanji,[1] Praveena Narayanan,[2] Alina M. Allen,[1] Harmeet Malhi,[1] and Kymberly D. Watt[1]

Testosterone therapy increases muscle mass in men with cirrhosis and low testosterone: A randomised controlled trial[3]

Marie Sinclair[1,2,*], Mathis Grossmann[1,3], Rudolf Hoermann[1], Peter W. Angus[1,2,†], Paul J. Gow[1,2,†]

[1]*The University of Melbourne, Australia;* [2]*Gastroenterology and Hepatology, Austin Health, Melbourne, Australia;* [3]*Endocrinology, Austin Health, Melbourne, Australia*

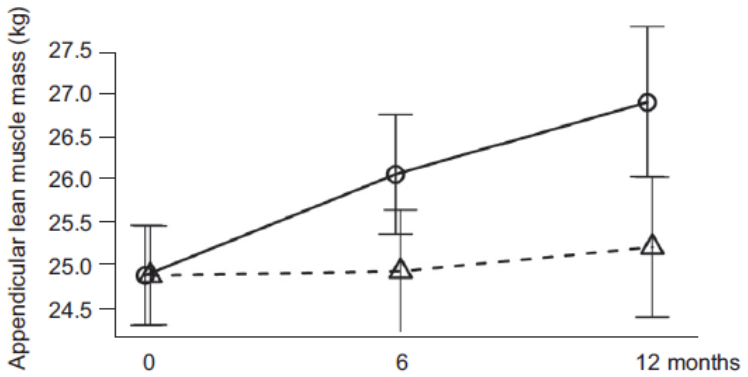

Fig. 1. Mean adjusted difference in appendicular lean mass. Appendicular lean mass progressively increased in the testosterone therapy group, while remaining stable in the placebo group. ○, testosterone therapy; Δ, placebo.

- ✓ Sarcopenia endpoint(s) under evaluation in the ongoing *LiFT* trial

1. Bhanji et al., Hepatol 2017
2. Bhasin S., J Gerontol 2003
3. Sinclair et al., J Gastroenterol Hepatol 2016

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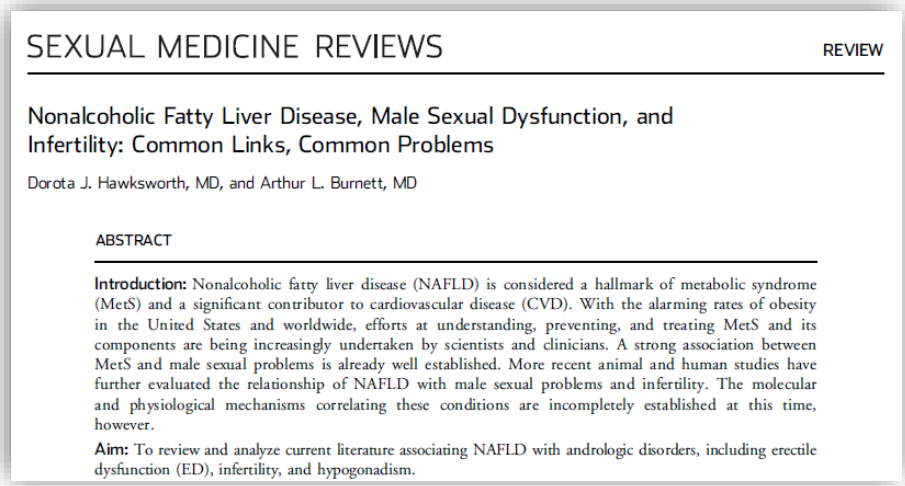
Sexual Dysfunction is Associated with NAFLD/NASH[1]

LPCN 1144 has Potential to Improve Sexual Dysfunction[2]



SEXUAL MEDICINE REVIEWS REVIEW

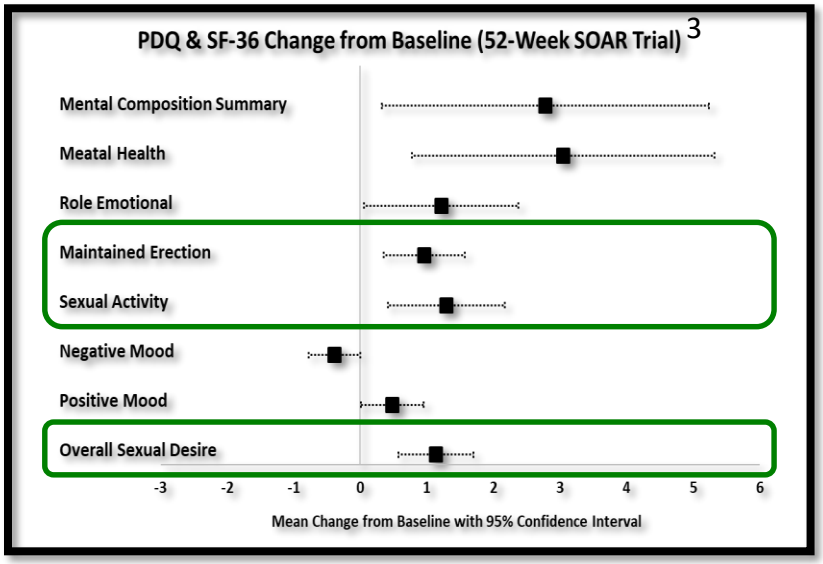
Nonalcoholic Fatty Liver Disease, Male Sexual Dysfunction, and Infertility: Common Links, Common Problems

Dorota J. Hawksworth, MD, and Arthur L. Burnett, MD

ABSTRACT

Introduction: Nonalcoholic fatty liver disease (NAFLD) is considered a hallmark of metabolic syndrome (MetS) and a significant contributor to cardiovascular disease (CVD). With the alarming rates of obesity in the United States and worldwide, efforts at understanding, preventing, and treating MetS and its components are being increasingly undertaken by scientists and clinicians. A strong association between MetS and male sexual problems is already well established. More recent animal and human studies have further evaluated the relationship of NAFLD with male sexual problems and infertility. The molecular and physiological mechanisms correlating these conditions are incompletely established at this time, however.

Aim: To review and analyze current literature associating NAFLD with andrologic disorders, including erectile dysfunction (ED), infertility, and hypogonadism.



✓ Sexual dysfunction endpoint(s) under evaluation in the ongoing *LiFT* trial

1. Hawksworth et al., Sex Med Rev 2019
2. Rizk et al., Curr Opin Urol 2017
3. SOAR Trial: SOAR Trial: subjects for ALT BL > 40 U/L (N=33)

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Depression is Prevalent in NAFLD Patients[1]

LPCN 1144 has Potential to Improve Mental Health[2]

Psychosomatics 2011:52:127–132

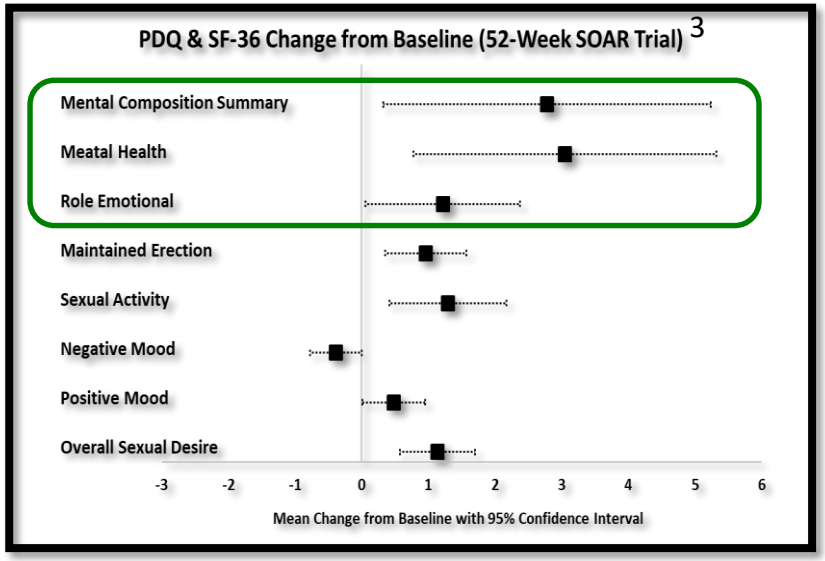
Original Research Reports

Depression in Patients with Nonalcoholic Fatty Liver Disease and Chronic Viral Hepatitis B and C

Ali A. Weinstein, Ph.D., Jillian Kallman Price, M.S., Maria Stepanova, Ph.D., Laura W. Poms, M.S., M.P.H., Yun Fang, M.S., Juhi Moon, M.D., Fatema Nader, M.S.B.M., Zobair M. Younossi, M.D., M.P.H.

Background: *Patients with chronic liver disease (CLD) and depression may be at a higher risk for various complications, including impaired quality of life and more advanced liver disease. The purpose of this study was to determine the prevalence of depression in CLD patients (non-alcoholic fatty liver disease (NAFLD), Hepatitis B (HBV), and Hepatitis C (HCV)) and to identify potential clinical and laboratory correlates of depression in these patients.* **Methods:** *We used a database of CLD patients that contains extensive clinical (including self-reported depression) and laboratory data for each patient. We compared the prevalence of depression in patients with HBV, HCV, and NAFLD. We also used regression models to find independent predictors of depression in these patients.* **Results:** *Of 878 CLD patients, 207 (23.6%) had a diagnosis of depression (NAFLD 27.2%, HCV 29.8%, and HBV 3.7%). Examination of predictors of depression differed by the type of chronic liver disease. For NAFLD, independent predictors of depression were the presence of hypertension, smoking, history of lung disease, being female, and non-African-American. For HBV patients, the only independent predictor of depression was excessive alcohol consumption (defined as >10 g/d), while for HCV patients, independent predictors were being female and non-Asian, presence of fatigue, and excessive alcohol intake.* **Conclusions:** *This study demonstrates that individuals with NAFLD and HCV have a higher prevalence of depression than HBV patients and the rates of depression reported for the general population. The most consistent correlates of depression status in CLD patients are being female and excessive alcohol consumption.*

(Psychosomatics 2011; 52:127–132)



✓ Mental health endpoint(s) under evaluation in the ongoing *LiFT* trial

1. Ali et al., Psychosomatics 2011 2. Celec et al., Front Neurosci 2015
3. SOAR Trial: subjects for ALT BL > 40 U/L (N=33)

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Quality of Life is Poor in NAFLD Patients[1]

LPCN 1144 has Potential to Improve Quality of Life[2]

Journal of Psychosomatic Research 112 (2018) 73–80

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Review article

Quality of life in patients with nonalcoholic fatty liver disease: A systematic review

Konstantinos Assimakopoulos[a], Katerina Karaivazoglou[a], Evangelia-Eirini Tsermpini[b], Georgia Diamantopoulou[c], Christos Triantos[c,*]

[a] Department of Psychiatry, School of Medicine, University of Patras, Rion Patras, Greece
[b] Department of Pharmacy, Laboratory of Molecular Biology and Immunology, School of Health Science, University of Patras, Rion Patras, Greece
[c] Department of Gastroenterology, School of Medicine, University of Patras, Rion Patras, Greece

ARTICLE INFO

Keywords:
Nonalcoholic fatty liver disease
Quality of life
Systematic review

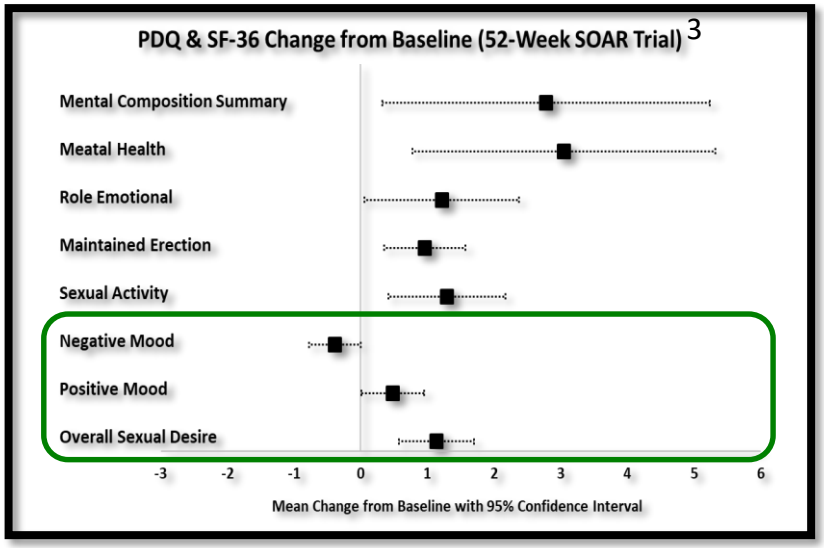
ABSTRACT

Objective: Non-alcoholic fatty liver disease (NAFLD) is a highly prevalent medical condition, which may lead to severe complications including cirrhosis and hepatocellular carcinoma. Its chronic course and its association with obesity and diabetes mellitus augment the long-term impact of NAFLD on patients' health and quality of life (QoL) and put great strain on healthcare systems worldwide. Research is growingly focusing on NAFLD patients' QoL in an attempt to describe the full spectrum of disease burden and tackle its future consequences. Relevant studies are characterized by sample heterogeneity and provide conflicting findings which should be interpreted with the use of a systematic and integrative approach. In this context, our aim was to conduct a systematic literature review on the topic of NAFLD patients' QoL.

Methods: We performed a systematic search of PubMed, ScienceDirect and GoogleScholar databases according to the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) protocol.

Results: Our search yielded 14 suitable articles reporting data from almost 5000 patients. All authors agree that NAFLD patients' QoL is impaired especially in the physical sub-domain. In addition, several demographic, clinical and histopathological parameters have emerged as major determinants of patients' QoL. However, future studies are needed to further clarify these issues.

Conclusions: NAFLD patients report poor physical QoL. QoL impairment is associated with a variety of disease-related parameters, mostly the presence of fatigue and cirrhosis.



- ✓ Quality of life endpoint(s) under evaluation in the ongoing *LiFT* trial

1. Assimakopoulos et al., J Psychosom Res 2018
2. Almehmadi et al., Arab J Urol 2016
3. SOAR Trial: subjects for ALT BL > 40 U/L (N=33)

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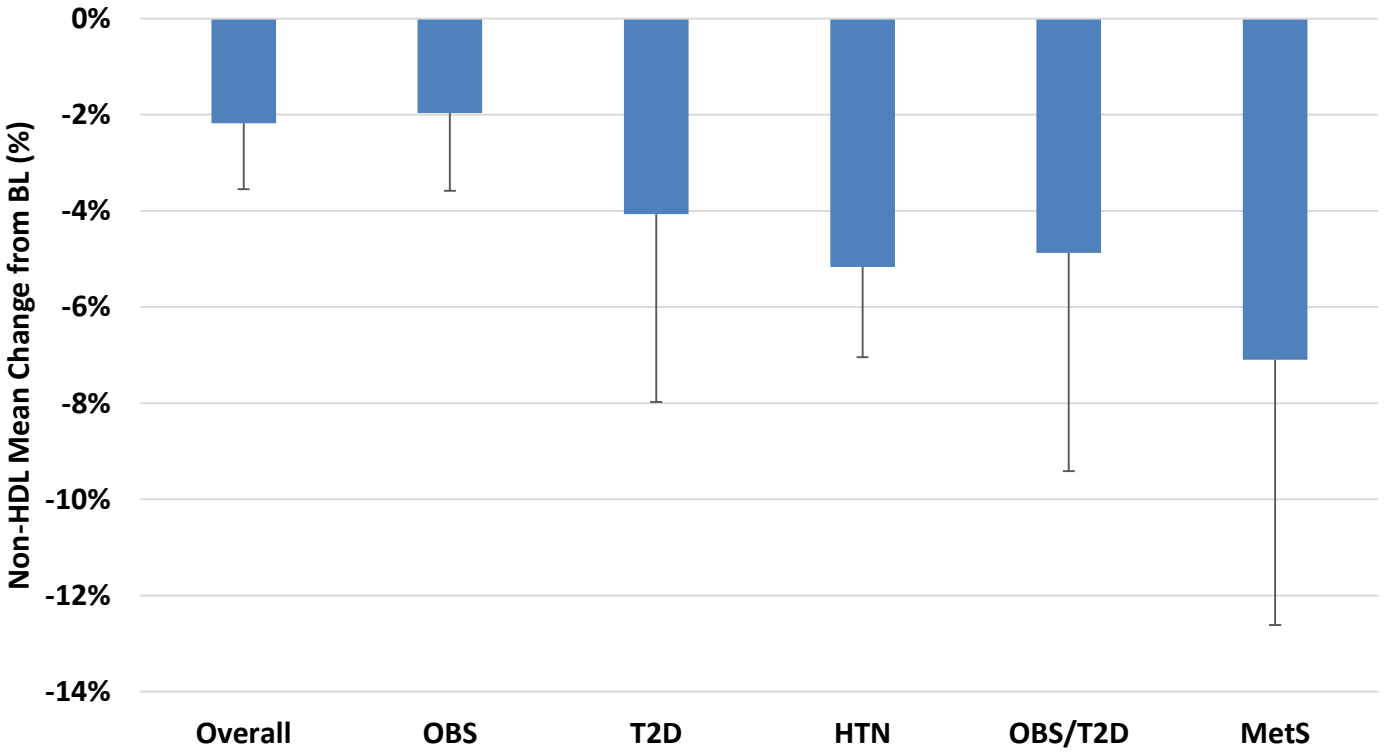
LPCN 1144: Decreased Non-HDL Lipid

Decrease in Non-HDL in 1-Yr SOAR Trial (NCT02081300)



n	207	116	45	98	24	18
Baseline, mg/dL	149	151	135	143	140	138

OBS: Obesity, T2D: Type 2 diabetes, HTN: Hypertension, MetS: Metabolic Syndrome

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