HepAssure

Pilot Phase 2 Clinical Study

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Proof-Of-Concept Pilot Phase 2 Clinical Study

The following study is IRB-approved (OHRP #IRB00010024, #3).

The Principal Investigator is Dr. Richard Melde

Because this is an on-going, longitudinal, in-house study these patient results have not been reviewed by IRB or published in a peer-review journal. The limitations of this study are that there is no control group, no placebo, and no randomization. Therefore it is impossible to deduce comparatively how often the medical food works compared to control.

Also, the fibroscan scores reported below were performed in-house at The LiverLab Fibroscan clinic, rather than by an independent third-party clinic. The purpose of the study is to ensure that the study patients sign informed-consent agreements prior to enrollment and to ensure consistency in study protocol while compiling individual case reports and in-house data for further analysis. The results reported in the following study report demonstrate the need for further randomized, placebo-controlled studies.

Please Note: Every patient with Chronic Liver Disease (CLD) is unique. Many patients with CLD have additional co-morbidities, including: age, obesity, diabetes, poor diet, sedentary lifestyle, alcohol use/abuse, etc., that may increase the underlying metabolic disruption associated with each patient's condition. Therefore, there is no guarantee that a patient's experience with HepAssure medical food will be the same as patients enrolled in the study described below, and no inferences should be taken that HepAssure medical food is guaranteed to reverse liver fibrosis in every patient.

Abstract

Progressive liver fibrosis is a common clinical manifestation of chronic liver disease (CLD). While liver fibrosis was once considered irreversible, recent studies have shown that liver fibrosis can be reversed once the fibrogenic trigger is inactivated or eliminated, as in patients with hepatitis C (HCV) who successfully treat with antiviral therapy, or obese patients with bariatric surgery or recently, obese people with metabolic syndrome who lose weight with weight-loss drugs.

Collagen deposition in the exctracellular matrix is actually a homeostatic system. Our body has a method to create collagen scar tissue in the liver through the conversion of hepatic stellate cells to collagen-secreting myofibroblasts. Conversely, secretion of collagen-absorbing (resorbing) enzymes called collagenase metalloproteinase enzymes reduce collagen content in the extracellular matrix.

The trigger to liver fibrosis - stellate cell conversion and fibrogenesis is initially triggered by depletion of dietary methyl donors. The depletion of dietary methyl donors triggers a downstream cascade of metabolic disruptions to methionine, glutathione and

choline metabolism. Depletion of dietary methyl donors causes depletion of S-adenosylmethionine (SAM), accumulation of S-adenosylhomocysteine (SAH) and homocysteine (HCY), glutathione (GSH) depletion, and phosphatidylcholine (PC) depletion. Glutathione depletion and extreme oxidative stress combine to deplete small-molecule antioxidants across the board.

Depletion of diet-derived methyl donors and disruption of these metabolic pathways in patients with CLD triggers liver fibrogenesis (stellate cell conversion) and stellate cell conversion.

The protocol of the present IRB-approved study consists of the administration of a targeted nutritional repletion formula containing 31 GRAS ingredients formulated specifically designed to replete dietary methyl donors and other nutrients known to be depleted in people who have liver fibrosis or liver cirrhosis. Enrolled patients included those with liver fibrosis stage F3 and F4 associated with various forms of CLD.

Successful reversal of liver fibrosis/cirrhosis in patient is a positive indication that the formula is successful in promoting homeostasis and nutritional sufficiency in people who have liver fibrosis or liver cirrhosis.

Introduction

Latest studies shows that depletion of dietary of methyl donors (one-carbon metabolism) is known to cause a cascade of metabolic disruptions that trigger liver fibrosis (1).

Studies show that depletion of dietary one-carbon methyl donors, including methyl tetrahydrofolate (mTHF), betaine, and folic acid cycle nutrients including L-glycine, vitamin B12, and vitamin B2 cause a chain of disruption in the metabolic pathways of methionine and choline metabolism.
These metabolic disturbances trigger the event called liver fibrosis.*

One-Carbon depletion is characterized by disruption of methionine and choline metabolism: depletion of S-adenosylmethionine (SAM), accumulation of both S-adenosylhomocysteine (SAH) and homocysteine (HCY), depletion of glutathione (GSH), depletion of phosphatidylcholine (PC), decreased methylation of homocysteine to methionine, decreased transsulfuration of homocysteine to cysteine, extreme oxidative stress and depletion of small molecule antioxidants. Science has shown that depletion of one-carbon methyl donors and the associated disruption of methionine metabolism triggers stellate cell conversion and the generation of liver fibrosis.

Patients with liver fibrosis also experience disruption in methionine (3), choline (4) and one-carbon metabolic pathways (5,6), leading to steatosis (7), hepatocellular injury (3), hepatocellular carcinoma (HCC) (8).

In fact, methionine deficient (MD) diets, choline deficient (CD) diets and methionine/choline deficient (MCD) diets have long been used to produce steatosis, hepatocellular injury and liver fibrosis in lab mice for animal liver studies. While the CD diet is associated with steatosis, the MD and MCD diets are associated with inflammation, hepatocellular injury and liver fibrosis (9,10). Methionine and choline metabolism are linked by one-carbon metabolism and folate deficient (FD) diets are studied separately and in conjunction with MD (MFD) and/or CD (CFD) diets to produce steatosis, liver fibrosis and HCC in animal studies (11-14).

Disruption of these closely-linked metabolic pathways increases the patient's metabolic demand for specific diet-derived nutrients involved in these pathways. Patients with liver fibrosis and liver cirrhosis experience depletion of key nutrient metabolites and biosynthetic end products of these metabolic pathways, including S-adenosylmethionine (SAM) (15-17), phosphatidylcholine (PC) (18-20), glutathione (GSH) (22,23). One carbon metabolites including folate, betaine, vitamin B6, B12, and folic acid cycle nutrients (23-24). Concurrent rise in S-adenosylhomocysteine (SAH) and homocysteine (HCY) is also seen (25).

The current longitudinal study is designed to document the possible decrease in hepatic collagen content (reversal of liver fibrosis) by administration of a nutritional repletion formula addressing known nutritional deficiencies that drive liver fibrosis and liver cirrhosis.

This formula consists of 31 GRAS (Generally Regarded As Safe by FDA) diet-derived nutrients associated with methionine, choline, and one-carbon metabolism. Glutathione depletion promotes oxidative stress and further depletion of small-molecule antioxidants. A complex blend of cooperative antioxidants is also provided to promote cellular and mitochondrial redox homeostasis.

Background

According to **Section 403(r)(6)(A) of the Federal Food, Drug & Cosmetic Act** certain nutritional deficiencies can cause what are known as nutritional deficiency diseases.*

Studies show that depletion of dietary of methyl donors (one-carbon metabolism) is known to create a cascade of metabolic disruptions that trigger liver fibrosis. This scientific concensus illustrates that the generation and progression of liver fibrosis is a nutritional deficiency disease per Section 403(r)(6)(A) of the Federal Food, Drug & Cosmetic Act.*

Latest studies show that depletion of dietary one-carbon methyl donors cause a chain of disruption in the metabolic pathways of methionine and choline metabolism in people with both Non-Alcoholic Steatohepatitis (NASH) (1,2) and Alcohol Liver Disease (ALD) (3,4). These metabolic disturbances trigger the event called progressive liver fibrosis (1,4,5,6) which is the excessive accumulation of scar tissue (collagen) in the extracellular spaces of the liver. Liver fibrosis was once considered irreversible but studies have shown that reversal is possible in people in which the underlying

fibrogenic impulse is eliminated – meaning their underlying chronic liver disease was cured (7,8,9,10). Examples are hepatitis B and hepatitis C patients who undergo antiviral therapy, obese people who undergo bariatric surgery, or forms of weight loss drugs. Studies have also shown that regression of liver fibrosis is possible even in people with deep cirrhosis (7). Once the fibrogenic trigger is eliminated collagenase enzymes called matrix metalloproteinases may resorb excess collagen from the extracellular matrix enzymatically (12).*

One-Carbon depletion is characterized by disruption of methionine metabolism: depletion of S-adenosylmetnionine (SAM) (13,14,15,16), accumulation of both S-adenosylhomocysteine (SAH) and homocysteine (HCY) (1,17) depletion of glutathione (GSH) (18,19), depletion of phosphatidylcholine (PC) (20,21,22), decreased methylation of homocysteine to methionine (1,2,3,4), decreased transsulfuration of homocysteine to cysteine (18,19), extreme oxidative stress (23,24)) and depletion of small molecule antioxidants (23,24).*

Science has shown that depletion of one-carbon methyl donors and the associated disruption of methionine metabolism triggers stellate cell conversion and the generation of liver fibrosis (1,2,3,4).*

PathFinder provides 34 specific key nutrients whose depletion causes and drives Liver Fibrosis and Liver Cirrhosis. This special patented formula is designed to promote healthy nutritional balance in these disrupted metabolic pathways (choline, methionine and one carbon metabolism (see below).*

HepAssure Inc.'s PathFinder formula has been **clinically studied** on people with advanced Liver Fibrosis and Liver Cirrhosis (please see our Clinical Studies above).* Between 5.5 million and 6.5 million people in the US have advanced Liver Fibrosis or Liver Cirrhosis (see below).*

PathFinder is a science-based nutritional management program that consists of 34 diet-derived nutrients involved in the linked Choline/Methionine/One-Carbon metabolic pathways.*
PathFinder also provides a broad spectrum of antioxidants to support the body's natural defenses against oxidative stress and mitochondrial nutrients to aid in energy production.*
Additionally, PathFinder includes amino acids, minerals, and botanical ingredients carefully formulated to support liver metabolism. The formula is designed to provide balanced amounts of specific nutrients while avoiding excessive amounts that could result in un-metabolized by-products.*

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4661801/

Materials and Methods

The present study is a longitudinal study in which we enrolled patients with chronic liver disease with liver fibrosis stage F3 and F4 by fibroscan.

Fibroscan is a non-invasive medical device used to assess liver fibrosis stage and can be used in place of biopsy to assess liver fibrosis stage. Fibroscan is accurate in differentiating between early stage liver fibrosis (stage F0-1 to F3) and stage F4 liver fibrosis (cirrhosis), the most advanced liver fibrosis stage (33).

Upon enrollment, patients signed Informed Consent Agreements and were given a bottle of nutritional formula containing a month's supply of capsules to be taken orally three times per day. Patients were instructed to eat the contents of one pouch (containing 8 capsules) in the morning, one pouch in the afternoon and one at night. Eight capsules eaten three times per day is a total of 24 capsules per day. Patients were requested to closely adhere to the three times per day schedule because an eight hour administration period approximates a metabolic cycle, and a this nutritional repletion formula is designed to prevent nutritional depletion throughout the whole day.

Patients were encouraged to try to improve their diets and to increase their activity levels, within the range of their physical limitations. Some patients reported decreased appetite and increased energy levels. However, no study patients lost a significantly large amount of weight. Compliance was self-reported and relied on the honor system. Therefore, compliance was assumed, but not verified.

Upon enrollment, fibroscan and routine lab blood tests, including liver function tests (LFTs) were performed. Fibroscans were repeated at least every six months; however most patients were given fibroscan evaluations monthly to every three months. Lab blood tests were repeated every three months, especially out-of-range results.

Lab blood tests were taken every three to six months. Most patients who have shown improvement in fibrosis staging also showed improvement in blood tests over time.

Results

Thirty two study patients were enrolled in one of three arms of the study and their results are reported here.

Results grouped by Disease Etiology

Reported results were data compiled from 32 study patients who had completed a minimum of six months on the protocol. Of the 32 study patients, 19 were patients with NASH, six were patients with ALD who were abstaining from alcohol, four patients had resolved HCV, two patients had unresolved HCV, one patient suffered from cholestatic primary biliary cholangitis (PBC) and one patient suffered from HIV but was not co-infected with HCV (see Table 1, page 12).

Results grouped by by Fibrosis Stage

Of the study patients with NASH, one patient started enrollment at liver fibrosis stage F2, 12 patients started enrollment at liver fibrosis stage F3 and three patients started at liver fibrosis stage F4 (cirrhosis). All but one of the patients who began the protocol with stage F3 NASH and the patient with stage F2 NASH finished the protocol at stage F1-F2 or better (see Table 2, page 29).

Of the 32 patients enrolled in the study, 3 patients began enrollment at liver fibrosis stage F2, 14 patients began at liver fibrosis stage F3 and 15 patients began enrollment at liver fibrosis stage F4 (cirrhosis).

Of the three patients who enrolled at liver fibrosis stage F2, all decreased their fibroscan scores down to liver fibrosis stage F0-F1 (normal).

Of the 14 patients who began enrollment at liver fibrosis stage F3, eight patients decreased their fibroscan stage back to F0-F1 (normal), while five patients decreased one stage to liver fibrosis stage F2. One patient remained in liver fibrosis stage F3.

Of the 15 patients who began enrollment in the study with liver fibrosis stage F4 (cirrhosis), 7 patients reversed at least one stage to F3. Of those patients, three patients ended with liver fibrosis stage F3, one patient finished at liver fibrosis stage F2, two patients decreased to liver fibrosis stage F1-F2 (between 7.0 kPa-7.5 kPa), and one patient ended with liver fibrosis F0-F1 (normal).

Of the eight patients who did not reverse out of cirrhosis (≤14kPa), six patients nonetheless experienced impressive numerical decreases (≥30%) in their fibroscan kPa values, while two patients did not respond, but did not progress. Many of the study patients who experienced extremely large decreases but still did not technically reverse out of Stage F4 (cirrhosis) began enrollment with extremely high kPa values.

On the fibroscan machine, liver fibrosis stage F4 (cirrhosis) begins at around 12.0-14.0 kPa but goes up to 75 kPa, which is the maximum reading on the fibroscan machine. Therefore stage F4, cirrhosis is a very long and complicated stage of liver fibrosis. Many of the study patients (see above) experienced extremely large decreases in the numerical fibroscan values, but still did not technically reverse out of Stage F4 (cirrhosis).

Of the three patients with NASH who began the protocol at liver fibrosis stage F4, two reversed out of stage F4 to stage F3, while the third stage F4 study patient showed a significant decrease in pKa scores from 27 pKa to 11.0 kPa, but remained technically classified as liver fibrosis stage F4 (see Table 3, page 35).

All six of the ALD study patients began enrollment with liver fibrosis stage F4 and showed impressive improvement (see Table 4). Five of the six Arm 3 patients began enrollment with fibroscan pKa values between 27.0 and 48.8, indicating mid-range cirrhosis (stage F4). The sixth patient began enrollment with a fibroscan pKa value of 63.2 kPa. However, the patient's fibroscan readings decreased to 42.1 after a TIPS shunt procedure was performed to decrease portal hypertension shortly after enrollment.

All ALD patients experienced a sizable (≥30%) decrease in fibroscan kPa scores. One patient decreased from fibrosis stage F4 (27.0 kPa) to fibrosis stage F1-F2 (7.0 kPa). The other five Arm 3 patients experienced large decreases in numerical fibroscan pKa values by the end of the study period but still remained technically classified as liver fibrosis stage F4 (see Table 4, page 37). Duration on the protocol varied from patient to patient. Four of the original Arm 3 patients are now on a follow-up protocol with the goal of further decreasing fibrosis staging over time. Those results are not reported.

Several ALD study patients had pre-existing ascites and/or banded esophageal varices. No study patients experienced ascites or esophageal bleeding while enrolled in the study.

Four patients who had successfully treated for HCV were enrolled. One patient decreased fibrosis score from stage F4 (16.9 kPa) to stage F0-F1 (7.3 kPa), two patients experienced moderate decrease from stage F3 to stage F2 (10.3 kPa to 8.3 kPa and 13.9 kPa to 9.1 kPa), while the third study patient began at stage F4 with a fibroscan score of 19.4 kPa and ended at 20.4 kPa, showing no positive net effect (see Table 5, page 41).

Two patients with unresolved HCV infection were enrolled in Arm 1. One study patient experienced a decrease in fibrosis stage by fibroscan from stage F4 (15.3 kPa) to stage F0-F1 (6.1 kPa). The other study patient experienced an initial decrease in fibroscan scores from 37.5 kPa to 26.4 kPa but appeared to plateau afterwards with ending scores reflecting a value of 26.3 kPa. These two patients included a patient who did not have insurance coverage and a patient who had co-morbidities that excluded patients from receiving treatment. Eventually both untreated HCV study patients did receive treatment for HCV (see Table 6, page 46).

One patient with cholestatic primary biliary cholangitis (PBC) and who was concurrently treated with obeticholic acid showed a decrease from liver fibrosis stage F3 (13.0 kPa) to F2 (8.6 kPa) (see Table 1, page 12).

Compliance with diet and/or alcohol use was self-reported and therefore, assumed but not verified. Alcohol abuse the night before a fibroscan may elevate scores the next day due to acute inflammation, while sugar intake for NASH patients may increase oxidative stress, lipotoxicity, increase fibrogenic activity and promote progression of liver fibrosis. Weight gain in NASH patients might be assumed to be associated with increased fibrogenesis, especially in obese patients.

While adverse event reporting was in-place and patients were monitored on a monthly basis, no enrolled patients reported adverse events associated with the medical food.

Interestingly, the six Arm 3 patients reported no problems (cravings, relapse, etc.) with alcohol compliance while enrolled in the study. Some commented that their doctors were surprised to learn that they had no cravings and apparently were not tempted to drink alcohol.

Discussion

The present study is designed to investigate whether a nutritional repletion program can promote the reversal of liver fibrosis in patients.

The study results do not show reversal of liver fibrosis in all study patients. However, the majority of study patients who completed at least six months of the protocol did show improvement in liver fibrosis biomarkers, including Fibroscans, indicating that a nutritional repletion program may have a profoundly positive effect on liver fibrosis and liver cirrhosis, as well as on metabolic disruption common in patients with CLD.

This study investigates whether a nutritional repletion program can promote reversal of liver fibrosis and liver cirrhosis biomarkers in patients with CLD. Past studies have shown that reversal of fibrosis may occur naturally in patients when the fibrogenic trigger is eliminated or successfully treated.

However, elimination or decrease of the fibrogenic trigger (as in obese patients on Semaglutide) may result in liver histology improvement, but nutritional insufficiency status is still unknown. The present study explores whether a nutritional repletion program can help promote reversal of liver fibrosis in patients with resolved and unresolved CLD.

Further, lack of previous studies involving ALD leave the question of reversal of liver fibrosis in compliant patients with liver cirrhosis (liver fibrosis stage F4) as an open question. It is widely assumed that there is a point of no return, past which reversal of liver cirrhosis may prove to be impossible.

In this study we enrolled a number of patients who were abstaining from alcohol who initially demonstrated mid-to-deep range cirrhosis at the time of enrollment. Several of these patients had previously experienced ascites and/or bleeding esophageal varices, which had been banded. All six of these patients showed improvement and their lab blood tests were generally normalized.

For hepatic fibrosis reversal to occur in on-going chronic liver disease it would seem logical that the fibrogenic impulse must be down-regulated or eliminated to a large degree.

It is reasonable to assume that regarding reversal of liver cirrhosis there is a “point of no return” after which successful reversal of liver fibrosis may be impossible. In the results of the present study, reversal of liver fibrosis was successful in almost all stage F3 patients, while many stage F4 patients experienced very positive results, but some did not.

The limitations of this study are obvious: there is no control group, no placebo and no randomization. Therefore it is impossible to deduce comparatively how often the medical food works compared to control. The fibroscan scores were performed in-

house, rather than by an independent third-party clinic. Because this is an on-going, longitudinal, in-house study these patient results have not been IRB-reviewed or published in a peer review journal. The purpose of the study in its current form is to inform consent study patients and to ensure consistency in study protocol while compiling individual case reports and data for further analysis.

Based on our results a further randomized, placebo-controlled study is necessary.

Please scroll down to see the Study Patient fibroscan results.

Tables

Table 1

Table 1 consists of 32 study patients who completed at least six months in the HepAssure medical food study.

	Enrollment Date	Type of CLD	Date	Fibroscan Result	Fibrosis Stage	Reference Range
Patient 1	12/14/2015	NASH				
Fibroscan 1 (initial)			12/14/15	**9.9** kPa	F3	10.0-14.0 kPa
Fibroscan 2			3/1/16	**4.5** kPa	F0-F1	<7.0 kPa
Fibroscan 3			6/10/16	**8.2** kPa	F2	7.5-10.0 kPa
Patient 2	2/24/2016	NASH				
Fibroscan 1 (initial)			2/24/16	**10.6** kPa	F3	10.0-14.0 kPa
Fibroscan 2			9/28/16	**6.6** kPa	F0-F1	<7.0 kPa

Patient 3	3/1/2016	Resolved HCV				
Fibroscan 1 (initial)			3/1/16	**16.9** kPa	F4	>12.0 kPa
Fibroscan 2			6/1/16	**12.0** kPa	F4	>12.0 kPa
Fibroscan 3			9/7/16	**10.2** kPa	F3	9.5-12.0 kPa
Fibroscan 4			12/8/16	**9.8** kPa	F3	9.5-12.0 kPa
Fibroscan 5			3/6/17	**7.3** kPa	F1-F2	7.0-7.5 kPa

Patient 4	3/25/2015	NASH				
Fibroscan 2			10/18/15	**6.9** kPa	F0-F1	<7.0 kPa
Fibroscan 3			3/4/16	**4.1** kPa	F0-F1	<7.0 kPa
Patient 5	1/15/2016	ALD				
Fibroscan 1 (initial)			1/15/16	**27.0** kPa	F4	>12.5 kPa
Fibroscan 2			4/14/16	**19.2** kPa	F4	>12.5 kPa
Fibroscan 3			7/16/16	**14.0** kPa	F3	9.5-12.5 kPa
Fibroscan 4			11/3/16	**11.6** kPa	F3	9.5-12.5 kPa
Fibroscan 5			2/3/17	**10.3** kPa	F3	9.5-12.5 kPa
Fibroscan 6			4/6/18	**7.0** kPa	F1-F2	<7.0 kPa
Patient 6	3/20/2015	Un-resolved HCV				
Fibroscan 1 (initial)			3/20/15	**15.3** kPa	F4	>12.0 kPa

Fibroscan 2			11/23/16	**9.2** kPa	F2	7.0-9.5 kPa
Fibroscan 3			4/27/16	**6.1** kPa	F0-F1	<7.0 kPa
Patient 7	12/22/2015	NASH				
Fibroscan 1 (initial)			12/22/15	**11.2** kPa	F3	10.0-14.0 kPa
Fibroscan 2			3/1/16	**4.6** kPa	F0-F1	<7.0 kPa
Fibroscan 3			7/26/16	**4.7** kPa	F0-F1	<7.0 kPa
Patient 8	12/21/2015	NASH				
Fibroscan 1 (initial)			12/21/15	**8.7** kPa	F2	7.5-10.0 kPa
Fibroscan 2			3/1/16	**6.4** kPa	F0-F1	<7.0 kPa
Fibroscan 3			8/3/16	**4.5** kPa	F0-F1	<7.0 kPa
Patient 9	2/5/2016	NASH				
Fibroscan 1 (initial)			2/3/16	**9.2** kPa	F2	7.5-10.0 kPa

Fibroscan 2			5/20/16	**4.7** kPa	F0-F1	<7.0 kPa
Fibroscan 3			9/9/16	**5.6** kPa	F0-F1	<7.0 kPa
Patient 10	1/29/2016	NASH				
Fibroscan 1 (initial)			1/29/16	**10.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			6/2/16	**6.0** kPa	F0-F1	<7.0 kPa
Fibroscan 3			8/8/16	**6.2** kPa	F0-F1	<7.0 kPa
Patient 11	5/9/2016	NASH				
Fibroscan 1 (initial)			5/9/16	**10.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			8/26/16	**6.1** kPa	F0-F1	<7.0 kPa
Fibroscan 3			12/6/16	**5.2** kPa	F0-F1	<7.0 kPa
Patient 12	4/22/2016	NASH				
Fibroscan 1 (initial)			4/22/16	**16.0** kPa	F4	>14.0 kPa
Fibroscan 2			7/25/16	**11.5** kPa	F3	10.0-14.0 kPa
Fibroscan 3			10/28/16	**9.4** kPa	F2	7.5-10.0 kPa
Fibroscan 4			1/30/17	**11.0** kPa	F3	10.0-14.0 kPa

Patient 13	4/22/2016	NASH				
Fibroscan 1 (initial)			4/22/16	**8.7** kPa	F2	7.5-10.0 kPa
Fibroscan 2			7/25/16	**4.3** kPa	F0-F1	<7.0 kPa
Fibroscan 3			10/28/16	**4.3** kPa	F0-F1	<7.0 kPa
Patient 14	8/2/2016	NASH				
Fibroscan 1 (initial)			8/2/16	**11.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			10/27/16	**5.9** kPa	F0-F1	<7.0 kPa
Fibroscan 3			1/31/17	**5.3** kPa	F0-F1	<7.0 kPa
Patient 15	7/25/2016	NASH				
Fibroscan 1 (initial)			7/21/16	**11.2** kPa	F3	10.0-14.0 kPa
Fibroscan 2			11/14/16	**7.7** kPa	F2	7.5-10.0 kPa
Fibroscan 3			2/7/17	**5.5** kPa	F0-F1	<7.0 kPa

Patient 16	8/8/2016	NASH				
Fibroscan 1 (initial)			8/3/16	**13.4** kPa	F3	10.0-14.0 kPa
Fibroscan 2			11/7/16	**14.0** kPa	F4	>14.0 kPa
Fibroscan 3			1/11/17	**12.0** kPa	F3	10.0-14.0 kPa
Fibroscan 4			2/8/17	**9.7** kPa	F2	7.5-10.0 kPa
Fibroscan 5			4/13/17	**9.9** kPa	F2	7.5-10.0 kPa
Fibroscan 6			7/11/2017	**10.6** kPa	F3	10.0-14.0 kPa
Patient 17	7/25/2016	Resolved HCV				
Fibroscan 1 (initial)			7/25/16	**10.3** kPa	F3	10.0-14.0 kPa
Fibroscan 2			9/23/16	**8.1** kPa	F2	7.5-10.0 kPa
Fibroscan 3			11/2/16	**6.7** kPa	F0-F1	<7.0 kPa
Fibroscan 4			2/3/17	**8.0** kPa	F2	7.5-10.0 kPa
Patient 18	9/6/2016	NASH				
Fibroscan 1 (initial)			8/31/16	**13.6** kPa	F3	10.0-14.0 kPa

Fibroscan 2			12/6/16	**7.9** kPa	F2	7.5-10.0 kPa
Fibroscan 3			4/5/17	**4.2** kPa	F0-F1	<7.0 kPa
Patient 19	6/23/2016	Resolved HCV				
Fibroscan 1 (initial)			9/23/16	**19.4** kPa	F4	>12.0 kPa
Fibroscan 2			11/22/16	**23.4** kPa	F4	>12.0 kPa
Fibroscan 3			1/11/16	**23.0** kPa	F4	>12.0 kPa
Fibroscan 4			3/15/17	**27.7** kPa	F4	>12.0 kPa
Fibroscan 5			4/19/17	**21.3** kPa	F4	>12.0 kPa
Fibroscan 6			7/19/17	**20.4** kPa	F4	>12.0 kPa
Patient 20	10/19/2016	PBC				
Fibroscan 1 (initial)			9/8/16	**13.0** kPa	F3	10.0-17.0 kPa
Fibroscan 2			12/21/16	**12.4** kPa	F3	10.0-17.0 kPa
Fibroscan 3			1/26/17	**10.0** kPa	F2	7.5-10.0 kPa
Fibroscan 4			2/24/17	**12.0** kPa	F3	10.0-17.0 kPa
Fibroscan 5			3/23/17	**11.5** kPa	F3	10.0-17.0 kPa
Fibroscan			4/28/17	**12.3** kPa	F3	10.0-17.0 kPa

6						
Fibroscan 7			7/7/17	**8.8** kPa	F2	7.5-10.0 kPa
Fibroscan 8			8/11/17	**8.6** kPa	F2	7.5-10.0 kPa
Patient 21	2/26/2016	NASH				
Fibroscan 1 (initial)			2/26/16	**27.4** kPa	F4	>14.0 kPa
Fibroscan 2			5/20/16	**23.5** kPa	F4	>14.0 kPa
Fibroscan 3			8/29/16	**21.2** kPa	F4	>14.0 kPa
Fibroscan 4			12/14/16	**13.2** kPa	F3	10.0-14.0 kPa
Fibroscan 5			3/13/17	**15.4** kPa	F4	>14.0 kPa
Fibroscan 6			7/21/17	**16.5** kPa	F4	>14.0 kPa
Patient 22	11/17/2016	NASH				
Fibroscan 1 (initial)			11/7/16	**13.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			1/31/17	**8.1** kPa	F2	7.5-10.0 kPa
Fibroscan 3			5/15/17	**8.6** kPa	F2	7.5-10.0 kPa

Patient 23	12/16/2015	ALD				
Fibroscan 1 (initial)			12/16/15	**48.8** kPa	F4	>12.5 kPa
Fibroscan 2			4/18/16	**25.1** kPa	F4	>12.5 kPa
Fibroscan 3			9/14/16	**24.9** kPa	F4	>12.5 kPa
Fibroscan 4			1/17/17	**24.4** kPa	F4	>12.5 kPa
Fibroscan 5			5/31/18	**22.7** kPa	F4	>12.5 kPa
Fibroscan 6			10/23/18	**20.1** kPa	F4	>12.5 kPa
Fibroscan 7			4/25/19	**21.7** kPa	F4	>12.5 kPa
Patient 24	10/20/2016	ALD				
Fibroscan 1 (initial)			10/18/16	**40.8** kPa	F4	>12.5 kPa
Fibroscan 2			11/17/16	**37.6** kPa	F4	>12.5 kPa
Fibroscan 3			12/15/16	**29.0** kPa	F4	>12.5 kPa
Fibroscan 4			1/12/17	**29.2** kPa	F4	>12.5 kPa
Fibroscan 5			2/17/17	**24.7** kPa	F4	>12.5 kPa
Fibroscan 6			3/23/17	**21.0** kPa	F4	>12.5 kPa

Fibroscan 7			4/19/17	**18.2** kPa	F4	>12.5 kPa
Fibroscan 8			7/7/17	**19.9** kPa	F4	>12.5 kPa
Patient 25	9/20/2016	ALD				
Fibroscan 1 (initial)			9/20/16	**33.8** kPa	F4	>12.5 kPa
Fibroscan 2			1/9/17	**41.2** kPa	F4	>12.5 kPa
Fibroscan 3			3/21/17	**44.2** kPa	F4	>12.5 kPa
Fibroscan 4			5/3/17	**41.1** kPa	F4	>12.5 kPa
Fibroscan 5			9/29/17	**27.8** kPa	F4	>12.5 kPa
Fibroscan 6			11/7/17	**29.6** kPa	F4	>12.5 kPa
Fibroscan 7			5/7/18	**30.4** kPa	F4	>12.5 kPa
Fibroscan 8			8/7/18	**21.4** kPa	F4	>12.5 kPa
Fibroscan 9			12/11/18	**24.5** kPa	F4	>12.5 kPa
Fibroscan 10			2/25/19	**18.2** kPa	F4	>12.5 kPa
Fibroscan 11			5/20/19	**18.1** kPa	F4	>12.5 kPa

Patient 26	9/22/2016	Un-resolved HCV				
Fibroscan 1 (initial)			9/22/16	**37.5** kPa	F4	>12.5 kPa
Fibroscan 2			11/7/16	**26.4** kPa	F4	>12.5 kPa
Fibroscan 3			12/14/16	**28.0** kPa	F4	>12.5 kPa
Fibroscan 4			1/31/17	**24.7** kPa	F4	>12.5 kPa
Fibroscan 5			4/5/17	**21.6** kPa	F4	>12.5 kPa
Fibroscan 6			5/15/17	**27.1** kPa	F4	>12.5 kPa
Fibroscan 7			7/24/17	**25.4** kPa	F4	>12.5 kPa
Fibroscan 8			9/29/17	**20.1** kPa	F4	>12.5 kPa
Fibroscan 9			11/17/17	**27.8** kPa	F4	>12.5 kPa
Fibroscan 10			1/19/18	**25.4** kPa	F4	>12.5 kPa
Fibroscan 11			3/16/18	**34.8** kPa	F4	>12.5 kPa
Fibroscan 12			5/14/18	**26.3** kPa	F4	>12.5 kPa
Patient 27	12/20/2016	NASH				
Fibroscan 1 (initial)			12/20/16	**19.8** kPa	F4	>14.0 kPa
Fibroscan 2			2/20/17	**14.2** kPa	F4	>14.0 kPa
Fibroscan 3			4/4/17	**13.7** kPa	F3	10.0-14.0 kPa

Fibroscan 4			6/6/17	**12.4** kPa	F3	10.0-14.0 kPa
Fibroscan 5			7/21/17	**13.3** kPa	F3	10.0-14.0 kPa
Fibroscan 6			8/29/17	**10.7** kPa	F3	10.0-14.0 kPa
Fibroscan 7			10/16/17	**10.4** kPa	F3	10.0-14.0 kPa
Fibroscan 8			12/26/17	**10.1** kPa	F3	10.0-14.0 kPa
Patient 28	4/12/2018	HIV				
Fibroscan 1 (initial)			4/12/18	**22.7** kPa	F4	>14.0 kPa
Fibroscan 2			6/4/18	**17.1** kPa	F4	>14.0 kPa
Fibroscan 3			10/19/18	**13.7** kPa	F3	>14.0 kPa
Fibroscan 4			12/20/18	**12.8** kPa	F3	>14.0 kPa
Fibroscan 5			3/7/19	**11.8** kPa	F3	>14.0 kPa
Fibroscan 6			5/14/19	**10.7** kPa	F3	>14.0 kPa
Patient 29	3/13/2017	ALD-TIPS Shunt				
Fibroscan 1 (initial)		Pre-TIPS Shunt	2/14/17	**63.2** kPa	F4	>12.5 kPa

Fibroscan 2		Post-TIPS Shunt	8/2/17	**42.1** kPa	F4	>12.5 kPa
Fibroscan 3			12/14/17	**31.3** kPa	F4	>12.5 kPa
Fibroscan 4			4/26/18	**32.4** kPa	F4	>12.5 kPa
Fibroscan 5			6/4/18	**20.1** kPa	F4	>12.5 kPa
Fibroscan 6			7/23/18	**27.2** kPa	F4	>12.5 kPa
Fibroscan 7			9/4/18	**25.6** kPa	F4	>12.5 kPa
Fibroscan 8			10/31/18	**22.3** kPa	F4	>12.5 kPa
Fibroscan 9			12/20/18	**19.4** kPa	F4	>12.5 kPa
Fibroscan 10			2/14/19	**22.6** kPa	F4	>12.5 kPa
Fibroscan 11			3/28/19	**21.9** kPa	F4	>12.5 kPa
Fibroscan 12			5/16/19	**18.1** kPa	F4	>12.5 kPa

Patient 30	7/27/2017	ALD				
Fibroscan 1 (initial)			7/27/17	**43.9** kPa	F4	>12.5 kPa
Fibroscan 2			9/29/17	**39.6** kPa	F4	>12.5 kPa
Fibroscan 3			10/27/17	**40.5** kPa	F4	>12.5 kPa
Fibroscan 4			11/28/17	**34.5** kPa	F4	>12.5 kPa
Fibroscan 5			3/28/18	**26.9** kPa	F4	>12.5 kPa
Fibroscan 6			4/26/18	**29.2** kPa	F4	>12.5 kPa
Fibroscan 7			5/24/18	**25.7** kPa	F4	>12.5 kPa
Fibroscan 8			6/27/18	**32.8** kPa	F4	>12.5 kPa
Fibroscan 9			7/24/18	**27.4** kPa	F4	>12.5 kPa
Fibroscan 10			8/22/18	**28.7** kPa	F4	>12.5 kPa
Fibroscan 11			10/24/18	**24.5** kPa	F4	>12.5 kPa
Fibroscan 12			11/23/18	**26.6** kPa	F4	>12.5 kPa
Patient 31	3/3/2017	NASH				
Fibroscan 1 (initial)			3/3/17	**11.6** kPa	F3	10.0-14.0 kPa
Fibroscan 2			8/23/17	**8.9** kPa	F2	7.5-10.0 kPa

Fibroscan 3			11/30/17	**10.1** kPa	F2	10.0-14.0 kPa
Fibroscan 4			4/23/18	**8.9** kPa	F2	7.5-10.0 kPa
Fibroscan 5			1/23/19	**9.9** kPa	F2	7.5-10.0 kPa
Patient 32	6/5/2017	Resolved HCV				
Fibroscan 1 (initial)			6/5/17	**13.9** kPa	F4	>12.5 kPa
Fibroscan 2			7/7/17	**9.2** kPa	F2	7.5-10.0 kPa
Fibroscan 3			8/3/17	**10.6** kPa	F3	10.0-14.0 kPa
Fibroscan 4			10/5/17	**8.4** kPa	F2	7.5-10.0 kPa
Fibroscan 5			12/6/17	**15.1** kPa	F4	>12.5 kPa
Fibroscan 6			2/19/18	**11.8** kPa	F3	10.0-14.0 kPa
Fibroscan 7			5/17/18	**10.9** kPa	F3	10.0-14.0 kPa
Fibroscan 8			10/31/18	**9.1** kPa	F2	7.5-10.0 kPa

Table 2 consists of 12 study patients with NASH and fibrosis stage F2 and F3 who completed at least six months in the HepAssure medical food study.

	Enrollment Date	Type of CLD	Date	Fibroscan Result	Fibrosis Stage	Reference Range
Patient 1	12/14/2015	NASH				
Fibroscan 1 (initial)			12/14/15	**9.9** kPa	F3	10.0-14.0 kPa
Fibroscan 2			3/1/16	**4.5** kPa	F0-F1	<7.0 kPa
Fibroscan 3			6/10/16	**8.2** kPa	F2	7.5-10.0 kPa
Patient 2	2/24/2016	NASH				
Fibroscan 1 (initial)			2/24/16	**10.6** kPa	F3	10.0-14.0 kPa
Fibroscan 2			9/28/16	**6.6** kPa	F0-F1	<7.0 kPa
Patient 4	3/25/2015	NASH				
Fibroscan 1 (initial)			3/25/15	**12.1** kPa	F3	10.0-14.0 kPa
Fibroscan 2			10/18/15	**6.9** kPa	F0-F1	<7.0 kPa
Fibroscan 3			3/4/16	**4.1** kPa	F0-F1	<7.0 kPa
Patient 7	12/22/2015	NASH				
Fibroscan			12/22/15	**11.2** kPa	F3	10.0-14.0

1 (initial)						kPa
Fibroscan 2			3/1/16	**4.6** kPa	F0-F1	<7.0 kPa
Fibroscan 3			7/26/16	**4.7** kPa	F0-F1	<7.0 kPa
Patient 10	1/29/2016	NASH				
Fibroscan 1 (initial)			1/29/16	**10.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			6/2/16	**6.0** kPa	F0-F1	<7.0 kPa
Fibroscan 3			8/8/16	**6.2** kPa	F0-F1	<7.0 kPa
Patient 11	5/9/2016	NASH				
Fibroscan 1 (initial)			5/9/16	**10.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			8/26/16	**6.1** kPa	F0-F1	<7.0 kPa
Fibroscan 3			12/6/16	**5.2** kPa	F0-F1	<7.0 kPa
Patient 14	8/2/2016	NASH				
Fibroscan 1 (initial)			8/2/16	**11.0** kPa	F3	10.0-14.0 kPa
Fibroscan			10/27/16	**5.9** kPa	F0-F1	<7.0 kPa

2						
Fibroscan 3			1/31/17	**5.3** kPa	F0-F1	<7.0 kPa
Patient 15	7/25/2016	NASH				
Fibroscan 1 (initial)			7/21/16	**11.2** kPa	F3	10.0-14.0 kPa
Fibroscan 2			11/14/16	**7.7** kPa	F2	7.5-10.0 kPa
Fibroscan 3			2/7/17	**5.5** kPa	F0-F1	<7.0 kPa
Patient 16	8/8/2016	NASH				
Fibroscan 1 (initial)			8/3/16	**13.4** kPa	F3	10.0-14.0 kPa
Fibroscan 2			11/7/16	**14.0** kPa	F4	>14.0 kPa
Fibroscan 3			1/11/17	**12.0** kPa	F3	10.0-14.0 kPa
Fibroscan 4			2/8/17	**9.7** kPa	F2	7.5-10.0 kPa
Fibroscan 5			4/13/17	**9.9** kPa	F2	7.5-10.0 kPa
Fibroscan 6			7/11/2017	**10.6** kPa	F3	10.0-14.0 kPa

Patient 18	9/6/2016	NASH				
Fibroscan 1 (initial)			8/31/16	**13.6** kPa	F3	10.0-14.0 kPa
Fibroscan 2			12/6/16	**7.9** kPa	F2	7.5-10.0 kPa
Fibroscan 3			4/5/17	**4.2** kPa	F0-F1	<7.0 kPa
Patient 22	11/17/2016	NASH				
Fibroscan 1 (initial)			11/7/16	**13.0** kPa	F3	10.0-14.0 kPa
Fibroscan 2			1/31/17	**8.1** kPa	F2	7.5-10.0 kPa
Fibroscan 3			5/15/17	**8.6** kPa	F2	7.5-10.0 kPa
Patient 31	3/3/2017	NASH				
Fibroscan 1 (initial)			3/3/17	**11.6** kPa	F3	10.0-14.0 kPa

Fibroscan 2			8/23/17	**8.9** kPa	F2	7.5-10.0 kPa
Fibroscan 3			11/30/17	**10.1** kPa	F2	10.0-14.0 kPa
Fibroscan 4			4/23/18	**8.9** kPa	F2	7.5-10.0 kPa
Fibroscan 5			1/23/19	**9.9** kPa	F2	7.5-10.0 kPa

Table 3

Table 3 consists of 3 study patients with stage F4 NASH who completed at least six months in the HepAssure medical food study.

	Enrollment Date	Type of CLD	Date	Fibroscan Result	Fibrosis Stage	Reference Range
Patient 12	4/22/2016	NASH				
Fibroscan 1 (initial)			4/22/16	**16.0** kPa	F4	>14.0 kPa
Fibroscan 2			7/25/16	**11.5** kPa	F3	10.0-14.0 kPa
Fibroscan 3			10/28/16	**9.4** kPa	F2	7.5-10.0 kPa
Fibroscan 4			1/30/17	**11.0** kPa	F3	10.0-14.0 kPa

Patient 21	2/26/2016	NASH				
Fibroscan 1 (initial)			2/26/16	**27.4** kPa	F4	>14.0 kPa
Fibroscan 2			5/20/16	**23.5** kPa	F4	>14.0 kPa
Fibroscan 3			8/29/16	**21.2** kPa	F4	>14.0 kPa
Fibroscan 4			12/14/16	**13.2** kPa	F3	10.0-14.0 kPa
Fibroscan 5			3/13/17	**15.4** kPa	F4	>14.0 kPa
Fibroscan 6			7/21/17	**16.5** kPa	F4	>14.0 kPa
Patient 27	12/20/2016	NASH				
Fibroscan 1 (initial)			12/20/16	**19.8** kPa	F4	>14.0 kPa
Fibroscan 2			2/20/17	**14.2** kPa	F4	>14.0 kPa
Fibroscan 3			4/4/17	**13.7** kPa	F3	10.0-14.0 kPa
Fibroscan 4			6/6/17	**12.4** kPa	F3	10.0-14.0 kPa
Fibroscan 5			7/21/17	**13.3** kPa	F3	10.0-14.0 kPa
Fibroscan 6			8/29/17	**10.7** kPa	F3	10.0-14.0 kPa
Fibroscan 7			10/16/17	**10.4** kPa	F3	10.0-14.0 kPa

Fibroscan 8			12/26/17	**10.1** kPa	F3	10.0-14.0 kPa

Table 4

Table 4 consists of 6 study patients with stage F4 ASH who completed at least six months in the HepAssure medical food study.

	Enrollment Date	Type of CLD	Date	Fibroscan Result	Fibrosis Stage	Reference Range
Patient 5	1/15/2016	ALD				
Fibroscan 1 (initial)			1/15/16	**27.0** kPa	F4	>12.5 kPa
Fibroscan 2			4/14/16	**19.2** kPa	F4	>12.5 kPa
Fibroscan 3			7/16/16	**14.0** kPa	F3	9.5-12.5 kPa
Fibroscan 4			11/3/16	**11.6** kPa	F3	9.5-12.5 kPa
Fibroscan 5			2/3/17	**10.3** kPa	F3	9.5-12.5 kPa
Fibroscan 6			4/6/18	**7.0** kPa	F1-F2	<7.0 kPa
Patient	12/16/2015	ALD				

23						
Fibroscan 1 (initial)			12/16/15	**48.8** kPa	F4	>12.5 kPa
Fibroscan 2			4/18/16	**25.1** kPa	F4	>12.5 kPa
Fibroscan 3			9/14/16	**24.9** kPa	F4	>12.5 kPa
Fibroscan 4			1/17/17	**24.4** kPa	F4	>12.5 kPa
Fibroscan 5			5/31/18	**22.7** kPa	F4	>12.5 kPa
Fibroscan 6			10/23/18	**20.1** kPa	F4	>12.5 kPa
Fibroscan 7			4/25/19	**21.7** kPa	F4	>12.5 kPa
Patient 24	10/20/2016	ALD				
Fibroscan 1 (initial)			10/18/16	**40.8** kPa	F4	>12.5 kPa
Fibroscan 2			11/17/16	**37.6** kPa	F4	>12.5 kPa
Fibroscan 3			12/15/16	**29.0** kPa	F4	>12.5 kPa
Fibroscan 4			1/12/17	**29.2** kPa	F4	>12.5 kPa
Fibroscan 5			2/17/17	**24.7** kPa	F4	>12.5 kPa
Fibroscan 6			3/23/17	**21.0** kPa	F4	>12.5 kPa

Fibroscan 7			4/19/17	**18.2** kPa	F4	>12.5 kPa
Fibroscan 8			7/7/17	**19.9** kPa	F4	>12.5 kPa
Patient 25	9/20/2016	ALD				
Fibroscan 1 (initial)			9/20/16	**33.8** kPa	F4	>12.5 kPa
Fibroscan 2			1/9/17	**41.2** kPa	F4	>12.5 kPa
Fibroscan 3			3/21/17	**44.2** kPa	F4	>12.5 kPa
Fibroscan 4			5/3/17	**41.1** kPa	F4	>12.5 kPa
Fibroscan 5			9/29/17	**27.8** kPa	F4	>12.5 kPa
Fibroscan 6			11/7/17	**29.6** kPa	F4	>12.5 kPa
Fibroscan 7			5/7/18	**30.4** kPa	F4	>12.5 kPa
Fibroscan 8			8/7/18	**21.4** kPa	F4	>12.5 kPa
Fibroscan 9			12/11/18	**24.5** kPa	F4	>12.5 kPa
Fibroscan 10			2/25/19	**18.2** kPa	F4	>12.5 kPa
Fibroscan 11			5/20/19	**18.1** kPa	F4	>12.5 kPa

Patient 30	7/27/2017	ALD				
Fibroscan 1 (initial)			7/27/17	**43.9** kPa	F4	>12.5 kPa
Fibroscan 2			9/29/17	**39.6** kPa	F4	>12.5 kPa
Fibroscan 3			10/27/17	**40.5** kPa	F4	>12.5 kPa
Fibroscan 4			11/28/17	**34.5** kPa	F4	>12.5 kPa
Fibroscan 5			3/28/18	**26.9** kPa	F4	>12.5 kPa
Fibroscan 6			4/26/18	**29.2** kPa	F4	>12.5 kPa
Fibroscan 7			5/24/18	**25.7** kPa	F4	>12.5 kPa
Fibroscan 8			6/27/18	**32.8** kPa	F4	>12.5 kPa
Fibroscan 9			7/24/18	**27.4** kPa	F4	>12.5 kPa

Fibroscan 10			8/22/18	28.7 kPa	F4	>12.5 kPa
Fibroscan 11			10/24/18	24.5 kPa	F4	>12.5 kPa
Fibroscan 12			11/23/18	26.6 kPa	F4	>12.5 kPa

Table 5

Table 5 consists of 4 study patients with stage F4 resolved HCV who completed at least six months in the HepAssure medical food study.

	Enrollment Date	Type of CLD	Date	Fibroscan Result	Fibrosis Stage	Reference Range
Patient 3	3/1/2016	Resolved HCV				
Fibroscan 1 (initial)			3/1/16	16.9 kPa	F4	>12.0 kPa
Fibroscan 2			6/1/16	12.0 kPa	F4	>12.0 kPa
Fibroscan 3			9/7/16	10.2 kPa	F3	9.5-12.0 kPa
Fibroscan 4			12/8/16	9.8 kPa	F3	9.5-12.0 kPa
Fibroscan 5			3/6/17	7.3 kPa	F1-F2	7.0-7.5 kPa
Patient 17	7/25/2016	Resolved HCV				
Fibroscan 1 (initial)			7/25/16	10.3 kPa	F3	10.0-14.0 kPa
Fibroscan 2			9/23/16	8.1 kPa	F2	7.5-10.0 kPa

Fibroscan 3			11/2/16	**6.7** kPa	F0-F1	<7.0 kPa
Fibroscan 4			2/3/17	**8.0** kPa	F2	7.5-10.0 kPa
Patient 19	6/23/2016	Resolved HCV				
Fibroscan 1 (initial)			9/23/16	**19.4** kPa	F4	>12.0 kPa
Fibroscan 2			11/22/16	**23.4** kPa	F4	>12.0 kPa
Fibroscan 3			1/11/16	**23.0** kPa	F4	>12.0 kPa
Fibroscan 4			3/15/17	**27.7** kPa	F4	>12.0 kPa
Fibroscan 5			4/19/17	**21.3** kPa	F4	>12.0 kPa
Fibroscan 6			7/19/17	**20.4** kPa	F4	>12.0 kPa
Patient 32	6/5/2017	Resolved HCV				
Fibroscan 1 (initial)			6/5/17	**13.9** kPa	F4	>12.5 kPa
Fibroscan 2			7/7/17	**9.2** kPa	F2	7.5-10.0 kPa
Fibroscan 3			8/3/17	**10.6** kPa	F3	10.0-14.0 kPa
Fibroscan 4			10/5/17	**8.4** kPa	F2	7.5-10.0 kPa

Fibroscan 5			12/6/17	**15.1** kPa	F4	>12.5 kPa
Fibroscan 6			2/19/18	**11.8** kPa	F3	10.0-14.0 kPa
Fibroscan 7			5/17/18	**10.9** kPa	F3	10.0-14.0 kPa
Fibroscan 8			10/31/18	**9.1** kPa	F2	7.5-10.0 kPa

Table 6

Table 6 consists of 2 study patients with stage F4 un-resolved HCV who completed at least six months in the HepAssure medical food study.

	Enrollment Date	**Type of CLD**	**Date**	**Fibroscan Result**	**Fibrosis Stage**	**Reference Range**
Patient 6	3/20/2015	Un-resolved HCV				
Fibroscan 1 (initial)			3/20/15	**15.3** kPa	F4	>12.0 kPa
Fibroscan 2			11/23/16	**9.2** kPa	F2	7.0-9.5 kPa

Fibroscan 3			4/27/16	**6.1** kPa	F0-F1	<7.0 kPa

Patient 26	9/22/2016	Un-resolved HCV				
Fibroscan 1 (initial)			9/22/16	**37.5** kPa	F4	>12.5 kPa
Fibroscan 2			11/7/16	**26.4** kPa	F4	>12.5 kPa
Fibroscan 3			12/14/16	**28.0** kPa	F4	>12.5 kPa
Fibroscan 4			1/31/17	**24.7** kPa	F4	>12.5 kPa
Fibroscan 5			4/5/17	**21.6** kPa	F4	>12.5 kPa
Fibroscan 6			5/15/17	**27.1** kPa	F4	>12.5 kPa
Fibroscan 7			7/24/17	**25.4** kPa	F4	>12.5 kPa
Fibroscan 8			9/29/17	**20.1** kPa	F4	>12.5 kPa
Fibroscan 9			11/17/17	**27.8** kPa	F4	>12.5 kPa
Fibroscan 10			1/19/18	**25.4** kPa	F4	>12.5 kPa
Fibroscan 11			3/16/18	**34.8** kPa	F4	>12.5 kPa
Fibroscan 12			5/14/18	**26.3** kPa	F4	>12.5 kPa

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