Greetings and welcome to the Genfit GFT505 Phase 2b results conference call. At this time, all participants are in a listen-only mode. A brief question-and-answer session will follow the formal presentation. As a reminder, this conference is being recorded. I would now like to turn the conference over to Brian Korb. Thank you Mr. Korb. You may begin.

Thank you operator. Welcome and thank you for joining us on the call today. As a reminder, this call will include certain statements of the company with respect to the company’s historical and anticipated future performance. Such statements reflect various assumptions of management that may or may not prove to be correct and involve various risks and uncertainties. No representations or warranties are made as to the accuracy of such statements of anticipated performance. Finally, an archive of today’s call will be posted on the Genfit website. I will now turn the call over to Jean-François Mouney, Genfit’s Chief Executive Officer.

Good afternoon, I would like to welcome everybody to our webcast. I also have with me Sophie Mégnien, our Chief Medical Officer and Dean Hum, our CSO to discuss the trial design and top line results. This is a very exciting day for Genfit as we are announcing the top line results of the GOLDEN-505 trial, and in this webcast we would like to walk through what was presented in the press release and provide a bit more detail on why we and the KOLs are excited. You will appreciate and understand that we will not be able to provide full detail on the results, since we are preparing the publication in a major scientific journal.

To start off, I would like to sincerely thank all the patients and the clinical investigators who participated in this trial. I think the robust screening of the patients and the rapid rate by which they were enrolled into the trial is an attestation of the motivation of both the investigators as well as the patients to help develop a therapy for the treatment of NASH, and to address this large unmet medical need. I also think that the high proportion of patients who underwent the 2 biopsies, as required in this study, is also an indication of the confidence that the physicians and their patients have in GFT505; and last but not least, I would like to express my gratitude to the experts in the NASH field with whom we work closely for the design and undertaking of the trial, and with whom we will continue to collaborate as we move into phase 3 which we expect to launch by year end. Now I will turn it over to Sophie.

The GOLDEN trial is a truly international phase 2 study which assessed the efficacy and safety of GFT505 at 2 different doses, and the study was conducted in 56 centers, and 9 countries, within the United States and Europe. This was a double blind placebo controlled study which required one liver biopsy at the beginning of the trial to histologically diagnose the patient as having NASH, and then a second biopsy was required after one year of treatment to assess the effect of GFT505 on liver histology. Importantly there was a central reading for all of the biopsies. To be randomized into the trial the patients needed to have a score of at least 1 in all three histological components of NASH, which means a total NAS score of at least 3, and then “resolution of NASH” means a score of zero on at least one of the 3 components at the end of the treatment period. The primary endpoint is Reversal of NASH without worsening of fibrosis, as discussed with the FDA, and this is still aligned with the FDA/AASLD workshop recommendations which have been published last December.
Due to the unexpected 57% placebo effect in patients with early NASH (NAS score of 3), along with the large number of sites for a limited number of patients, the initial design didn’t enable to meet directly the primary endpoint.

To avoid these biases, we collaborated closely with one of the world leading healthcare statisticians (expert for WHO, FDA, EMA, pharma companies). We applied a statistical analysis based on mixed linear logistic regression model, well known by those familiar with biostatistics. Importantly, the use of this type of model had been discussed previously and approved in our recent meetings with the FDA.

Using this analysis to control for baseline severity of disease and the heterogeneity due to the large number of sites, this study demonstrates that GFT505 at the dose of 120mg attains the primary endpoint on the ITT population for the reversal of NASH without worsening of fibrosis, with a p value of 0.027. This pre-specified ITT population (237) was defined by the patients who were randomized, treated, and who had both biopsies.

As a robustness analysis, including patients without end of treatment biopsy and considered as non responders (274); even in this more stringent condition, we also saw a statistical significant benefit (p=0.016).

Importantly, we have also looked at the patients with more severe disease, with a baseline NAS of 4 or more (this represents 202 patients or 85% of our ITT population). Just to note, this is the same baseline population as some of the other recent NASH trial PIVENS & FLINT, which have excluded the early NASH patients with NAS=3.

In this population, the beneficial effect is even more pronounced where GFT505 120 mg doubles the response rate on the primary endpoint versus placebo. There are 22.4% of the treated patients who attained reversal of NASH without worsening of fibrosis vs 12.7% in the placebo group, with a p value < 0.046 so this provides further evidence that treatment with GFT505 leads to clinical benefit for the NASH patient.

As we had previously discussed, the GOLDEN-505 trial also has other secondary histological endpoints, including the NAFLD Activity Score. The study shows that GFT505 has a significant effect over placebo to decrease the NAS score by 2 points or more, with a p value of 0.04.

The 80mg dose which was the lower of the 2 doses tested provides evidence of efficacy on histological NASH but it did not reach statistical significance. However, the choice to test these 2 doses in this trial was also to address a dose response, and in fact, we found a linear dose response going from 80 to 120. So, the results of this trial show that 120mg is the minimal effective dose required to provide histological benefit in NASH patients.

When examining fibrosis there was no impact of the treatment on the histology; however, we should all keep in mind that this was a 1-year treatment trial which was not designed to address improvement on that; and to look appropriately at fibrosis, a trial should be at least 18 months in duration.

However, importantly, the study also looked at several scores that were developed to assess liver fibrosis, and here the results show that the 120 mg dose of GFT505 has a statistically significant beneficial impact on the “Fibrotest” and the “NAFLD Fibrosis score”. This is very important because these results indicate that GFT505 has a beneficial impact on liver fibrosis, and these scores may be considered as “early markers” of decreasing liver fibrosis.

Now I will turn over to Dean, to talk about the other non histological endpoints and biomarkers.
DEAN HUM

Thanks Sophie for handing it over to me, before I jump into the data, just to note, that all non histological data are analyzed through the initial statistical plan.

The following data I’ll talk about are relative to the 120mg dose, however 80mg also has significant activity on many of these important markers.

Beyond the assessment of histological endpoints, the study also looked at a comprehensive set of biological endpoints to determine and confirm the efficacy and safety of GFT505 as previously found in the other studies of the phase 2 program.

The assessment of liver markers such as GammaGT, ALT, and alkaline phosphatase show a statistically significant decrease which is consistent with GFT505 having a beneficial positive impact on the liver.

In addition, a comprehensive set of markers were also measured to inform on different composite scores, which have been developed by various groups to assess the status of NAFLD. Among these scores treatment with GFT505 shows a significant positive impact on the “Steatotest” and the “Fatty Liver Index”, which is not all that surprising considering the positive effect demonstrated on liver histology.

Another important aspect of GFT505 is that it has beneficial activities on cardiometabolic disease and metabolic parameters, as it had been demonstrated in several previous studies in the phase 2 program.

So in the present GOLDEN-505 trial, the drug demonstrates for the first time that in “NASH” patients it also has consistent positive impacts on cardiometabolic disease.

The results in NASH patients clearly show the beneficial effects on lipids, where the bad lipids such as triglycerides, total cholesterol, and importantly LDL-cholesterol are significantly decreased; whereas the good lipids such as HDL-cholesterol are increased.

This beneficial lipid profile or so-called cardioprotective lipid profile is very important for the NASH patient, considering that most NASH patients have metabolic disease, and there are some KOLs who even consider NASH to be the liver manifestation of metabolic disease, so it is a well known fact that many NASH patients have dyslipidemia; therefore, a NASH therapy such as GFT505 that can provide a beneficial or cardioprotective lipid profile in addition to benefit on liver histology represents a very important advantage.

As well, the drug decreases circulating free fatty acids; and in the patients of the trial who also had type 2 diabetes, the drug also demonstrated the ability to significantly decrease HbA1c, fasting plasma glucose, and fasting insulin. These findings are again important for the treatment of metabolic NASH patients, because many of them have type 2 diabetes.

To confirm the anti-inflammatory activity of GFT505 in NASH patients, it was shown that it decreases several markers of acute phase inflammation, such as hs CRP, Haptoglobin and Fibrinogen. It is also important to note that these markers are secreted from the liver so the ability of GFT505 to decrease their levels is consistent with the drug having an anti-inflammatory effect on the liver of NASH patients.

From the standpoint of clinical management or clinical benefit and treatment of NASH patients, the activity of GFT505 on these different biomarkers provide evidence that this drug has a global beneficial effect for the patient.

Remember that cardiovascular disease is the leading cause of death in NASH patients.
So if we consider the different activities of GFT505 on these different markers that were measured in this trial, these improvements actually translate into a significant reduction of the Procam predictive score for cardiovascular outcomes. Procam is a cardiovascular risk score like the Framingham score, that is widely used in Europe.

Moreover, it is very important to note that these beneficial metabolic effects are obtained “on top of the standard of care” that these patients are receiving for their metabolic disorders.

When considering the development of a pharmacological treatment of NASH we have to take into account that the drug will most likely be for long term use; therefore the safety and tolerability profile of the drug must be clean. At the end of the 1-year treatment period, the 80 and 120mg dose of GFT505 did not lead to any safety or tox issues, so this drug has a very clean and favorable safety profile.

At the dose of 120mg, there was a small increase in creatinine levels of under 5%, which is most probably related to the PPARalpha activity of GFT505, which is known to increase creatinine. This small increase is readily reversible upon discontinuation of treatment, and easy to monitor.

The most frequent adverse events encountered in the trial are mild and common, and were balanced across all groups. There were no cardiac events, signal on cancer, nor death in the GFT505 treatment groups.

The present trial confirms and is consistent with the previous trials where GFT505 does not induce any edema signal nor weight gain.

We frequently get the question of whether it increases puritus, so we would like to reiterate that based on the mechanism of action of GFT505 it should not induce nor increase puritus, and the present study is consistent with this and does not show any induction of puritus signal.

I will turn it back to Jean-François who will provide concluding remarks and next steps.

JEAN-FRANCOIS MOUNEY

Therefore, taking all the results of this trial together, GFT505 is a safe and efficacious drug, ready to go into phase 3. This is exactly what was confirmed by some top KOLs from the US and Europe we have met in the past week.

First, I would like to add that Genfit is working in parallel in the NASH biomarker field. The large amount of data collected in this phase 2 is an important asset to be successful in this discovery. This biomarker approach is necessary to finding non-invasive markers to replace the biopsy when our drug is on the market.

As you can imagine, there are still additional analysis to perform, such as sub-group and biomarker analysis, which will be completed shortly in preparation for the next phase of development and for upcoming meetings with the FDA and EMA.

As well, we would like to submit these exciting results for publication in a major journal so the next few weeks and months will be very busy for the team. We plan to host an analyst/investor event during the EASL congress in Vienna, and will also be present at some investor conferences and look forward to seeing some of you soon.