ARROWHEAD PHARMACEUTICALS

Fiscal 2022 Year End Conference Call – Prepared Remarks

November 22, 2021

1:30 PM Pacific time

Operator

Ladies and gentlemen welcome to the Arrowhead Pharmaceuticals conference call. Throughout today's recorded presentation all participants will be in a listen-only mode. After the presentation, there will be an opportunity to ask questions. I will now hand the conference call over to Vincent Anzalone, Vice President of Investor Relations for Arrowhead. Please go-ahead Vince.

Vince Anzalone

Good afternoon everyone. Thank you for joining us today to discuss Arrowhead's results for its 2022 fiscal year ended September 30, 2021.

With us today from management are president and CEO Dr. Christopher Anzalone, who will provide an overview of the quarter; Dr. Javier San Martin, our chief medical officer, who will provide an update on our pipeline; and Ken Myszkowski, our chief financial officer, who will give a review of the financials. In addition, Dr. James Hamilton, our senior vice president of Discovery & Translational Medicine, will be available during the Q&A session of today’s call.

Before we begin, I would like to remind you that comments made during today’s call contain certain forward-looking statements within the meaning of Section 27A

of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. All statements other than statements of historical fact are forward-looking statements are subject to numerous risks and uncertainties that could cause actual results to differ materially from those expressed in any forward-looking statements. For further details concerning these risks and uncertainties, please refer to our SEC filings, including our most recent annual report on Form 10-K.

With that said, I'd like to turn the call over to Christopher Anzalone, President and CEO of the Company. Chris?

Chris Anzalone

Thanks Vince. Good afternoon everyone and thank you for joining us today.

Before I review the last quarter, I want to discuss the announcement this afternoon regarding our license agreement with GlaxoSmithKline for ARO-HSD, our investigational therapeutic in a Phase 1/2 study that is currently being developed as a treatment for patients with NASH.

Upon closing, GSK will receive an exclusive license to develop and commercialize ARO-HSD in all territories except Greater China, which will be retained by Arrowhead. GSK will be wholly responsible for further clinical development and commercialization, outside of Greater China.

This partnership is a great fit for us because GSK has articulated a clear commitment to genetic medicine, it has substantial capabilities to clinically develop ARO-HSD for NASH, and has impressive commercial infrastructure and

expertise to bring this potentially important medicine to the tens of millions of patients who need it.

This is also an important transaction because it enables us to take a large spend over the next several years off our books and focus our development capital on programs we may commercialize ourselves. In addition, it brings in substantial non-dilutive capital while providing us with exposure to future success should this drug candidate offer patients the kinds of benefits we believe possible.

Arrowhead will receive an upfront payment of $120 million and is eligible for the following additional payments:

- $30 million at the start of Phase 2;
- $100 million at the start of Phase 3;
- Up to $190 million at launch in the US and major markets; and
- up to $590 million for key sales milestones.

Taken together, Arrowhead stands to receive up to $1.03bn. Arrowhead is further eligible to receive tiered royalties of mid double-digit to 20% on net product sales.

From a strategic standpoint, this deal is another demonstration of our ability to use partnering selectively in areas that are outside of our commercial focus. This also feels like the right time to partner because we believe our clinical data from the Phase 1/2 study demonstrate proof of concept for inhibiting the liver production of HSD17B13. We presented data at AASLD earlier this month showing deep dose-dependent reductions of intrahepatic mRNA and protein levels and a marked reduction in ALT. We believe these are compelling results.

The next steps for this program, if we had decided to retain global product rights, would have been to initiate a placebo-controlled Phase 2 study to evaluate whether HSD17B13 inhibition, over time, would lead to clinically significant improvements in NASH. The published genetic data suggest that people with loss-of-function mutations in HSD17B13 have some level of protection against fibrosis associated with NASH and other liver diseases. We think we have shown that ARO-HSD does what it is designed to do. However, there have not been any prior HSD17B13 inhibitors studied in clinical trials, so human proof of concept of a clinical benefit needs to be established in future clinical studies.

This is where GSK steps in. They have a global reach and extensive experience and resources in clinical, regulatory, medical affairs, and commercial. They are in a strong position to pick up the program and advance it efficiently. As I mentioned we think this deal is a net positive for the ARO-HSD program and the patients with NASH. We are confident that GSK is the right company to take the next steps in clinical development and to chaperone the program through the regulatory and commercial opportunities that lie ahead. We are thrilled to welcome GSK as our newest partner and look forward to working with them to advance this potentially important new medicine toward patients in need.

I will now move on to some of the recent highlights and accomplishments during the prior quarter and the period since our last call.

Our collaboration and license agreement with Janssen, which we signed in 2018, covered our hepatitis B program, previously called ARO-HBV and now called JNJ-3989, and three potential additional programs that we would develop preclinically for Janssen. JNJ-75220795 is the first program for which Janssen

exercised its option to take an exclusive license, which earned Arrowhead a $10 million milestone payment earlier in the year.

JNJ-75220795, currently in a Phase 1 clinical study, is an investigational RNAi therapeutic developed using Arrowhead's proprietary TRIM™ platform and is designed to reduce expression in the liver of PNPLA3 as a potential treatment for patients with NASH. PNPLA3 has strong genetic and preclinical validation as a driver of liver fat accumulation and damage. During the quarter Arrowhead earned an additional $10 million milestone payment after Janssen dosed the fifth patient in a Phase 1 clinical study.

Staying with the Janssen collaboration, data was presented at AASLD from REEF-1, a Phase 2b study to assess the efficacy and safety over 48 weeks of monthly subcutaneous injections of JNJ-3989 at a dose of 40, 100, or 200mg. This was used on top of daily NUC therapy with or without daily oral JNJ-6379, one of Janssen's capsid assembly modulators (or CAMs).

JNJ-3989 is an investigational RNAi therapeutic that targets all HBV RNAs, thereby intended to reduce levels of all viral HBV proteins. The primary endpoint of the study is the proportion of patients meeting NUC stopping criteria, which is ALT levels less than 3 times the upper limit of normal, HBV DNA less than the lower limit of quantitation, HBeAg negative, and HBsAg less than 10 IU/mL) at the end of treatment. Data from 24 weeks off treatment was also presented at AASLD.

The data were very encouraging to us. The greatest reduction in HBsAg was observed in the JNJ-3989 200 mg with NUC cohort. A dose dependent response was observed in other cohorts and it didn't appear that adding the CAM had any

beneficial effect. At week 48, the mean reduction in HBsAg from baseline was 2.6 log and 74.7% of patients achieved HBsAg less than 100 IU/mL. 19.1% of patients met NUC stopping criteria, the primary endpoint. Up to week 72, an additional 10.6% of patients met stopping criteria for a total of 29.7%. This is an important finding. Additional patients continued to meet stopping criteria six months after therapy was removed. We are eager to see data with longer follow up and individual patient profiles for this study.

If you recall the studies with our first-generation compound, ARC-520, in which some patients went on to achieve functional cures, the HBsAg clearance didn't happen within six months of therapy being removed. Some patients took 9, 12, 18 months or longer to clear HBsAg, so we are excited to see additional results from REEF-1.

We are also eager to see data from the various other studies that Janssen is conducting. These include:

- the ongoing REEF-2 study, where patients come off all therapy as they achieve NUC stopping criteria;
- an ongoing study in patients with HBV and the hepatitis delta virus, which is a patient population in desperate need of therapeutic options due to the rapid progression of the disease;
- and various studies with immunomodulatory agents added.
    - There are currently multiple studies ongoing with pegylated interferon, called PENGUIN, and a study called OSPREY with a DNA vaccine, JNJ-64300535.

We have been extremely impressed with how comprehensive the development program is for JNJ-3989, and we believe it has the potential to play a central role as a backbone therapy for chronic HBV.

Staying with AASLD, we also presented additional data on ARO-AAT, also called TAK-999, our investigational candidate designed to treat liver disease associated with alpha-1 antitrypsin deficiency, which received Breakthrough Therapy Designation from the FDA during the quarter and is being co-developed with Takeda.

We presented additional interim clinical data from the ongoing AROAAT2002 study, an open-label Phase 2 clinical study to assess the response to ARO-AAT in approximately 16 patients with AATD associated liver disease and baseline liver fibrosis. We think the data continue to reflect that ARO-AAT is highly active against its target. Specifically, the data suggest that ARO-AAT strongly inhibits production of the mutant Z-AAT protein, which we believe has been established as a clear cause of progressive liver disease. Further, the data suggest that livers in these patients are clearing the accumulated Z-AAT and may be showing signs of healing.

In this study, ARO-AAT treatment lead to a 72-100% reduction of liver Z-AAT protein. ARO-AAT treatment reduced histological globule burden in all patients, with 13 of 13 patients having a 1 point or greater reduction in PAS+D globule burden. ARO-AAT treatment also may have improved liver fibrosis. 6 patients had a 1 point or greater improvement in METAVIR fibrosis stage from baseline to week 24 or 48. Since the presentation, we analyzed an additional 12-month paired biopsy that showed improvement in fibrosis, giving us 7 of 15 with fibrosis

improvement now. When looking only at the 200mg cohorts, we saw 7 of 12 patients with improved fibrosis.

ARO-AAT treatment also improved multiple biomarkers of liver health. The mean reduction of ALT from baseline ranged from 42% to 56% and from 33% to 54% for GGT at week 28 and week 72. Importantly, all groups showed normalized ALT and GGT following treatment.

We believe these are all encouraging data. The program is on schedule and we continue to be confident about its potential. We have begun a productive dialogue with FDA about approvable endpoints and the potential for an accelerated approval pathway. We look forward to continuing this dialogue as the SEQUOIA study continues. We expect to have data on the reduction of circulating levels of AAT from SEQUOIA over the next few months, which should allow us to select a dose to move forward with. We should also be collecting the last 12-month biopsy from the last patient enrolled sometime in the summer of 2022.

Let's move on to ARO-HSD, which is our investigational candidate designed to treat NASH that we announced today has been licensed to GSK. We presented data at AASLD on the pharmacodynamic effect of ARO-HSD and safety of various dose levels. In AROHSD1001, a Phase 1/2 clinical study, we observed a dose-dependent pharmacodynamic effect on hepatic HSD17B13 mRNA in all patients. At the 200 mg dose, all patients showed greater than 90% mRNA reductions. Hepatic HSD17B13 protein levels were reduced at all ARO-HSD dose levels, with multiple measurements below the assay's lower limit of quantitation. Decreases in ALT and AST were observed at doses of 100 mg ARO-HSD and greater. ARO-HSD was well-tolerated in patients, with no drug-related serious adverse events

reported, no adverse events leading to drug discontinuations, and no drug-related clinically significant adverse laboratory trends observed.

We believe these data suggest that ARO-HSD is highly active at silencing liver production of HSD17B13. There is clearly an enormous unmet need for patients with NASH, and we look forward to GSK designing future studies to evaluate the compound in a Phase 2 and beyond.

Moving to our wholly owned cardiometabolic pipeline. Javier will give an update in a moment on the studies that are ongoing and their status, but I wanted to highlight another recent data presentation. At AHA last week, we presented additional Phase 1/2 clinical data on ARO-APOC3, Arrowhead's investigational therapeutic targeting apolipoprotein C-III, or APOC3, being developed as a treatment for patients with hypertriglyceridemia, severe hypertriglyceridemia, and familial chylomicronemia syndrome, or FCS.

The presentation was assessing four genetically confirmed FCS patients and 26 patients with multifactorial chylomicronemia, which we refer to as MCM or non-FCS. The latter patients tend to have extraordinarily high triglycerides and exhibit the same or a very similar phenotype as genetically defined FCS. We wanted to evaluate whether there is a different response to ARO-APOC3 in these two groups. This is important because we are now initiating a Phase 3 study of ARO-APOC3, which Javier will describe.

In our study of patients with FCS compared with non-FCS, ARO-APOC3 achieved similar levels of reduction of APOC3, similar changes in key lipid parameters, and similar and comparable safety parameters. APOC3 was reduced by 98% in FCS patients and 96% in MCM patients. Both groups showed similar maximum median

reductions in triglycerides of 91% and 90%, respectively. Non-HDL-cholesterol was reduced by 58% and 49%, respectively, and HDL-cholesterol was increased by 152% and 111%, respectively.

Across our programs, preclinical data have been largely predictive of early clinical data, and early clinical data has been predictive of later stage clinical data. We see this with respect to pharmacodynamic response and safety and tolerability. I believe this is part of what makes RNAi and Arrowhead special, and one of the main reasons we can go into early clinical development with confidence that we have a good idea about what to expect. In our view, this serves to increase the probability of success and potentially reduce risk.

That bring me to our newest clinical program. ARO-C3 is an investigational therapeutic designed to reduce production of complement component 3, or C3, as a potential therapy for various complement mediated diseases. During the quarter we announced the previously undisclosed candidate, we filed a CTA to begin clinical studies, and hosted a key opinion leader webinar to discuss the complement pathway and the diseases we will initially focus on. These include IgA nephropathy, Complement 3 Glomerulopathy, and Paroxysmal Nocturnal Hematuria. There are also other renal and hematologic diseases that we intend to evaluate in the future. The complement pathway is very complex, and we did our best on the webinar to explain why we think a C3 targeted drug has the potential to address multiple complement mediated or complement associated diseases. We and the KOLs also explained the theoretical advantages that an RNAi therapeutic, like ARO-C3, may have over other mechanisms and other complement targets. If you haven’t listened to the webcast, I recommend you view it on our website for more information about ARO-C3. This is an early clinical program, but as I

mentioned we have a good track record of preclinical data translating well to clinical studies for investigational medicines developed with the TRiM™ platform.

I want to provide a quick update on our pulmonary programs, including ARO-ENaC, which is currently voluntarily paused to new enrollment as we assess some potential preclinical toxicology findings. We are still conducting studies internally to understand the toxicology findings, and we don't have clarity yet on the path forward. While we conduct those studies, we continue to make progress on our two new pulmonary programs, which are on track for CTA filings in the first half of 2022. In addition, we are working on a next-generation ENaC candidate in parallel, should that prove to be helpful or necessary. We believe in ENaC as a target for cystic fibrosis and are confident that our pulmonary targeted TRiM™ platform has the potential to address multiple diseases in the lung without adequate treatment options. We have less clinical experience applying the TRiM platform to pulmonary tissue, so we don't yet have the predictability that we see when we apply the TRiM™ platform in the liver, but we are committed to getting there and we are convinced that we can.

Before turning it over to Javier to discuss the status of our mid and later stage cardiometabolic programs, I want to discuss our growth plans. We now have 10 clinical stage programs and intend to expand our pipeline by 2-3 new programs per year. To support this growing pipeline, we are in the planning stages of expanding our R&D footprint in both San Diego and Madison. We will be leasing a new space in San Diego that is scheduled to be built over the coming year. In Wisconsin, we are in discussions with local and state government authorities and economic development agencies to explore potential tax and other incentives to build a new facility. Should those discussions be successful, we intend to build an

Arrowhead campus in Wisconsin with two new facilities. These facilities will house expanded R&D and a GMP drug manufacturing plant.

Our pipeline is advancing both in size and proximity to commercialization to the point where our buy versus build analysis indicates that internal control of manufacturing now makes a lot of sense. This is true financially and, importantly, strategically. We value speed at every stage of development and in every function. Building out drug manufacturing capabilities for preclinical, clinical, and commercial drug product will give us more control over timing, process, and cost.

We have not yet closed on the purchase of the land so we have not yet started to incur significant costs, but Ken will talk later about our estimates for capex should we move forward with this planned expansion.

With that overview, I'd now like to turn the call over to Dr. Javier San Martin. Javier?

Javier San Martin

Thank you, Chris, and good afternoon everyone.

I want to focus on the status of our most advanced, wholly owned cardiometabolic programs, ARO-APOC3 and ARO-ANG3. Between these two programs there several clinical studies that are either active now or will be active soon.

I will start with ARO-APOC3. This is our investigational medicine targeting apolipoprotein C-III being studied in patients with various lipid disorders including hypertriglyceridemia, severe hypertriglyceridemia, mixed dyslipidemia,

multifactorial chylomicronemia, and familial chylomicronemia syndrome. The set of mid and late-stage studies for ARO-APOC3 is called the SUMMIT program, with each study named for a mountain peak. We currently have three open studies:

- 2001 is Phase 2 study in patients with severe hypertriglyceridemia, which we are calling SHASTA-2
- 2002 is a Phase 2 study in patients with mixed dyslipidemia, which we are calling MUIR
- 3001 is a Phase 3 study in patients with FCS, which we are calling PALIDASE

I will describe each of these studies briefly and give a current status for each.

SHASTA-2, is a double-blind, placebo-controlled Phase 2b study to evaluate the efficacy and safety of ARO-APOC3 in adults with severe hypertriglyceridemia, or sHTG. Three dose levels of ARO-APOC3 (10 mg, 25 mg, and 50 mg) will be evaluated against placebo in participants who have mean fasting triglycerides of greater than or equal to 500 mg/dL at screening. A total of approximately 216 participants will be enrolled in the study. All dose cohorts will enroll in parallel with 72 participants per dose cohort randomly assigned in a 3:1 ratio to receive ARO-APOC3 or placebo. Each participant will receive subcutaneous injections on day 1 and week 12. The duration of the study is approximately 54 weeks from screening to the week 48 end-of-study examination. The primary objective of the SHASTA-2 study is to evaluate the safety and efficacy of ARO-APOC3 in adults with sHTG and to select a dosing regimen for later stage clinical studies in this patient population.

SHASTA-2 has enrolled 40 of the planned 216 patients, with an additional 56 patients currently in screening to potentially be enrolled. We have activated 60 of the planned 80 sites. Our goal is to have the study fully enrolled around Q3 of 2022.

Moving on to the MUIR study. It is a double-blind, placebo-controlled Phase 2b study to evaluate the efficacy and safety of ARO-APOC3 in adults with mixed dyslipidemia. Four dose cohorts of ARO-APOC3 will be evaluated against placebo in participants who had the following at screening:

- Elevated triglycerides greater than or equal to 150 mg/dL but less than 500 mg/dL
- And, non-HDL-cholesterol greater than or equal to 100 mg/dL or LDL-cholesterol greater than or equal to 70 mg/dL

A total of approximately 320 participants will be enrolled in the study. All dose cohorts will enroll in parallel with approximately 80 participants per dose cohort, randomly assigned in a 3:1 ratio to receive ARO-APOC3 or placebo. In three cohorts (10 mg, 25 mg, and 50 mg), each participant will receive a subcutaneous injection on day 1 and week 12 for a total of 2 injections. In one additional 50 mg cohort, each participant will receive a subcutaneous injection on day 1 and week 24 for a total of 2 injections. The duration of the study is approximately 54 weeks from screening to the week 48 end-of-study examination. The primary objective of the MUIR study is to evaluate the safety and efficacy of ARO-APOC3 in adults with mixed dyslipidemia and to select a dose and dosing regimen for later stage clinical studies in this patient population.

The MUIR study has enrolled 22 of the planned 320 patients, with an additional 38 patients currently in screening to potentially be enrolled. We have activated 15 of the planned 32 sites. Our goal is to have the study fully enrolled in Q4 of 2022

The last active study for ARO-APOC3 is PALISADE. PALISADE is a Phase 3 study to evaluate the efficacy and safety of ARO-APOC3 in adults with familial chylomicronemia syndrome. Two dose levels of ARO-APOC3 (25 mg and 50 mg) will be evaluated against placebo in participants with fasting triglycerides greater than 880 mg/dL that are refractory to standard lipid-lowering therapy and a diagnosis of FCS. Approximately 60 participants will be randomized in 2:1 ratio to receive 4 total doses of ARO-APOC3 or placebo administered subcutaneously once every 3 months. The duration of the study is approximately 56 weeks from screening to the month 12 end-of-study examination. After month 12, participants will be eligible and invited to consent and continue in an open-label extension study. All participants in the placebo group who opt to continue will switch to active drug during the extension study. The primary objective of the PALISADE study is to evaluate the efficacy and safety of ARO-APOC3 in adults with FCS. The primary endpoint is percent change from baseline at Month 10 in fasting triglycerides. Additional secondary and exploratory endpoints include the change in other lipid parameters, incidence of acute pancreatitis, and other measures.

We have activated 3 of the planned 55 sites globally. We are working hard to activate additional sites. There are currently patients in active screening, and we anticipate the first patient to be enrolled and dosed before the end of the year.

I will now move on to ARO-ANG3, our investigational medicine designed to reduce production of angiopoietin-like protein 3 ANGPTL3 as a potential treatment for patients with mixed dyslipidemia. The set of mid and late-stage

studies for ARO-ANG3 is called the VISTA program, with each study named for a national park. We currently have one open study in the VISTA program.

- AROANG3-2001 – a Phase 2 study in patients with mixed dyslipidemia, which we are calling ARCHES-2

ARCHES-2 is a double-blind, placebo-controlled Phase 2b study to evaluate the efficacy and safety of investigational ARO-ANG3 in adults with mixed dyslipidemia. Three dose levels of ARO-ANG3 (50 mg, 100 mg, and 200 mg) will be evaluated against placebo in participants who had the following at screening:

1. LDL-cholesterol greater than or equal to 70 mg/dL or non-HDL-cholesterol greater than or equal to 100 mg/dL; and,
2. Mean fasting triglycerides between 150 and 500 mg/dL

A total of approximately 180 participants will be enrolled in the study. All dose cohorts will enroll in parallel with 60 participants per cohort randomly assigned in a 3:1 ratio to receive a subcutaneous injection of ARO-ANG3 or placebo on day 1 and week 12. The duration of the study is approximately 42 weeks from screening to the week 36 end-of-study examination. After completing the week 36 visit, participants will be eligible to continue in an open-label extension study. The primary objective of the ARCHES-2 study is to evaluate the safety and efficacy of ARO-ANG3 in adults with mixed dyslipidemia and select a dosing regimen for later stage clinical studies in this patient population.

The ARCHES-2 study has reached 50% enrollment with 90 of the planned 180 patients enrolled and dosed, with an additional 68 patients currently in screening to

potentially be enrolled. We have activated all 25 of the initially planned 25 sites. Our goal is to have the study fully enrolled in Q2 of 2022.

I will now turn the call over to Ken Myszkowski, Arrowhead's Chief Financial Officer. Ken?

Ken Myszkowski

Thank you, Javier, and good afternoon everyone.

As we reported today, our net loss for fiscal 2021 was $140.8 million or $1.36 per share based on 103.7 million fully-diluted weighted average shares outstanding. This compares with net loss of $84.6 million, or $0.84 per share based on 100.7 million fully-diluted weighted average shares outstanding, for 2020.

Revenue for fiscal 2021 was $138.3 million, compared to $88.0 million for 2020. Revenue in the current period primarily relates to the recognition of a portion of the $300 million upfront payment received under our collaboration agreement with Takeda. Revenue for the Takeda agreement will be recognized as we continue to work toward completing our performance obligations of managing clinical trials in process and certain manufacturing related services. There remains $209 million of revenue to be recognized associated with the Takeda collaboration and it is anticipated to be recognized over approximately 2-3 years. Any additional milestones achieved with our collaboration partners would be additive to this projection. During fiscal 2021 we also entered into a new collaboration agreement with Horizon to develop a drug candidate to treat uncontrolled gout. We received a $40 million upfront payment for this agreement. A portion of this amount was recognized in fiscal 2021 and we expect the balance to be recognized by the end of

2022 as our performance requirements are completed. Revenue in the prior period primarily related to the recognition of a portion of the milestones received from our license and collaboration agreements with Janssen.

We also announced a new licensing agreement with GSK today for our ARO-HSD candidate. This agreement will result in an upfront payment of $120 million to Arrowhead. We anticipate the substantial majority of this to be recognized as revenue in fiscal 2022.

Total operating expenses for fiscal 2021 were $287.3 million, compared to $181.2 million for 2020. This increase is primarily due to increased candidate specific and discovery R&D costs as the Company's pipeline of clinical candidates has both increased and advanced, as well as additional non-cash stock-based compensation expense.

Net cash provided by operating activities during fiscal 2021 was $171.2 million, compared with net cash used by operating activities of $95.4 million during 2020. The key driver of this change was the $340 million in total upfront payments received from Takeda and Horizon in fiscal 2021. Excluding any potential milestone payments received from our collaboration partners, we estimate our operating cash burn to be $60 to $80 million per quarter in fiscal 2022. In addition, we are planning to expand our manufacturing capabilities, and expand our R&D facilities. These capital projects, along with routine capital expenditures, will add an incremental cash outlay of $80 to $90 million for full year fiscal 2022.

Turning to our balance sheet, our cash and investments totaled $613.4 million at September 30, 2021, compared to $453.0 million at September 30, 2020. The increase in our cash and investments was primarily due to the $340 million in total

upfront payments received from Takeda and Horizon, offset by cash used for operating activities.

Our common shares outstanding at September 30, 2021, were 104.3 million.

With that brief overview, I will now turn the call back to Chris.

Chris Anzalone

Thanks Ken. And thanks to all of you for joining us today.

We are executing on our strategy with respect to platform extension, pipeline expansion, and business development. Arrowhead's opportunities in the near-term and long-term are vast and continue to grow each day. The data from our clinical programs continue to be encouraging and strongly support further development of our investigational medicines.

Lastly, and importantly, we see the potential in the not too distant future where important medicines discovered and developed by Arrowhead start to get to the patients who need them. This is why we invest in R&D and why we are building manufacturing capabilities now to support clinical and commercial supply needs.

I would now like to open the call to your questions. Operator?

Operator