ARROWHEAD PHARMACEUTICALS

Fiscal 2023 Fourth Quarter Conference Call – Prepared Remarks

November 29, 2023

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 Vince Anzalone, Vice President of Investor Relations for Arrowhead. Please go-ahead Vince.

Vince Anzalone

Good afternoon and thank you for joining us today to discuss Arrowhead's results for its fiscal fourth quarter and year ended September 30, 2023.

With us today from management are president and CEO Dr. Chris Anzalone, who will provide an overview of the quarter; Dr. Javier San Martin, our chief medical officer, who will provide an update on our mid and later stage clinical pipeline; Dr. James Hamilton, our Chief of Discovery & Translational Medicine, who will provide an update on our earlier stage programs; and Ken Myszkowski, our chief financial officer, who will give a review of the financials. In addition, Tracie Oliver, our chief commercial officer, and Patrick O'Brien, our chief operating officer and general counsel, will be available during the Q&A portion of the 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 and 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 and our quarterly reports on Form 10-Q.

I'd now 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.

Arrowhead made significant progress toward reaching our "20 in '25" goal to grow our pipeline of RNAi therapeutics to a total of 20 clinical stage or marketed products by the year 2025. With yesterday's announcement of a CTA filing for ARO-DM1, our newest skeletal muscle targeted program being evaluated as a treatment for type 1 myotonic dystrophy, we now have 15 clinical stage programs: 10 are wholly-owned and 5 are being developed with partners. We expect these 15 clinical programs to grow to 16 over the next month, with the addition of 1 more CTA this year. This will complete an extraordinarily productive year on the development front. In 2023 we will have nominated 9 new potential clinical candidates using our TRiM platform across 4 different tissues: liver, pulmonary,

CNS, and skeletal muscle. In addition, we will have filed 4 CTAs for new clinical candidates during the calendar year. We believe this type of productivity is simply unmatched in our field, and it is particularly impressive given the size and market capitalization of our company. Even so, we expect more in 2024.

To understand these 15 clinical programs now, the 20 we will shortly have, the new targets we are planning to address, and the new cell types we will target over the years, is to understand the magnitude of patients we expect to treat and the value we can create over the long term. Of course there is too much there to discuss in this setting, so today we will focus on some of the accomplishments, events, and considerations that may drive and unlock value in the near term.

I see three primary areas:

- First, we are de-risking our pulmonary platform with knock-down and safety data in our clinical trials and toxicity data from our chronic tox studies. These enable us to move toward mid-stage studies addressing three main categories of chronic lung disease: inflammation, muco-obstruction, and interstitial lung disease; each of which have unmet treatment needs.
- Second, we are making good progress toward becoming a commercial company: We expect our initial commercial product to be plozasiran, formerly ARO-APOC3, in the treatment of familial chylomicronemia syndrome for which we will complete a Phase 3 study in Q2 2024, followed by our anticipated second indication for treating patients with severe hypertriglyceridemia (or sHTG), and later a potential indication for treating the broad population of patients with mixed dyslipidemia and atherosclerotic cardiovascular disease.

- And lastly, we have directional guidance toward strengthening our balance sheet in shareholder friendly ways.

Let's start with the pulmonary platform. We believe that Arrowhead is the first and only company to show clinically that RNAi can be harnessed to silence gene expression in the human lung. This is important and marks the accomplishment of a key long-term goal we set for ourselves several years ago. We have always thought that once we have human safety and activity proof of concept with one candidate, it will unlock value in the entire platform and provide confidence that other programs could work similarly, much like our current expectations for new liver programs. So, let's talk about important de-risking steps.

First, we think we have confirmation that we have adequately addressed the chronic GLP toxicology issues of our first-generation ARO-ENaC candidate. In that program, we saw findings of local lung inflammation in chronic rat and monkey toxicology studies. We determined that this result was consistent with macrophage overload syndrome, and thus we needed to make next generation candidates with improved potency and enhanced duration of effect so we could stretch out the dosing interval and reduce exposure.

I think we are now over that initial hurdle.

We've received chronic toxicology results in both rodent and primate species for ARO-RAGE and ARO-MMP7. James will talk about the specifics, but the takeaway is that the NOAELs (or No Observed Adverse Effect Levels) suggest sufficient safety margins to move confidently into Phase 2 studies. These were welcomed results and, I believe, represent substantial de-risking for the entire pulmonary platform. Once we select a dose and dose interval for each candidate,

we plan to interact with regulatory authorities in 2024 to discuss all results to date, including toxicology, and our plans for further clinical development.

Next, we want to ensure that clinical safety and tolerability are acceptable. We now have three pulmonary programs in first-in-human studies and safety results have been consistent with no concerning safety signals across all three programs in 145 patients or healthy volunteers on active drug.

Third, we need to ensure that our pulmonary drug candidates are doing what they are intended to do. We still need patient data in ARO-MMP7 and ARO-MUC5AC to understand this, but ARO-RAGE data have been very encouraging. Normal healthy volunteers showed 89% mean max knockdown and 95% max knockdown of circulating sRAGE after 2 doses of 184mg ARO-RAGE. At 92mg, healthy volunteers showed a mean max knockdown of 80% and max knockdown of 90% after 2 doses. We are still collecting data from asthma patients, but so far they are mapping on top of those from normal healthy volunteers, as we expected.

Together, I believe these data are important for the ARO-RAGE program and, more broadly, serve to de-risk the entire pulmonary franchise. These data give us confidence that:

1. We have chronic tox coverage to move confidently into Phase 2 studies for ARO-RAGE and ARO-MMP7;
2. The drug candidates have been generally well tolerated in humans; and
3. We are seeing deep and durable knockdown in the ARO-RAGE clinical program that tracks what we saw in animal studies.

The next step is to interrogate whether RAGE knockdown leads to a favorable clinical effect in patients. Upstream of hard clinical outcomes or FEV1, there are

biomarkers that can inform on whether ARO-RAGE is engaging inflammatory pathways. We are approaching a time during the coming months, where we may have data on ARO-RAGE in asthma patients to make that assessment. We are currently dosing mild-to-moderate asthma patients and enrolling patients with high baseline FeNO to potentially enrich for an anti-inflammatory signal. I believe that signal would represent a significant further de-risking event so we are working quickly to get high FeNO patients enrolled.

The next area where I think we are creating substantial value, is our progress toward becoming a commercial company. Our Phase 3 study of plozasiran in patients with familial chylomicronemia syndrome is approaching completion and we expect the last patient visit to be in the second quarter of 2024. That is a big step for a development stage biotech company. We are carefully considering launch strategies for plozasiran and look forward to speaking more about those soon.

So where do we go after FCS?

Data from Phase 2 studies of both plozasiran and zodasiran, formerly ARO-ANG3, have been very compelling, and our presentations and webcast around the American Heart Meeting earlier this month were well received by physicians, industry, and the investment community. For plozasiran, we see a clear opportunity to treat patients with severe hypertriglyceridemia, or sHTG. We believe there are 3-4m people in the US with triglycerides over 500 mg/dl, with approximately 1 million of them with TGs greater than 880. There are very limited treatment options for these patients. Further, we anticipate an sHTG approval based upon studies demonstrating a lowering of triglycerides during 1 year of treatment, with an adequate safety profile. In P2 studies, plozasiran reduced TGs to lower than

500 in virtually all patients, and many had TGs fall to normal levels. We are presented with a compelling set of facts:

- a large pool of patients without adequate treatment options
- a clear and relatively short regulatory pathway, and
- a drug candidate that has been consistent and very effective in P1 and P2 studies with good tolerability.

We have had productive interactions with FDA, including an end of P2 meeting, to discuss the design of a Phase 3 clinical program in sHTG patients. We are finalizing planning, and I expect we will launch the studies early in 2024.

Beyond FCS and sHTG, we continue to see attractive opportunities to help a broader population of patients with plozasiran or with zodasiran. Both candidates have shown to substantially reduce remnant cholesterol, an increasingly appreciated risk factor of cardiovascular disease. I expect that we will conduct a cardiovascular outcome trial, or CVOT, and that we will launch it by the middle of 2024. We have been planning to run a CVOT with plozasiran, but given the exciting data we presented at AHA in mixed dyslipidemia patients, we are now considering whether plozasiran or zodasiran would be the better candidate. We expect to decide which is better suited for this patient population over the coming months. This is a good problem to have, as both appear to be potentially powerful agents in this large market, and we simply want to try to ensure we are moving the best candidate forward in this space.

Also on the late-stage side of our business, Takeda is enrolling patients in the P3 study of fazirsiran. It is my understanding that they intend to open approximately

90 sites world-wide to help ensure the program moves quickly to an approvable endpoint that could be met after 2 years of treatment.

Our wholly-owned programs, discovery engine, and bourgeoning commercial presence are all exciting components of our business that, we believe, will create substantial value going forward. They will also require significant capital over the coming years, and we are focused on building out our balance sheet to ensure that we can make these important investments.

To that end, we are actively working on opportunities to bring in capital in shareholder friendly ways, and we believe there are several good options in front of us. For instance, we are exploring specific product financing for the plozasiran sHTG P3 study and separately a possible CVOT, whether done with plozasiran or zodasiran. We believe we could source sufficient capital for those studies in return for limited royalties on those products. In addition, we have discussed business development in the past. We now have 5 different platforms that incorporate the design of high-quality RNAi molecules that target 5 different cell types: hepatocytes, skeletal muscle, pulmonary, adipose, and CNS. We believe this broad ability to deliver highly potent RNAi molecules to a variety of tissues is both scarce and valuable, and could enable dozens of new drugs. We believe there is ample room to work with partners and also continue to build an extensive wholly-owned pipeline. This is intended to continue to let our discovery engine move quickly while ensuring that:

(1) we focus on a more limited set of wholly-owned assets that provide our commercial team with a level of synergy and efficiency;

(2) we continue to have access to necessary capital outside the capital markets;

(3) we can continue to build more passive value as our partners invest in development and commercialization; and
(4) we can continue to serve patients.

As we are able to provide better clarity relating to the sources and magnitude of new capital, I believe a clear overhang in our stock may be removed. There is a lot of high-quality work going on at Arrowhead, and substantial potential value to be unlocked as we solidify our balance sheet. Stay tuned for details when we're able to talk more about it.

I want to highlight one last event from the quarter that is important. We announced that GSK reached an agreement with Janssen to transfer exclusive worldwide rights to further develop and commercialize JNJ-3989 to GSK. If you recall, Janssen announced that they were discontinuing hepatitis B research and later announced that they were winding down most of their infectious disease and vaccine programs. JNJ-3989 was one of the discontinued programs of this strategic decision. That created uncertainty about its future. Janssen was a good partner and we are confident that GSK will continue the diligent work Janssen started.

This transaction also builds on Arrowhead's relationship with GSK, which includes the 2021 exclusive license of GSK4532990, formerly ARO-HSD, an investigational RNAi therapeutic currently in a Phase 2 study as a potential treatment for patients with alcohol-related and nonalcohol related liver diseases. We look forward to continuing our productive relationship with GSK.

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 significant progress we've made on plozasiran, formerly ARO-APOC3, and zodasiran, formerly ARO-ANG3. This includes presentations at the American Heart Association meeting with Phase 2 data on the MUIR and SHASTA-2 studies of plozasiran and the ARCHES-2 study of zodasiran, a KOL webinar on the significance of these data, and recent interactions we've had with the FDA on our plans for Phase 3 studies.

Let's start with a review of what plozasiran is and then discuss the data presented at AHA.

Plozasiran is designed to reduce production of Apolipoprotein C-III, or APOC3, a component of triglyceride rich lipoproteins, or TRLs, and a key regulator of triglyceride metabolism. APOC3 increases plasma TG levels by inhibiting breakdown of TRLs by lipoprotein lipase and uptake of TRL remnants by hepatic receptors in the liver. Plozasiran is being developed as a treatment for patients with familial chylomicronemia syndrome, severe hypertriglyceridemia, and mixed dyslipidemia. These are three distinct patient populations with very different phenotypes.

Familial chylomicronemia syndrome, or FCS, is a severe and ultrarare genetic disease characterized by extremely high TG levels, typically over 1000 mg/dL, leading to high risk of acute pancreatitis that usually requires hospitalization and can be fatal. We are currently conducting the PALISADE Phase 3 study in 75

patients with FCS. The primary endpoint of the study is percent change from baseline in fasting TG. PALISADE is on schedule to complete in Q2 of 2024.

Severe hypertriglyceridemia, or SHTG, is characterized by marked elevations in TG levels, typically over 500 mg/dL, which can also lead to increased risk of acute pancreatitis, as well as an increased risk of cardiovascular disease. We conducted the Phase 2 SHASTA-2 study and reported data at AHA. We are also working on initiating Phase 3 studies, SHASTA-3 and SHASTA-4 in early 2024. I will discuss the AHA data and Phase 3 study design in a moment.

Lastly, Mixed dyslipidemia is the presence of high TGs, and remnant cholesterol, often with low HDL. Remnant cholesterol is believed to be a major contributor to the residual risk of atherosclerotic cardiovascular disease after LDL is well controlled. We conducted the Phase 2 MUIR study in patients with mixed dyslipidemia and reported those data at AHA. We are currently working on key features of the study design including patient population selection for a potential Phase 3 study in patients with ASCVD and mixed dyslipidemia.

We presented data at AHA for these last two patient populations: SHTG and mixed dyslipidemia.

In the Phase 2 SHASTA-2 study of plozasiran in 226 subjects with SHTG who had baseline TGs greater than 500 mg/dL, two doses of 10, 25, or 50 mg of plozasiran once every 12 weeks reduced TGs to near normal levels and more than 90% of patients achieved TG levels below 500 mg/dL, which is the risk threshold for acute pancreatitis. Plozasiran achieved mean maximum reductions of up to 90% in APOC3 and 87% in TGs. At 24 weeks, 12 weeks after the second dose, serum APOC3 remained 79% below baseline, and TGs were 74% below baseline, with

reduction in remnant cholesterol of 63%, while HDL-cholesterol increased 68% above baseline.

In the Phase 2 MUIR study of plozasiran in 353 subjects with mixed dyslipidemia who had fasting TGs between 150-499 mg/dL and either LDL-cholesterol greater than 70 mg/dL or non-HDL-cholesterol greater 100 mg/dL, subjects in the study received two doses of 10, 25, or 50 mg of plozasiran at baseline and at week 12 or two doses of 50mg at baseline and week 24. Plozasiran-treated subjects demonstrated a mean maximum reduction in APOC3 of up to 89% and robust reductions in atherogenic lipoproteins. At 24 weeks, plozasiran reduced TGs by 64%, remnant cholesterol by 54%, ApoB by 19%, and Non-HDL-cholesterol by 27%, while increasing HDL-cholesterol by 51%.

These were very encouraging results, and they received a lot of attention at AHA. After the presentations, we hosted a webcast featuring three experts in the treatment and management of lipid and lipoprotein disorders:

- Daniel Gaudet, Professor of Medicine at Université de Montréal, who discussed plozasiran in the context of the current treatment landscape for severe hypertriglyceridemia
- Børge Nordestgaard, Professor & Chief Physician, Copenhagen University Hospital, University of Copenhagen, Denmark, who discussed the emergent role of remnant cholesterol in cardiovascular disease
- And, Steven Nissen, Chief Academic Officer for the Heart and Vascular Institute at the Cleveland Clinic, who discussed why the decrease in atherogenic lipoproteins observed with Plozasiran has the potential to prevent CV outcomes.

A replay of the webcast is available on our website if you missed it. My takeaway was that all three experts agreed that plozasiran has a unique profile and great potential in FCS, SHTG, and in patients with ASCVD and mixed dyslipidemia. The support of these notable experts gives us additional confidence as we embark on Phase 3 studies to further evaluate plozasiran.

So, what will the SHTG Phase 3 studies look like? We had an end of Phase 2 meeting with the FDA, and our plan is to do two similar studies: SHASTA-3 and SHASTA-4 that, together will be composed of approximately 700 patients, all with TGs greater than 500 mg/dl. The primary endpoint is lowering TGs after 1 year. We will include a subset of patients at higher risk of acute pancreatitis events. We will provide more detail on that as we get the studies initiated in early 2024. We will also have a third study that enrolls patients with moderately elevated TGs to add to our safety database. We expect these studies to all be completed around the same time.

All in all, our interactions with FDA have been productive and helpful. We believe that we have incorporated their feedback and look forward to continued dialogue with the agency as we get closer to an NDA filing following completion of the PALISADE study in patients with FCS and as we move forward with additional Phase 3 studies in SHTG and mixed dyslipidemia.

We also presented data at AHA on zodasiran, which received a lot of attention. Zodasiran is designed to reduce production of angiopoietin-like protein 3, or ANGPTL3. which is a hepatocyte expressed regulator of lipid and lipoprotein metabolism with multiple potential modes of action, including inhibition of lipoprotein lipase and endothelial lipase.

In the Phase 2 ARCHES-2 study of zodasiran in 204 subjects with mixed dyslipidemia who had baseline TGs between 150-499 mg/dL and either LDL-cholesterol greater than 70 mg/dL or non-HDL-cholesterol greater 100 mg/dL, two doses of 50 mg, 100 mg, or 200 mg of zodasiran once every 12 weeks reduced the expression of ANGPTL3 and decreased atherogenic lipoproteins. Treatment with zodasiran resulted in substantial reductions of ANGPTL3 up to 74%, TGs up to 63%, LDL-C up to 20%, remnant cholesterol up to 82%, and ApoB up to 22% all at week 24. Zodasiran was also associated with a relative reduction in liver fat fraction at week 24, with no adverse events related to liver function test changes reported to date.

Plozasiran and zodasiran continued to show favorable safety profiles. Treatment emergent adverse events reported to date reflect the comorbidities and underlying conditions of the study populations.

As I mentioned before, we are currently considering multiple Phase 3 study designs and making decisions on patient population selection for mixed dyslipidemia in patients with atherosclerotic cardiovascular disease. We will talk more about that in 2024 after we have further interactions with FDA about our proposed plan.

I will now turn the call over to Dr. James Hamilton. James?

James Hamilton

Thank you, Javier.

I believe the productivity of our discovery organization is unrivalled. This is partly due to the efficiency and scalability of siRNA therapeutics and specifically of our proprietary TRiM™ platform, but more importantly a product of the culture of speed and innovation at Arrowhead. We continue to find ways to outperform others in the RNA therapeutics space with a highly productive and lean organization.

In 2023 alone, we completed discovery and optimization work across 5 different delivery platforms and nominated 9 clinical candidates. Each then may go on to the IND-enabling phase, including GLP toxicology studies, clinical supply manufacturing, as well as preparation and submission of regulatory filings. We are also working on a discovery pipeline of similar size for 2024. This high level of productivity is how we intend to reach our 20 in 25 development goal.

Our discovery stage pipeline is, for the most part, kept confidential until we are approaching a CTA, or at times until we file a CTA. So, you will likely start hearing more about the newly nominated clinical candidates over the coming quarters.

For example, yesterday we announced that we filed a CTA for ARO-DM1, our clinical candidate for the treatment of patients with type 1 myotonic dystrophy, or DM1, and our second clinical program using the TRiM™ platform for delivery to skeletal muscle. The Phase 1/2a dose-escalating study will evaluate the safety, tolerability, and PK/PD profile of single and multiple ascending doses of ARO-DM1 compared to placebo in up to 48 patients with DM1.

ARO-DM1 is designed to reduce expression of the dystrophia myotonica protein kinase (DMPK) gene. DM1 is the most common adult-onset muscular dystrophy

and there is currently no approved disease-modifying therapy. Treatments have focused on symptomatic management, including physical therapy, exercise, ankle-foot orthoses, wheelchairs, and other assistive devices. ARO-DM1 represents a novel approach to treat DM1 by silencing aberrantly transcribed DMPK mRNA, which could lead to improvements in multiple symptoms, including muscle strength and function.

We have several exciting early-stage clinical programs that target genes expressed in the liver, lung, muscle, and CNS, each of which is moving toward proof-of-concept data. However, I will focus on our three pulmonary programs. Specifically, I'd like to review safety and tolerability data to date, recent chronic toxicology results that I think help to de-risk the pulmonary platform broadly, as well as some new PD data that further support our plans to rapidly move all programs forward.

To review, our three clinical stage pulmonary programs are the following:

- ARO-RAGE is designed to reduce expression of the receptor for advanced glycation end products, or RAGE, as a potential treatment for inflammatory pulmonary diseases
- ARO-MUC5AC is designed to reduce production of mucin 5AC, or MUC5AC, as a potential treatment for muco-obstructive pulmonary diseases
- And, ARO-MMP7 is designed to the reduce expression of matrix metalloproteinase 7, or MMP7, as a potential treatment for idiopathic pulmonary fibrosis, or IPF

All three of these programs are in Phase 1/2a clinical trials evaluating single and multiple doses in normal healthy volunteers and in patients.

Across the three programs, 145 total subjects, both normal healthy volunteers and patients, have received active drug via inhalation, with another 31 receiving ARO-RAGE via the subcutaneous route. There have been no serious adverse events deemed to be related to drug. There have been no patterns of drug related adverse events, pulmonary adverse events such as cough or shortness of breath, or adverse changes in lab or spirometry values. There has also been no evidence of local lung inflammation based on BALF cell count evaluation and all chest X-rays have been read as normal.

These safety and tolerability results have largely been consistent across the three programs and are highly encouraging for the pulmonary platform.

Next, I'd like to cover the chronic toxicology results for ARO-RAGE and ARO-MMP7, which we recently received. These results are also highly encouraging and suggest that we have sufficient safety margins to proceed confidently to phase 2.

Specifically, for ARO-RAGE the no observed adverse effect level, or NOAEL, in the six-month rat study was the mid dose and in the nine-month monkey study it was the highest dose studied. For ARO-MMP7 the rat NOAEL was the highest dose, and the monkey NOAEL was the mid dose.

Keep in mind that dose levels selected for GLP toxicology studies are high multiples of the desired clinical dose, so some findings in a toxicology study are expected. The results for both ARO-RAGE and ARO-MMP7 suggest that the learnings and improvements we have made since our first-generation pulmonary candidate, ARO-ENaC, have improved the therapeutic index for our inhaled

siRNA programs. Pending feedback from regulatory authorities, we are confident that we will have the required safety margins to begin phase 2 studies.

This is an important step for the pulmonary platform at an important time as we look to design and initiate Phase 2 studies in 2024.

Now, moving on to new pharmacodynamic data, ARO-RAGE continues to yield promising dose-dependent target engagement results. We previously reported impressive reductions of soluble RAGE protein, or sRAGE, in serum and in bronchoalveolar lavage fluid, or BALF, in healthy volunteers.

Previously reported at our June Analyst R&D Day, after two doses of 92 mg given on Days 1 and 29, serum sRAGE mean maximum reduction was 80% with a maximal knockdown of 90%. After a single dose of 184 mg, we observed a mean reduction of 90% and maximal reduction of 95% in BALF sRAGE, with mean maximum serum sRAGE reduction of 76% and maximal reduction of 91%.

We have since received additional sRAGE data after two doses of 184 mg in healthy volunteers. After a second dose of 184 mg, serum sRAGE mean maximum reduction was 89% with a maximal knockdown of 96%.

Additionally reduction of serum sRAGE was similar in healthy volunteers and in patients with asthma at the 44 mg dose level.

So, what are the next data points that we are watching? We are currently enrolling the top dose cohort in mild-to-moderate asthma patients and have initiated a cohort of asthma patients with high baseline levels of fractional exhaled nitric oxide, or FeNO, which is a biomarker for the degree of IL-13 driven type 2 inflammation in

the lung. These are both important to watch. If we continue to see consistency of PD effect in asthma patients, that would be encouraging. Also, it would be highly encouraging if RAGE lung knockdown leads to an anti-inflammatory effect, via the FeNO biomarker, in either the mild-to-moderate asthma patient cohorts or, more likely, in the high FeNO cohort. The former should have data available during the coming months and the latter will have data around Q3 of 2024.

I will now turn the call over to Ken Myszkowski. Ken?

Ken Myszkowski

Thank you, James, and good afternoon everyone.

As we reported today, our net loss for fiscal 2023 was $205.3 million or $1.92 per share based on 106.8 million fully-diluted weighted average shares outstanding. This compares with net loss of 176.1 million or $1.67 per share based on 105.4 million fully-diluted weighted average shares outstanding, for 2022.

Revenue for fiscal 2023 was $240.7 million, compared to $243.2 million for 2022. Revenue in the current period primarily relates to our collaboration agreements with Takeda, GSK & Amgen. Revenue is recognized as we complete our performance obligations or key developmental milestones are reached. For Takeda, Revenue is recognized commensurate to our performance obligation, which includes managing the ongoing AAT phase 2 clinical trials. There remains $866.2 thousand of revenue to be recognized associated with the Takeda collaboration which will be recognized in the next fiscal quarter. Revenue in the prior period primarily related to the recognition of payments received from our

license and collaboration agreements with GSK and a portion of payments received from our license and collaboration agreements with Takeda & Horizon.

Total operating expenses for fiscal 2023 were $445.7 million, compared to $421.7 million for 2022. This increase is driven primarily by increased candidate specific and discovery R&D costs as the Company's pipeline of clinical candidates has both increased & advanced into later stages of development.

Net cash used by operating activities during fiscal 2023 was $153.9 million, compared with net cash used by operating activities of $136.1 million during 2022. The increase in cash used by operating activities is driven primarily by higher research and development expenses. We expect our operating cash burn to be $110 to $130 million per quarter in fiscal 2024 and we expect full year capital expenditures of approximately $150 million as we near completion of our GMP manufacturing facility.

Turning to our balance sheet, our cash and investments totaled $403.6 million at September 30, 2023, compared to $482.3 million at September 30, 2022. The decrease in our cash and investments was primarily due to cash used for operating activities and capital expenditures, partially offset by cash inflows from financing activities.

Our common shares outstanding at September 30, 2023, were 107.3 million.

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

Chris Anzalone

Thanks Ken.

Arrowhead had another productive quarter and we see wide open space to accelerate our growth over the coming year. We expect 2024 to be a data- and event-rich year with many expected opportunities to create value including:

- Readout of the plozasiran FCS P3
- Filing our first NDA
- Launching a P3 for sHTG with plozasiran
- Launching a P3 CVOT with either plozasiran or zodasiran
- Readout in various patient populations with ARO-C3
- Initial CNS data in patients with ARO-SOD1
- ARO-RAGE FeNO and knockdown data in asthma patients
- ARO-MMP7 knock down data in IPF patients
- ARO-MUC5AC knock down data in asthma and COPD patients
- Initiation of first-in-human studies with our first adipose-targeting platform

Thank you for joining us today and I would now like to open the call to your questions. Operator?

Operator