ARROWHEAD PHARMACEUTICALS

Fiscal 2022 Year End Conference Call – Prepared Remarks

November 28, 2022

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 and thank you for joining us today to discuss Arrowhead's results for its 2022 fiscal year ended September 30, 2022.

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 mid and later stage clinical pipeline; Dr. James Hamilton, our senior vice president 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 both 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.

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.

Our 4th fiscal quarter and period since our last call has been highly productive. We've seen clear progress across our large and balanced pipeline: large because it now includes 12 drug candidates in clinical trials, and balanced because it spans multiple therapeutic areas and includes 6 partnered programs and 6 that are wholly-owned. It is a good representation of that which makes us different: we are a company built on an increasingly validated technological platform, applied to a large number of varied diseases across multiple organ systems, where development is uncommonly rapid from idea to the patients in need, and we use targeted, disciplined partnering to help finance development of our wholly-owned drugs.

This is who we are, and these factors are not new. What is new is the growing sense of clarity we are achieving. I think that is the recurring theme of this update. We have increased clarity as to the make up of our multiple phase 3 programs; increased clarity as to how we intend to use our late-stage drug candidates in different patient populations; increased clarity as to when we expect proof of concept from our earlier-stage programs; increased clarity as to where we plan to go next with the expansion of our platforms into new cell types; increased clarity as to how large we think our pipeline of clinical candidates will be over the next few years, and increased clarity about how we intend to finance our growing pipeline.

Let's touch on some of these.

First, we expect to report on progress for fazirsiran, our AAT program partnered with Takeda, in the near term. We would like to report topline data from the Phase 2 SEQUOIA study at the same time we give guidance on the Phase 3 study design. Ideally, Takeda and Arrowhead would do these together. Takeda submitted a Phase 3 protocol to the US FDA at the end of last quarter and is waiting for feedback. We expect Takeda to receive that feedback shortly, if there are any comments at all. We believe that FDA's feedback from prior meetings has been appropriately incorporated into the study design, so we do not expect any major surprises. I believe we have clarity on the future development path and timelines as well as what the SEQUOIA data are telling us and we will share that as soon as we can.

Second, we are gaining a clearer understanding about how our cardiometabolic programs perform in different patient populations, and thus are better able to determine the positioning of each, and importantly the development paths and

studies needed to seek approval for various indications. Javier will talk about this more in a moment, but the interim analyses for the SHASTA-2 and MUIR studies of ARO-APOC3 and the ARCHES-2 study of ARO-ANG3, which we presented at AHA and at an analyst investor event shortly thereafter, gave us some critical insights that are helping to accelerate the path to Phase 3 studies. We are working on determining the optimal paths, and we expect to have further clarity, including from multiple anticipated regulatory interactions, in 2023.

At present, we plan to pursue studies to enable us to treat homozygous familial hypercholesterolemia (HoFH) and heterozygous familial hypercholesterolemia (HeFH) patients with ARO-ANG3. We hope this would enable us to pursue a staged commercial strategy whereby we could serve the small HoFH market first and grow into the HeFH market after those larger studies are complete and supplemental regulatory approval is obtained.

For ARO-APOC3, we are conducting studies now to enable us to treat FCS patients, followed by treating patients with severe hypertriglyceridemia, and eventually the broad population with mixed dyslipidemia. As with the HoFH to HeFH approach, we like the staged commercial strategy and hope we can serve the small FCS market rather quickly, then expand to the larger sHTG population, and eventually the even larger mixed dyslipidemia populations when those studies are complete and their respective supplemental regulatory approvals are obtained.

Third, we have line of sight on timelines for initial interim clinical results for two of our pulmonary programs. James will give details on the status, but ARO-RAGE and ARO-MUC5AC are progressing well and we anticipate being able to provide interim data publicly in the first half of 2023. Should we have data that provides clinical proof of concept, we think it would be a potentially big de-risking event for

the candidates, and for the pulmonary platform generally. We believe we've made a lot of progress with the platform since our generation 1 candidate ARO-ENaC, and gaining clarity on how the generation 2 candidates perform will be exciting. Importantly, we are performing various analyses to assess pharmacodynamics using different methods, so we are confident that we should be able to define knockdown and duration of effect at different dose levels and different timepoints. The ARO-MMP7 Phase 1 started later than ARO-RAGE and ARO-MUC5AC, but dosing in healthy volunteers should begin imminently.

Fourth, our ARO-C3 program continues to progress well and we expect to have interim knockdown and safety data in the first half of 2023. This is an important program for us because: (a) it is squarely in our wheel-house as an hepatocyte target; and (b) because of the variety of opportunities we can pursue in various complement-mediated and complement-associated diseases.

Fifth, we continue to expand our platform into new cell types and have made enough progress to give us line of sight as to when we can discuss one of them publicly. I expect to provide guidance about our next cell type and initial targets by the end of the first half of 2023. Our goal is to continually expand our platform to gain access to a new cell type every 18-24 months. So far, we are ahead of the goal and you should be hearing more about the work that has gone into the newest cell type and the encouraging preclinical results we are generating.

Sixth, we have a good idea about how large we think we can grow our pipeline in the near- to mid-term and are announcing our 20 in '25 program. We plan to have 20 individual drug candidates in clinical trials or in the market in 2025. Between our hepatocyte-directed programs, our pulmonary programs, potential skeletal-muscle targeted programs, and new cell-types, we believe we will hit 20 in '25

between wholly-owned drug candidates and partnered programs. This will be a remarkable achievement that has the potential to touch millions of lives and create substantial value.

Seventh, we have better clarity about our financial resources. We currently have partnerships with 5 different companies and we expect to receive milestone payments from each over the next 12 months. Further, our expanding platforms give us the ability to continue to do new business development deals that could continue to provide capital to fund our own programs. Notwithstanding access to capital via these means, we recently decided to sell the potential royalties we would receive from Amgen on future olpasiran sales to Royalty Pharma. We received $250 million in cash upfront, and up to $160 million in additional payments contingent on the achievement of certain clinical, regulatory, and sales milestones. In addition, we retained rights to $400 million in development, regulatory, and sales milestone payments potentially due from Amgen from the 2016 license agreement. We have been very impressed with the data from that program and we are confident that it has the potential to be an important medicine. However, the next step in development is a cardiovascular outcomes study that will not readout for multiple years, so it made sense now for us to monetize the potential stream of future royalties. This allows us to continue investing in our wholly-owned programs which we think are advancing rapidly toward potential commercialization and also continue to invest in our expanding pipeline and platform technology. Our overarching goal is to bring important medicines to patients as quickly as possible, and I believe there are 2 critical, interrelated pieces to that: (1) develop and commercialize some drugs ourselves; and (2) substantially increase our market capitalization so we can do more of #1. That is the prize we need to keep our eye on, so every decision we consider should be made by asking

ourselves if it gets us closer to or farther from that goal. In my mind, the decision to sell these future royalties clearly gets us closer to that goal.

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 give updates on the two main areas of our late-stage development efforts: First on our cardiometabolic pipeline; and second, on fazirsiran, formerly called ARO-AAT and TAK-999.

Earlier this month, data was presented on all three of our cardiometabolic programs, ARO-APOC3, ARO-ANG3, and olpasiran at the American Heart Association Scientific Sessions 2022 and at a virtual analyst and investor event that we hosted a couple days after AHA. This was a very comprehensive review of the data and our plans for the programs, so if you want to hear more from us and from external key opinion leaders in the cardiometabolic space you can listen to a replay of the webcast or view the presentation slides. Both are available on the Arrowhead website.

Today I want to give some context about why we performed an interim analysis, highlight some of the important results, and provide guidance on where we see the programs going in the future. Chris mentioned earlier that we are gaining clarity across multiple programs, and this is a key point, specifically for the cardiometabolic programs. We now have more clarity on how each of the

candidates perform in various patient populations and, importantly, where we should focus late-stage development.

So, let me start with context on the interim analysis that led to the AHA presentations. Our wholly-owned cardiometabolic candidates, ARO-APOC3 and ARO-ANG3, each target a different gene and based on human genetic studies and preclinical animal models each affect lipid and lipoprotein levels in different ways.

Remember that we have data from different patient populations in the completed Phase 1/2 studies and multiple additional clinical studies going on now for each program.

For ARO-APOC3 we have the following studies:

- The SHASTA-2 Phase 2 study in patients with severe hypertriglyceridemia
- The MUIR Phase 2 study in patients with mixed dyslipidemia
- And the PALISADE Phase 3 study in patients with familial chylomicronemia syndrome

For ARO-ANG3 we have the following studies:

- The ARCHES-2 Phase 2 study in patients with mixed dyslipidemia
- And the GATEWAY Phase 2 study in patients with homozygous familial hypercholesterolemia

When combined with the Phase 1 data, we think these studies give us a good picture of how the different candidates may affect lipid and lipoprotein levels and thus which patient populations we should focus on for each. Therefore, the interim

analysis enabled us to start the important work required to prepare for Phase 3 studies. This includes:

- Dose and interval
- Patient population selection
- Length of study
- Modeling to estimate event rates and effect size
- Registrational path and Phase 3 design
- Where and how to execute the studies

We essentially gave ourselves a 6-month head start on all of that work. This is critical since we plan on having multiple end-of-Phase 2 meetings and moving forward with multiple Phase 3 studies over the next 12 months.

Next, I want to highlight some of the key results from the Phase 2 studies that we presented at AHA and our webcast event. ARO-APOC3, ARO-ANG3, and olpasiran were all highly active at silencing their respective gene targets, which resulted in encouraging changes in multiple relevant lipid and lipoprotein levels.

In the SHASTA-2 study in subjects with severe hypertriglyceridemia who had baseline triglycerides, or TGs, greater than 500 mg/dL, treatment with ARO-APOC3 at doses of 10 mg, 25 mg, and 50 mg all durably decreased APOC3 up to 87%, TGs up to 86%, non-HDL-C up to 45%, and increased HDL-C up to 99% through the week 16 timepoint. ARO-APOC3 has been well tolerated with treatment emergent adverse events reported to date that reflect the underlying comorbidities and conditions of the population under study.

In the MUIR study in subjects with mixed dyslipidemia who had baseline average TGs of 220 mg/dL, Non-HDL-C of 150, LDL-C of 110, ApoB of 95, remnant cholesterol of 46, and HDL-C of 42mg/dL, treatment with ARO-APOC3 at doses of 10 mg, 25 mg, and 50 mg resulted in substantial reductions of APOC3 of 80%, TGs of 65%, Non-HDL-C of 28%, LDL-C of 20%, ApoB of 20%, remnant cholesterol of 60%, and an increase in HDL-C of 50%. We believe these changes all represent key reductions in residual CVD risk factors.

In the ARCHES-2 study in subjects with mixed dyslipidemia who had baseline median TGs of 226 mg/dL, treatment with ARO-ANG3 at doses of 50 mg, 100 mg, or 200 mg resulted in substantial reductions of ANGPTL3 up to 71% at week 8, TGs up to 59% at week 16, and LDL-C up to 32% at week 16. ARO-ANG3 was also associated with median relative reduction in liver fat fraction at week 24 of 28% for the 100 and 200 mg dose, with no adverse events related to liver function test changes reported to date. ARO-ANG3 has been well tolerated with treatment emergent adverse events reported to date consistent with those expected in this patient population and with associated underlying comorbidities.

Amgen also presented end-of-treatment data from its Phase 2 OCEAN(a)-DOSE study of olpasiran in adults with elevated Lp(a) levels of greater than 150 nmol/L and a history of atherosclerotic cardiovascular disease. These data were also published in the New England Journal of Medicine. Placebo-adjusted mean percent reductions of LP(a) were 70.5% for patients receiving 10 mg every 12 weeks, 97.4% for patients receiving 75 mg every 12 weeks, 101.1% for patients receiving 225 mg every 12 weeks and 100.5% for patients receiving 225 mg every 24 weeks.

The totality of these data demonstrates the significant progress achieved in RNAi drug development and specifically suggests a potential future treatment paradigm

where Arrowhead's proprietary TRiM technology may be prominently leveraged in preventive cardiology.

So, what do we do with ARO-APOC3 and ARO-ANG3?

For ARO-ANG3, we are focusing on patients with hypercholesterolemia. ANGPTL3 is a key regulator of lipid and lipoprotein metabolism that inhibits Lipoprotein Lipase and Endothelial Lipase. ARO-ANG3 has a unique mechanism of action to address hypercholesterolemia distinct from other LDL-C-lowering therapies. It may address unmet need in patients with specific genetic mutations, for example patients with dysfunctional LDL receptor. It may also be added to other LDL-C-lowering therapies in patients not reaching goal.

Patients with heterozygous familial hypercholesterolemia, or HeFH, typically have LDL-C greater than 190 mg/dL and have increased risk of ASCVD. There are estimated to be around 1.4 million patients in the US with HeFH. Patients with homozygous familial hypercholesterolemia, or HoFH, typically have LDL-C greater than 400 mg/dL. There are around 1200 patients with HoFH in the US. These are the two indications that we are focusing on initially for ARO-ANG3. Our plan is to have end of Phase 2 meetings in the first half of 2023 and then potentially begin Phase 3 studies in the second half of 2023.

We view ARO-APOC3 as having a potentially broader set of indications and patient populations where it may provide a benefit. It potentially addresses the risk of pancreatitis in severe hypertriglyceridemia syndromes. ARO-APOC3 also modulates multiple lipids and lipoproteins that contribute to the residual risk of ASCVD in patients with mixed dyslipidemia, which has the potential to translate into a decrease in atherosclerosis and coronary disease progression.

APOC3 is a key regulator of lipid and lipoprotein metabolism that inhibits lipoprotein lipase and mediates hepatic uptake of remnant particles in an LPL-independent pathway. ARO-APOC3 improves multiple lipid parameters and may provide clinical benefit in a broad population with dyslipidemias. In clinical studies it has reduced TGs in patients with SHTG, including FCS, which has the potential to decrease the risk of acute pancreatitis. It has also reduced multiple residual cardiovascular risk factors, such as APOC3, LDL-C, ApoB, remnant cholesterol, and others in patients at risk of ASCVD.

We are already conducting the PALISADE Phase 3 study of ARO-APOC3 in patients with FCS, which is approximately 50% enrolled at this time. Our plan for the additional indications is to have regulatory interactions in the second half of 2023 and begin Phase 3 studies in the first half of 2024. These additional indications are SHTG, with a prevalence of around 4 million in the US, and patients at risk of ASCVD despite maximally tolerated statins, with a prevalence of around 12 million in the US.

Now I want to move on to fazirsiran, our investigational RNAi therapeutic designed to reduce production of a mutant form of the alpha-1 antitrypsin protein, called Z-AAT, as a potential treatment for the rare genetic liver disease associated with alpha-1 antitrypsin deficiency. Z-AAT accumulation is believed to be the cause of progressive liver disease in patients with AAT deficiency. Reducing production of the pro-inflammatory Z-AAT protein has the potential to halt the progression of liver disease and potentially allow the liver to regenerate and repair.

Data from our open label Phase 2 study were published earlier this year in the New England Journal of Medicine. Those data suggested that fazirsiran was very

effective at reducing the production of the Z-AAT protein and that the livers of these patients were able to begin the process of healing. This is includes breaking down and clearing the accumulated Z-AAT in the liver, decreasing the histologic globule burden, demonstrating histologic improvements in inflammation, reducing biomarkers of liver injury, and ultimately decreasing fibrosis severity. These were very encouraging signs for the potential of fazirsiran to help patients with AATD liver disease.

We now look to the fazirsiran Phase 2 placebo-controlled SEQUOIA study and to regulatory interactions on the Phase 3 study. The SEQUOIA data are mostly in now and we are waiting to receive feedback, if any, from the FDA on the proposed design for the Phase 3 study. These are expected soon, so we and our partners at Takeda will together determine the best way to communicate these publicly. Takeda is still on schedule to begin the Phase 3 study in the first quarter of 2023, and we are confident that we can have an update publicly on SEQUIOA and guidance on the Phase 3 prior to that.

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

James Hamilton

Thank you, Javier.

I want to give updates on four of our earlier stage programs that include three pulmonary candidates targeting RAGE, MUC5AC, and MMP7, and on our candidate targeting complement C3. Let's start with C3.

ARO-C3 is an investigational RNAi therapeutic designed to reduce hepatocyte expression of complement component 3, or C3, as a potential therapy for various complement mediated hematologic and renal diseases. We are conducting a Phase 1/2 clinical study now that includes two parts.

Part 1 is placebo controlled in healthy volunteers and includes single ascending dose, or SAD, cohorts and multiple ascending dose, or MAD, cohorts. All of the SAD and MAD cohorts are fully enrolled and participants are being followed to assess safety and tolerability, dose response based on serum C3 levels, and duration of effect at various dose levels. We are confident that we will have sufficient data in the first half of 2023 to report interim results from Part 1 of this study.

Part 2 is open label in eligible subjects with paroxysmal nocturnal hemoglobinuria, or PNH, and complement-mediated renal diseases, including IgA nephropathy and C3 glomerulopathy. Data from Part 1 will inform Part 2 dose selection, which we expect to happen in the coming months and then the patient cohorts will be opened for enrollment in the first half of 2023.

We are very excited about this program and believe it has the potential to address multiple serious complement-mediated or complement-associated diseases with unmet need in the renal and hematologic spaces. We know that complement C5 inhibitors are disease modifying in conditions such as PNH and believe that proximal C3 inhibition may confer advantages over C5 blockade. For example, C5 monoclonal antibodies only block the terminal complement pathway, and many of the proximal complement actions remain intact. In addition, clinical validation exists for C3 inhibitors, and we believe RNAi-based C3 inhibition could have clear dosing advantages over other mechanisms. Furthermore, alternative pathway

inhibition is likely of key relevance for treatment of conditions such as IgA nephropathy, C3 glomerulopathy and potentially other glomerular diseases. ARO-C3 is a subcutaneously administered candidate with an expected long dosing interval of once every 3 months or less frequent. We think this would be much more patient friendly than current C3 inhibitors that require a high volume infusion multiple times per week.

I will now move on to our three pulmonary candidates, starting with ARO-MMP7.

ARO-MMP7 is designed to the reduce expression of matrix metalloproteinase 7, or MMP7, as a potential treatment for idiopathic pulmonary fibrosis, or IPF. MMP7 is thought to play multiple roles in IPF pathogenesis, including promoting inflammation and aberrant epithelial repair and fibrosis. Silencing MMP7 expression in a rat IPF model reduced inflammatory cell infiltration, limited lung fibrosis, and preserved pulmonary function. In August, we filed a CTA to begin a Phase 1/2 clinical study of ARO-MMP7. The Phase 1/2 study will be similar in design to our other first in human studies and includes a healthy volunteer portion followed by a patient portion.

Now, moving on to our two other pulmonary programs, ARO-MUC5AC and ARO-RAGE, our investigational RNAi therapeutics designed to reduce production of mucin 5AC, or MUC5AC, and the receptor for advanced glycation end products, or RAGE, respectively, as potential treatments for various muco-obstructive and inflammatory pulmonary diseases. These two programs are on largely parallel paths and at approximately the same stage.

They are both in Phase 1/2 studies designed to assess safety and tolerability, pharmacokinetics, and pharmacodynamics in healthy volunteers first and then in

patients with asthma. For both programs, we are approaching full enrollment of the healthy volunteer SAD cohorts and are well into enrollment of the healthy volunteer MAD cohorts. In both the SAD and MAD, we have various methods to assess target engagement, including in induced sputum and bronchoalveolar lavage fluid, and for RAGE we are also measuring serum sRAGE protein, a circulating biomarker for RAGE target engagement in the lung. We anticipate that we will be able to report interim results from Part 1 of these studies and begin Part 2 in patients with asthma in the first half of 2023.

These are potentially important new medicines that address targets that have been difficult to drug using other modalities and are designed to treat muco-obstructive and inflammatory lung diseases in fundamentally new ways. We are excited to see and share these results and we are confident in the progress we've made on our pulmonary TRiM™ platform and these generation 2 candidates.

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 2022 was $176.1 million or $1.67 per share based on 105.4 million fully-diluted weighted average shares outstanding. This compares with net loss of $140.9 million or $1.36 per share based on 103.7 million fully-diluted weighted average shares outstanding, for 2021.

Revenue for fiscal 2022 was $243.2 million, compared to $138.3 million for 2021. Revenue in the current period primarily relates to our collaboration agreements

with Takeda and Horizon. Revenue will be recognized as we complete our performance obligations, which include managing the ongoing AAT phase 2 clinical trials for Takeda, and delivering a phase 1 ready candidate to Horizon. There remains $128.4 million of revenue to be recognized associated with the Takeda collaboration which we anticipate will be recognized over the next two to three years, and there remains $6.7 million of revenue to be recognized for Horizon, which we anticipate will be recognized by the end of calendar 2022. Revenue in the prior period primarily related to the recognition of a portion of the payments received from our license and collaboration agreements with Janssen and Takeda.

Total operating expenses for fiscal 2022 were $421.7 million, compared to $287.3 million for 2021. 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, as well as higher employee compensation expense including stock compensation expense.

Net cash used by operating activities during fiscal 2022 was $136.1 million, compared with net cash provided by operating activities of $171.2 million during 2021. 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 $70 to $90 million per quarter in fiscal 2023 and capital expenditures up to $200 million as we near completion on our footprint expansion projects, including GMP manufacturing.

Turning to our balance sheet, our cash and investments totaled $482.3 million at September 30, 2022, compared to $613.4 million at September 30, 2021. The decrease in our cash and investments was primarily due to cash used for operating

activities. Proforma cash and investments at September 30, 2022 including the Royalty Pharma cash receipt would be $732.3 million.

Our common shares outstanding at September 30, 2022, were 106.0 million.

As Chris mentioned earlier, on November 9, 2022, the Company and Royalty Pharma entered into a Royalty Purchase Agreement, pursuant to which Royalty Pharma agreed to pay up to $410.0 million in cash to the Company in consideration for the Company's future royalty interest in Olpasiran, originally developed by the Company and out-licensed to Amgen in 2016.

Pursuant to the Royalty Pharma Agreement, Royalty Pharma paid $250.0 million upfront and agreed to pay up to an additional $160.0 million contingent upon the achievement of certain clinical, regulatory & sales milestones.

The Company retained rights to $400 million in development, regulatory, and sales milestone payments potentially due from Amgen from the same 2016 out-licensing agreement.

I will now turn the call back to Chris.

Chris Anzalone

Thanks Ken.

In our business, opportunity abounds. There is no shortage of need that the biopharmaceutical industry can endeavor to serve and no shortage of lives that can

be touched. There is also no shortage of risk, as unknowns abound. As such, clarity is at a premium and will often be a primary value driver. We feel good about the clarity we have recently achieved and expect to achieve in the short-term. These include the following:

- Planning for the fazirsiran P3 is complete and currently under review with the FDA, and SEQUOIA data are in. We expect to be able to give guidance on the P3 and present topline SEQUOIA data with Takeda shortly.
- Interim P2 data from ARO-ANG3 and ARO-APOC3 suggest that both drug candidates are doing what they are designed to do, and we have good plans as to how to apply these in various patient populations. We expect multiple end-of-Phase 2 meetings in 2023 and to initiate multiple Phase 3 studies shortly thereafter.
- Progress with ARO-MUC5AC and ARO-RAGE in Phase 1/2 studies has been good. We expect interim data that could provide clinical proof of concept in the first half of 2023.
- ARO-C3 is progressing well in a Phase 1/2 study and we expect interim data that could provide proof of concept in the first half of 2023.
- Our discovery engine continues to perform and we expect to announce the next cell type we will be targeting in the first half of 2023.
- We have provided better clarity with respect to our balance sheet with our sale of olpasiran royalty rights for $250m upfront plus $160m in potential additional payments. This is on top of the remaining $400m we could access in clinical, regulatory, and sales milestone payments from Amgen.
- And, finally, we have announced our 20 in ’25 campaign. Our plan of having 20 individual drugs in clinical trials or at market in 2025 will be a remarkable accomplishment that, we believe, would represent a large leap

forward for medicine and position Arrowhead as a truly unique and impactful biopharmaceutical company.

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

Operator