Internal Medicine
Our discussions during Pfizer’s Investor Day include forward-looking statements about our anticipated future operating and financial performance, business plans and prospects; expectations for our product pipeline, in-line products and product candidates, including anticipated regulatory submissions, data read-outs, study starts, approvals, revenue contribution, growth, performance, timing of exclusivity and potential benefits; manufacturing and product supply; our efforts to respond to COVID-19, including our investigational vaccine candidate against SARS-CoV-2 and our investigational protease inhibitor, and our expectations regarding the impact of COVID-19; our ability to successfully capitalize on growth opportunities and prospects; plans for and prospects of our acquisitions and other business development activities, including our proposed transaction with Mylan N.V. (Mylan) to combine Upjohn and Mylan to create a new global pharmaceutical company; plans relating to share repurchases and dividends; and other statements about our business, operations and financial results that are each subject to substantial risks and uncertainties that could cause actual results to differ materially from those expressed or implied by such statements. Among other things, statements regarding revenue and earnings per share growth; the development or commercial potential of our product pipeline, in-line products, product candidates and additional indications, including expected clinical trial protocols, the timing of the initiation and progress of clinical trials and data read-outs from trials; the timing for the submission of applications for and receipt of regulatory approvals; expected breakthrough, best or first-in-class status, blockbuster status of our medicines or vaccines; and the impact of anticipated improvements to our clinical operation performance are forward-looking and are estimates that are subject to change and clinical trial and regulatory success. These statements are subject to risks, uncertainties and other factors that may cause actual results to differ materially from past results, future plans and projected future results. Additional information regarding these and other factors can be found in Pfizer’s Annual Report on Form 10-K for the fiscal year ended December 31, 2019 and in our subsequent reports on Form 10-Q, including in the sections thereof captioned “Risk Factors” and “Forward-Looking Information and Factors That May Affect Future Results”, as well as in our subsequent reports on Form 8-K, all of which are filed with the U.S. Securities and Exchange Commission and available at www.sec.gov and www.pfizer.com. Potential risks and uncertainties also include the impact of COVID-19 on our sales and operations, including impacts on employees, manufacturing, supply chain, marketing, research and development and clinical trials. The forward-looking statements in these presentations speak only as of the original date of the presentation and we undertake no obligation to update or revise any of these statements. Today’s discussions and presentations are intended for the investor community only; they are not intended to promote the products referenced herein or otherwise influence healthcare prescribing decisions. All trademarks in today’s presentations are the property of their respective owners.
Leadership Team Brings 75+ Collective Years of Cardiometabolic and Internal Medicine Expertise

Mike Gladstone
Global President, Internal Medicine

Jim Rusnak
Chief Development Officer, Internal Medicine

Morris Birnbaum
Chief Scientific Officer, Internal Medicine
We Develop Breakthroughs that Impact Millions

We prevent and treat the most prevalent health challenges facing society

Heritage and expertise in cardiometabolic conditions

Industry-leading footprint with cardiology and primary care

Strong internal pipeline enhanced via strategic partnerships

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Strong internal pipeline enhanced via strategic partnerships
Cardiovascular (CV) disease is leading cause of death worldwide
Advancing vupanorsen, a genetically validated investigational therapy to treat residual CV risk factors

NASH increases risk of serious outcomes (liver failure, CV events) – no approved therapies
Six potential first-in-class metabolic MOAs in our discovery and development portfolio

Cardiovascular Disease
Type 2 Diabetes and Obesity

Type 2 Diabetes (T2D) and Obesity are worldwide epidemics
Potential first small molecule oral GLP-1RA for T2D and Obesity
### Robust Early and Mid-Stage Pipeline with Multiple MOAs
Targeting Cardiometabolic Conditions

<table>
<thead>
<tr>
<th>NASH</th>
<th>Cardiovascular</th>
<th>T2D/Obesity</th>
<th>Cachexia</th>
<th>Additional</th>
</tr>
</thead>
<tbody>
<tr>
<td>DGAT2 Inhibitor ervogastat¹</td>
<td>Vupanorsen</td>
<td>Oral GLP-1RA danuglipron³</td>
<td></td>
<td>Tanezumab</td>
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<tr>
<td>KHK Inhibitor PF-06835919</td>
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<tr>
<td>ACCi / DGAT2i Combo clesacostat² / ervogastat¹</td>
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<tr>
<td>Oral GLP-1RA danuglipron³</td>
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<tr>
<td>CDK 4/6 (PAH) PF-06842874</td>
<td>Heart Failure</td>
<td>Insulin Sensitization</td>
<td>Appetite Stimulation</td>
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<tr>
<td>PNPLA3 Inhibitor</td>
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<tr>
<td>HSD17b13i Inhibitor</td>
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</table>

1. DGAT2i: ervogastat (PF-06865571)
2. ACCi: clesacostat (PF-05221304)
3. Oral GLP-1RA: danuglipron (PF-06882961)

*Highlighted in today's presentation*
Reducing significant residual CV risk with a genetically-validated, potential first-in-class treatment
Cardiovascular Disease is Still the Leading Cause of Death, Driven by Residual Cardiovascular Risk – Despite LDL-C Lowering

- Cardiovascular disease (CVD) accounts for **1 of every 3 deaths** in the US\(^1\)

- The average annual cost of CVD in the US is **>$200 billion**\(^1\)

- >6 million US patients with CVD or diabetes are at high **risk** despite effective LDL lowering with statins\(^2\)

- Residual risk is **driven in part by non-HDL-C and triglyceride-rich lipoproteins**\(^4,5,6\)

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### Substantial Residual CV Risk Remains Despite LDL-C Lowering

**FOURIER\(^3\)**

Median LDL-C with evolocumab = 30 mg/dL

**A. Primary Efficacy End Point**

<table>
<thead>
<tr>
<th>Hazard ratio, 0.85 (95% CI, 0.79–0.92)</th>
<th>P&lt;0.001</th>
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</table>

Vupanorsen May Provide a Potential First-in-Class Treatment for CV Risk Reduction Through Reducing ANGPTL3, a Genetically Validated Risk Factor

ApoB-containing lipoprotein turnover in the circulation is governed by ANGPTL3

Liver

Hepatic secretion → LPL → EL → Lipolysis → VLDL → VLDL Remnant → Uptake by liver and/or extra-hepatic tissues → LDL

ANGPTL3 inhibition promotes lipolysis and VLDL remnant clearance

Liver

Vupanorsen (ASO) → ANGPTL3

Hepatic secretion → LPL → EL → Lipolysis → VLDL → VLDL Remnant → LDL

Figure adapted from Adam, R.C. et al. J Lipid Res. https://www.jlr.org/content/early/2020/07/09/jlr.RA120000888.full.pdf. Commons license: https://creativecommons.org/licenses/by/4.0/legalcode
In a Phase 1 Study, Vupanorsen Demonstrated Robust Reductions in Triglycerides and Non-HDL Cholesterol, Important CVD Risk Factors

Reduction in non-HDL-C is strongly associated with lower risk of major vascular events

- Phase 2a: Presented at ESC 2020; encouraging biomarker results in hypertriglyceridemia, T2D and NAFLD
- Phase 2b: Initiating in 3Q 2020; investigating optimal dose to maximize target engagement and lipid lowering

In Ph1 (data above) doses administered QW. In Ph2a and Ph2b doses administered QW, Q2W or Q4W.

Vupanorsen Development Program is Intended to Support Multiple Indications and Advance the Science on ANGPTL3

<table>
<thead>
<tr>
<th>Year</th>
<th>SHTG (vupanorsen)</th>
<th>CVRR (vupanorsen)</th>
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<tbody>
<tr>
<td>2020</td>
<td></td>
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<td>2021</td>
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<td>2022</td>
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<tr>
<td>2023</td>
<td>Phase 2b</td>
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<tr>
<td>2024</td>
<td>Phase 3 vs Placebo</td>
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<tr>
<td>2025</td>
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<td>2026</td>
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<td>2027</td>
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<tr>
<td>2028</td>
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</table>

Timings are approximate and subject to change.

Peak Year Assumptions (US)

<table>
<thead>
<tr>
<th>Severe Hypertriglyceridemia (SHTG): 2025 Launch</th>
<th>Cardiovascular Risk Reduction (CVRR): 2028 Launch</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;2M Diagnosed Prevalence¹</td>
<td>&gt;6M Diagnosed Prevalence¹,²</td>
</tr>
<tr>
<td>35-60% Treatment Rate²</td>
<td>50-70% Treatment Rate²</td>
</tr>
<tr>
<td>20-35% Market Share³</td>
<td>20-35% Market Share³</td>
</tr>
</tbody>
</table>

CVRR: Statin-treated CVD patients with significant residual risk. SHTG: TGs >500 mg/dL

2. Pfizer Data on File. RWD analysis using Optum and Truven
3. Order of Entry Model, 1st 3 competitors
Clesacostat / Ervogastat (ACC / DGAT2 Inhibitors)

Leveraging our deep metabolic knowledge to develop breakthrough medicines for NASH
Driven by Increases in Obesity, the Impact of Nonalcoholic Steatohepatitis (NASH) is Growing and There are Currently No FDA- or EMA-Approved Therapies

Challenging to Diagnose and Treat

- Non-specific symptoms\(^1\)
- No currently approved therapies\(^2\)

Prevalence Expected to Grow

- **18 million** today in the US\(^3\)
- **24 million** by 2035 in the US\(^3\)

Health Consequences are Significant

- **10 to 17x** increase in liver-related mortality risk for F2, F3 patients\(^4\)
- Increased risk of liver failure, transplant, hepatocellular carcinoma and CV events\(^5,6,7,8,9\)

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\(^1\) National Institute of Diabetes and Digestive and Kidney Diseases, Definition and Facts of NAFLD and NASH.  
\(^2\) National Institute of Diabetes and Digestive and Kidney Diseases, Treatment for NAFLD & NASH.  
\(^5\) Organ Procurement and Transplantation Network Liver Transplant data, March 2019.  
Pfizer’s Strategy is to Address the Underlying Metabolic Engine that Drives Disease Pathogenesis

- Normal liver
- Steatosis
- NASH with or without fibrosis
- Cirrhosis

Disordered metabolism → Inflammation and cellular injury → Progressive fibrosis

Anti-steatotic → Anti-inflammatory → Anti-fibrotic

Hepatocellular Carcinoma
Liver Failure
Esophageal Varices
Complementary Mechanisms of Action for DGAT2 and ACC Inhibitors Offer Potential for Best-in-Class Therapy

SREBP = sterol regulatory element-binding protein. TG = Triglyceride.

DGAT2 Inhibition with Ervogastat (PF-06865571) Demonstrated Reductions in Steatosis and Serum Triglyceride

Liver Fat Content (% relative change from baseline)

- Placebo
- 50 mg Q12
- 300 mg Q12

21 – 26% decrease in serum TG

ACC Inhibition with Clesacostat (PF-05221304) Demonstrated Statistically Significant Reductions in Steatosis and ALT, but Increases in Serum Triglycerides

HYPOTHESIS: Co-administration of DGAT2i will mechanistically mitigate the TG increase observed with ACCi via off-setting regulation of SREBP

ALT = alanine aminotransaminase.

Clesacostat / Ervogastat Maintains Statistically Significant Liver Fat Lowering and Mitigates Serum Triglyceride Increases of ACCi

Placebo (BID) DGAT2i (300 mg BID) ACCi (15 mg BID) ACCi/DGAT2i (15 mg/300 mg BID)

Number of subjects evaluable

>400 mg/dl 2 (14.3) 2 (7.4) 11 (37.9) 3 (11.5)
>600 mg/dl 1 (7.1) 1 (3.7) 4 (13.8) 0
>800 mg/dl 0 0 3 (10.3) 0

We are Positioned to Advance Potential First- or Best-in-Class NASH Compounds

Timings are approximate and subject to change.

Potential Subpart H NDA Submission
Potential Conditional Approval
Addressing a metabolic epidemic with the potential first small molecule oral GLP-1RA for both T2D and Obesity
Oral GLP-1RA Has the Potential to Address Treatment Gaps in Two Global Epidemics: Type 2 Diabetes and Obesity

Rising Rates of Diabetes and Obesity Carry Significant Health Consequences

**Diabetes**

- **463 million** today
- By 2030: **578 million**
- In US, only ~50% have HbA1c below treatment goal

**Obesity**

- **650 million** today
- By 2030: **1.12 billion**
- Increased comorbidity risk and development of >200 chronic diseases

Injectable GLP-1RAs Are Underutilized – Potential for Oral GLP-1RA to Address This Gap

- While GLP-1RAs have 44.7% share of non-insulin diabetes $ sales in US, they only have **9.6% share of total prescriptions**
- About 1 in 3 oral DPP4i prescribers for diabetes in US have not written a single prescription for injectable GLP-1RAs
- Ex-US represents 35% of global diabetes non-insulin sales but only **15% of GLP-1RA sales**

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2. WHO. Obesity and overweight. (February 2018) https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight;
5. Obesity as a Disease: The Obesity Society 2018 Position Statement.
7. IQVIA MIDAS, Xponent
Innovative Research Led to Discovery of Breakthrough Small Molecule GLP-1RA That Differentiates from Injectable and Oral Peptide Class

Clinical Candidate

Danuglipron

Phase 2

Potential to Offer a Uniquely Differentiated Profile

- Expected to deliver potent effects on blood sugar and weight loss
- Expected to have safety and tolerability comparable to peptide GLP-1RA class with a convenient oral formulation
- Good oral bioavailability
- No expected food or dose restrictions, unlike large molecule oral GLP-1RAs
- Believed to be suitable for monotherapy or combination therapies

HYPOTHESIS: Danuglipron may differentiate from injectable and oral peptide-based GLP-1RAs based on its oral absorption profile
Danuglipron Demonstrated Robust Reduction in Fasting Glucose, HbA1c and Body Weight at 28 Days in Type 2 Diabetes

Subjects with T2D on Stable Metformin Background
Baseline: BMI 32.9 kg/m²
Baseline: HbA1c: 8.3%

Dosed twice daily
PBO, 15 mg, 70 mg, 120 mg*

Consistent with the GLP-1RA class, nausea, vomiting and diarrhea were most common AEs; dose-dependent increases of these AEs were observed

*Represents a subset of the doses administered in the study.
We Have the Potential to Dramatically Improve Treatment of Diabetes and Obesity with Our Small Molecule Oral GLP-1RA, Danuglipron

Timings are approximate and subject to change.
Our aspiration is to reduce the morbidity and mortality burden of these highly prevalent cardiometabolic diseases

**NASH**: 18 million US patients, ~30% have F2/F3\(^1,2\)

**CVD**: >6 million US patients with CVD or diabetes are at high risk, despite statin treatment\(^3\)

**T2D**: Only ~50% of US patients have HbA1c below treatment goal\(^4\)

**Obesity**: Increased comorbidity risk and development of >200 chronic diseases\(^5,6\)
Thank You