Investor Presentation

June 2022

Forward-Looking Statements

This presentation contains “forward-looking” statements about Annexon, Inc. and our industry that involve substantial risks and uncertainties. All statements other than statements of historical facts, including statements regarding our clinical and preclinical programs, timing and commencement of future nonclinical studies and clinical trials and research and development programs, timing of clinical results, strategic plans for our business and product candidates, including additional indications which we may pursue, our financial position, runway and anticipated milestones, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “aim,” “anticipate,” “assume,” “believe,” “contemplate,” “continue,” “could,” “design,” “due,” “estimate,” “expect,” “focus,” “goal,” “intend,” “may,” “objective,” “plan,” “positioned,” “potential,” “predict,” “seek,” “should,” “target,” “will,” “would” and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology.

Forward-looking statements are not guarantees of future performance and are subject to risks and uncertainties that could cause actual results and events to differ materially from those anticipated, including, but not limited to, risks and uncertainties related to: our history of net operating losses; our ability to obtain necessary capital to fund our clinical programs; the early stages of clinical development of our product candidates; the effects of COVID-19 or other public health crises on our clinical programs and business operations; our ability to obtain regulatory approval of and successfully commercialize our product candidates; any undesirable side effects or other properties of our product candidates; our reliance on third-party suppliers and manufacturers; the outcomes of any future collaboration agreements; and our ability to adequately maintain intellectual property rights for our product candidates. These and other risks are described in greater detail under the section titled “Risk Factors” contained in our Annual Report on Form 10-K filed with the Securities Exchange Commission (SEC) on March 1, 2022, Form 10-Q filed with the SEC on May 9, 2022, and our other filings filed with the SEC from time to time. All forward-looking statements in this presentation speak only as of the date of this presentation. Except as required by law, we undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise.

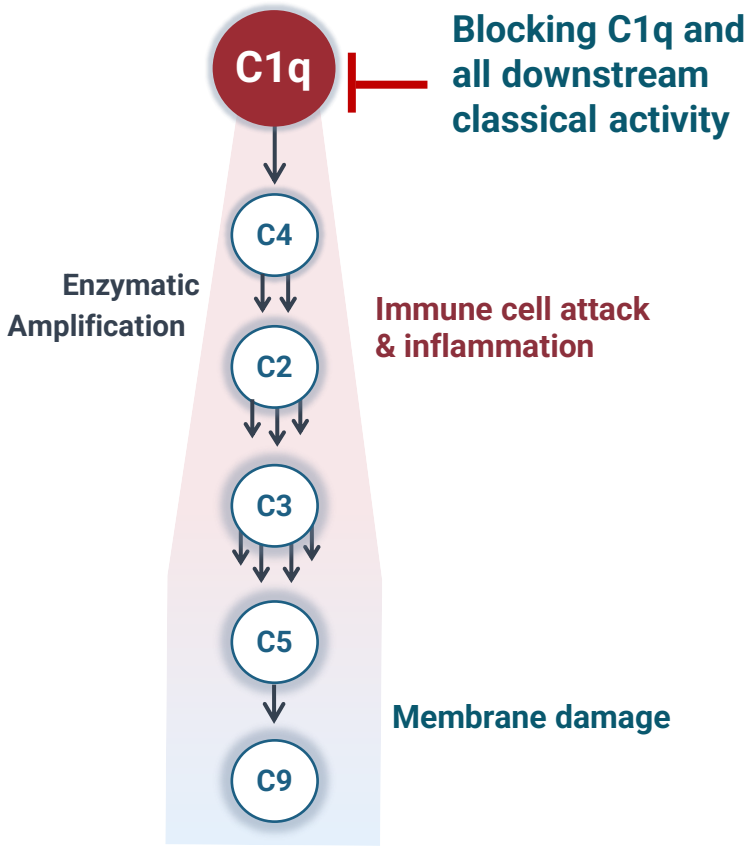
This presentation concerns drug candidates that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA). These are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates or statistical data. Neither we nor any other person makes any representation as to the accuracy or completeness of such data or undertakes any obligation to update such data after the date of this presentation.

ANNEXON biosciences

Pioneering a Powerful Approach to Classical Complement Inhibition

Classical Complement Pathway



- Complement inhibition is a **validated clinical and commercial** approach
- Annexon's next-generation approach blocks **both upstream & downstream complement** for enhanced outcomes
- Advancing **5 fit-for-purpose drug candidates** for complement-mediated diseases of the body, brain & eye
- **Clinical benefit with ANX005 shown** in multiple indications
- Well-positioned to drive significant value with **7 clinical trial readouts anticipated in 2022-2023**

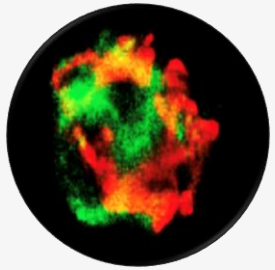
C1q: A Key Driver of Complement-Mediated Disease

Initiator of aberrant or excess complement activity in autoimmune and neurodegenerative diseases

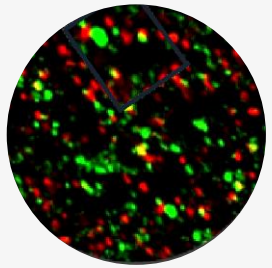
Key Takeaways

- C1q is initiating molecule in classical complement cascade
- C1q binds tissue surfaces to **anchor and amplify** complement activation and drive disease
- **C1q marks cells and synapses for elimination by microglia and tissue macrophages**

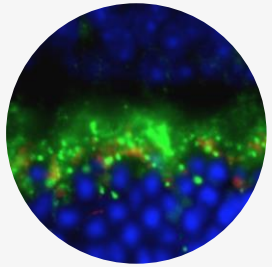
Initiator: C1q Binding to Tissues in Disease



Autoimmune
Guillain-Barré Syndrome
C1q Targeting the Neuromuscular Junction[1]



Neurodegeneration
Huntington's Disease
C1q Targeting Striatal Synapses[2]



Ophthalmologic
Geographic Atrophy
C1q Targeting Photoreceptor Synapses[3]

[1] Halstead, et al. 2004 Brain 127: 2109–2123 [2] Jia, et al, 2018 Mol Neurodegen 14:45 [3] C1q bound to photoreceptor synapses in aged mice: Annexon data on file

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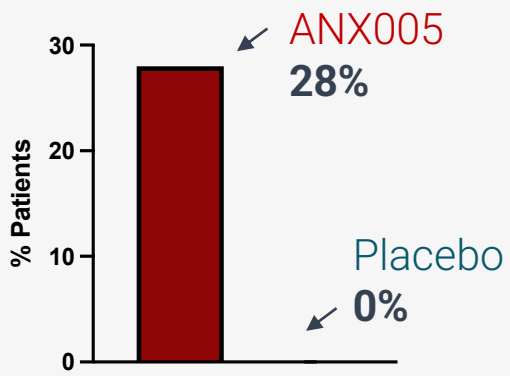
Upstream Classical Complement Inhibition Associated with Clinical Benefit

Guillain-Barré Syndrome

Patients achieving **≥3 point improvement** in 8 weeks

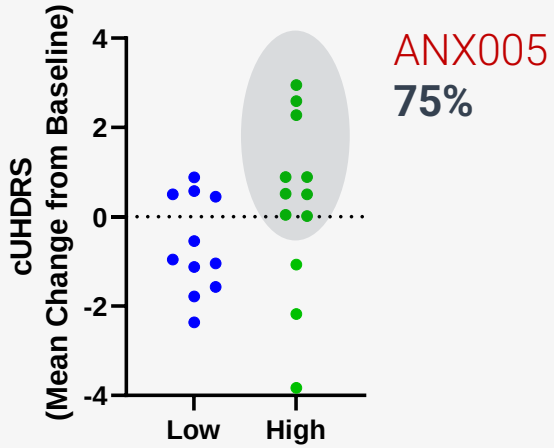


GBS Disability Scale

Patients improved from being bed bound to walking unassisted

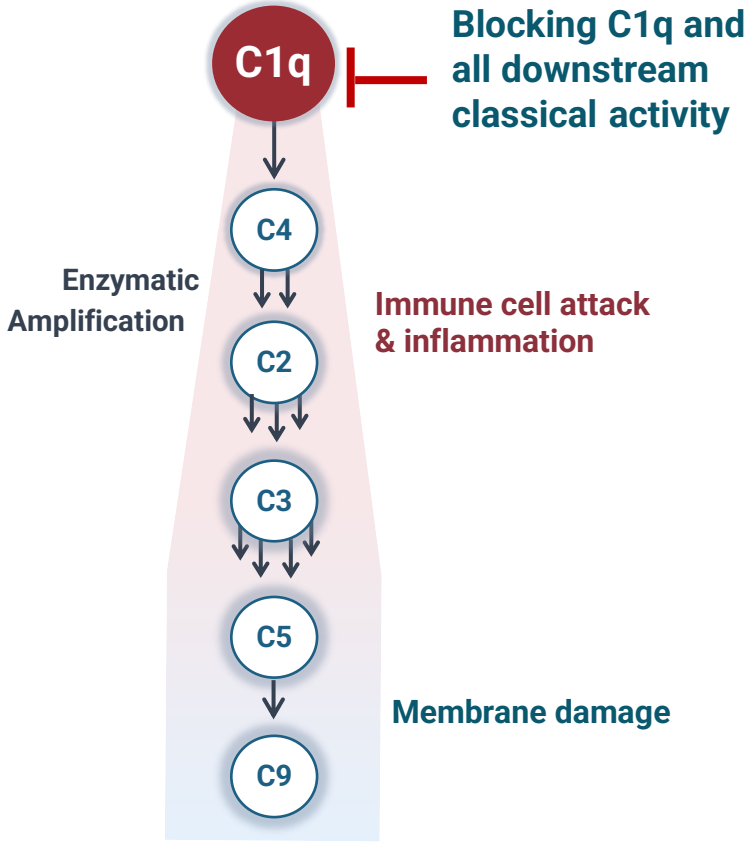
Huntington's Disease

75% of patients with high baseline complement activity maintained improvement at week 36



Composite Unified Huntington's Disease Rating Scale

Classical Complement Pathway



Annexon data on file.
GBS Disability Scale and composite Unified Huntington's Disease Rating Scale are accepted regulatory endpoints.

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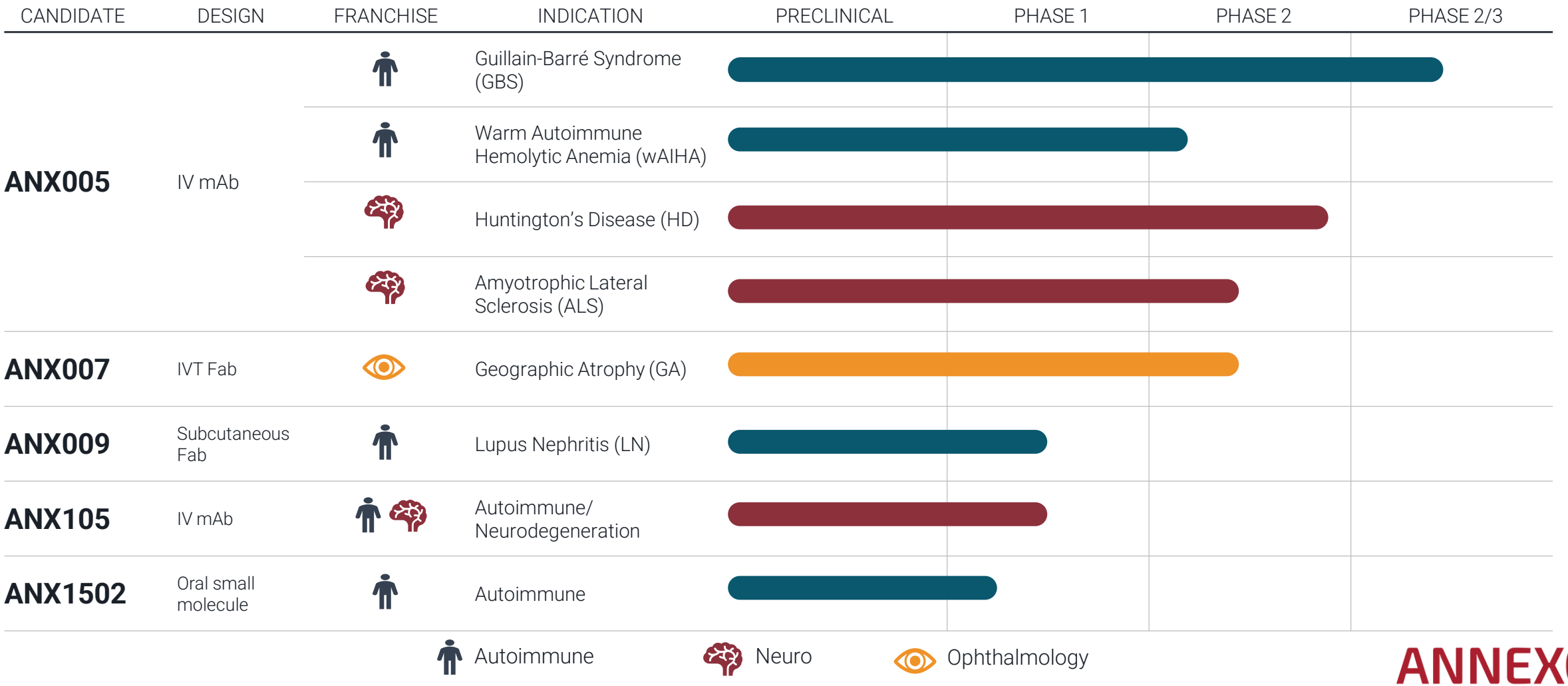
Advancing Pipeline of 'Fit for Purpose' Drug Candidates Across Multiple Complement-Targeted Diseases

Targeting Both Rare & Large Patient Populations



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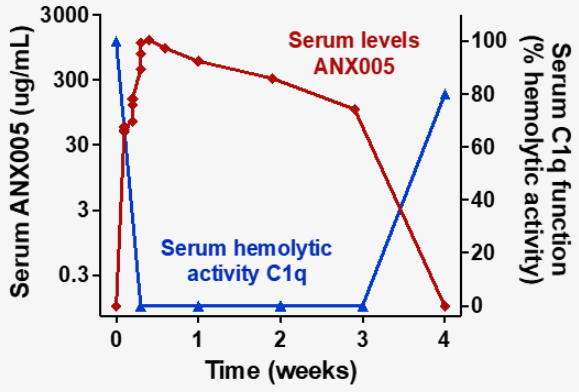
Lead Candidates Demonstrated Robust Target Engagement in Body, Brain and Eye in Clinical Trials

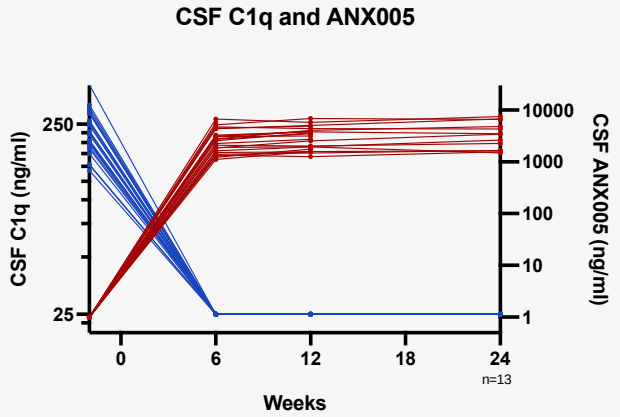
C1q Body Inhibition

ANX005 Full C1q Inhibition in Serum



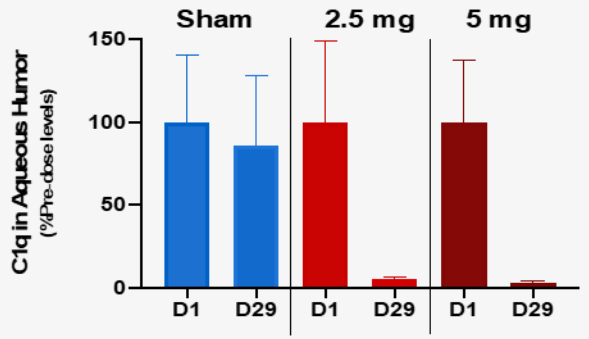
C1q Brain Inhibition

ANX005 Full C1q Inhibition in CSF



C1q Eye Inhibition

ANX007 Full C1q Inhibition in Aqueous Humor



D1 = Day 1 (before ANX007 dosing)

D29 = Day 29 (post-1st dose)

Autoimmune

Neuro

Ophthalmology

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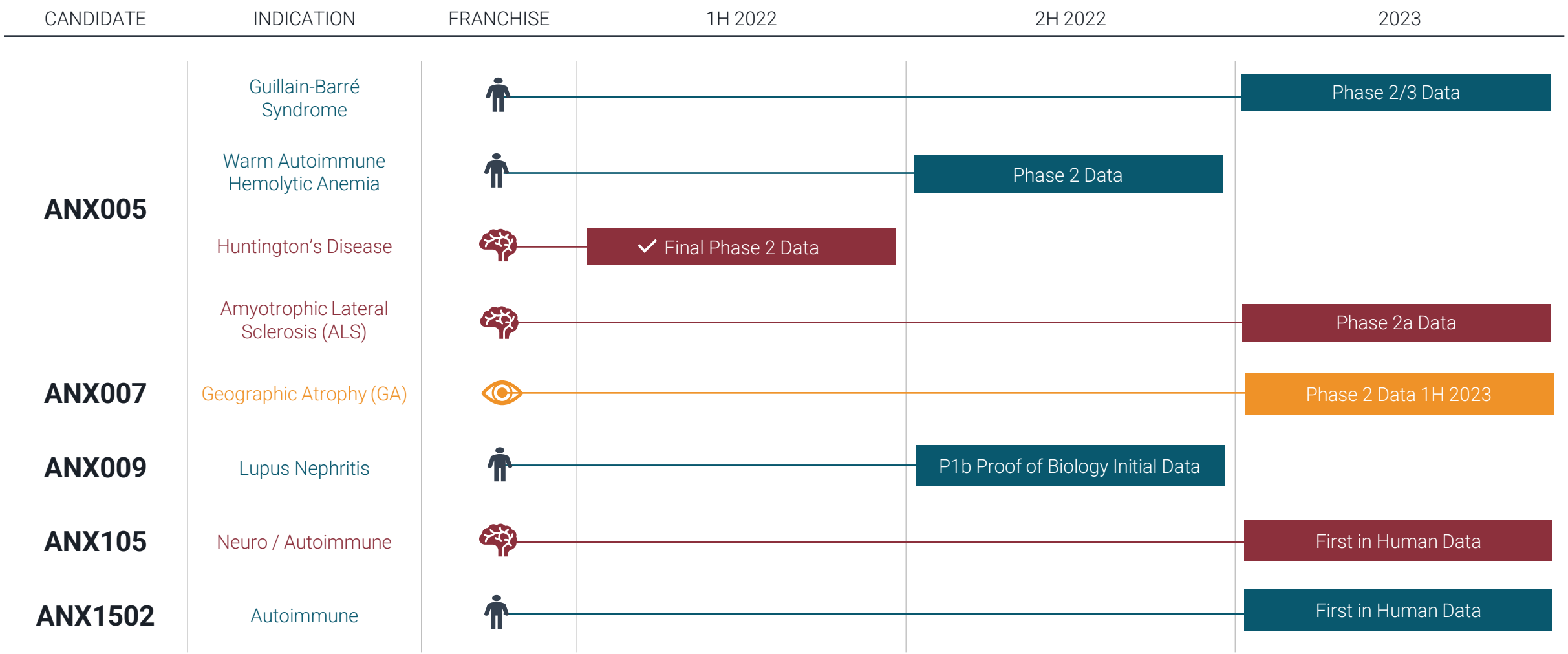
Multiple Value-Creating Opportunities in 2022 and 2023



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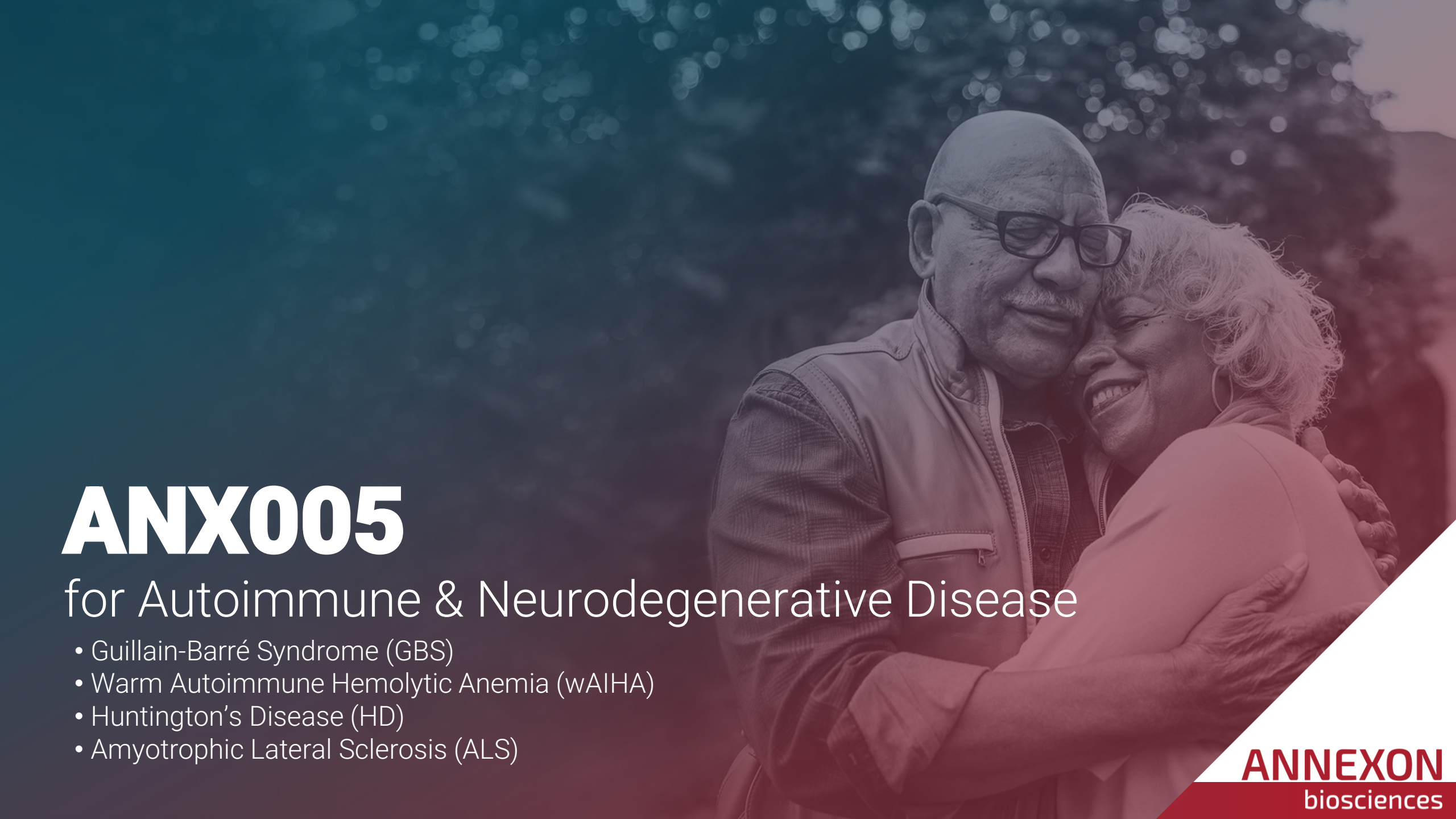

ANX005

for Autoimmune & Neurodegenerative Disease

- Guillain-Barré Syndrome (GBS)
- Warm Autoimmune Hemolytic Anemia (wAIHA)
- Huntington's Disease (HD)
- Amyotrophic Lateral Sclerosis (ALS)

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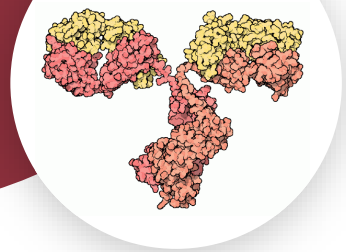
ANX005: Designed to Fully Inhibit C1q and the Entire Classical Complement Pathway

ANX005



ANX005 Key Attributes

- ✓ Full-length monoclonal antibody formulated for intravenous administration
- ✓ **Fully inhibits C1q in the body and brain**, with complete target engagement in blood and CSF
- ✓ **Well-tolerated** in clinical trials; **>170 patients treated to date**
- ✓ Demonstrated **early improvement in clinical outcomes** for patients with Guillain-Barré Syndrome and Huntington's Disease
- ✓ High binding affinity (<10 pM)

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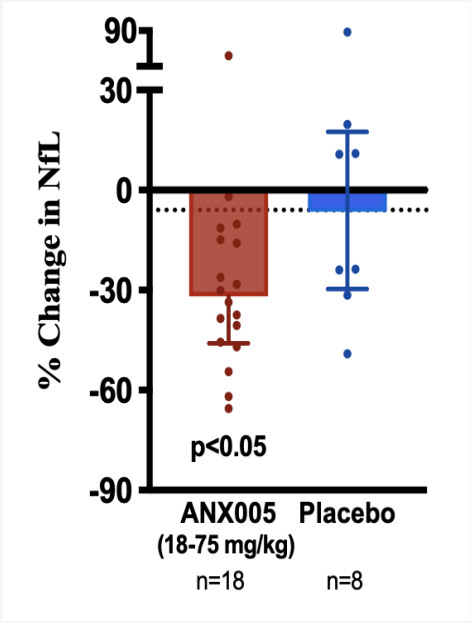
ANX005 for GBS
Inhibiting Upstream to Stop Downstream Neuroinflammation

Guillain-Barré Syndrome Overview

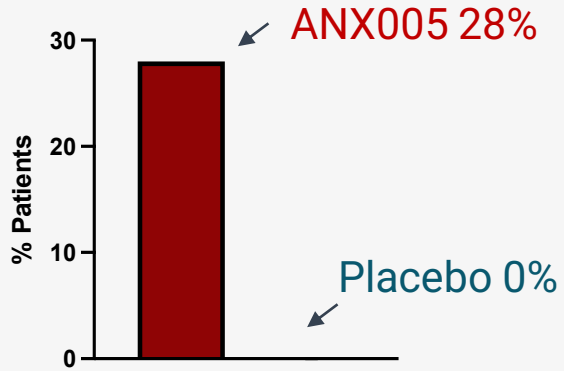
- **C1q binds autoantibodies on nerves and amplifies complement** cascade
- **Inhibiting C1q with ANX005 stops downstream activation** of tissue-damaging components in GBS
- In POC trial, ANX005 was well-tolerated, achieved full target engagement, **early reduction in neuronal death and improvement in muscle strength preceding gain of function**

ANX005 POC Data in GBS

Statistically Significant Early NfL Reduction (Weeks 2-4)



Patients achieving ≥3 point improvement in 8 weeks



GBS 6-point disability scale:

1. Slight symptoms
2. Walk / no running
3. Walk with support
4. Bedridden / chair bound
5. Ventilator-assisted breathing
6. Death

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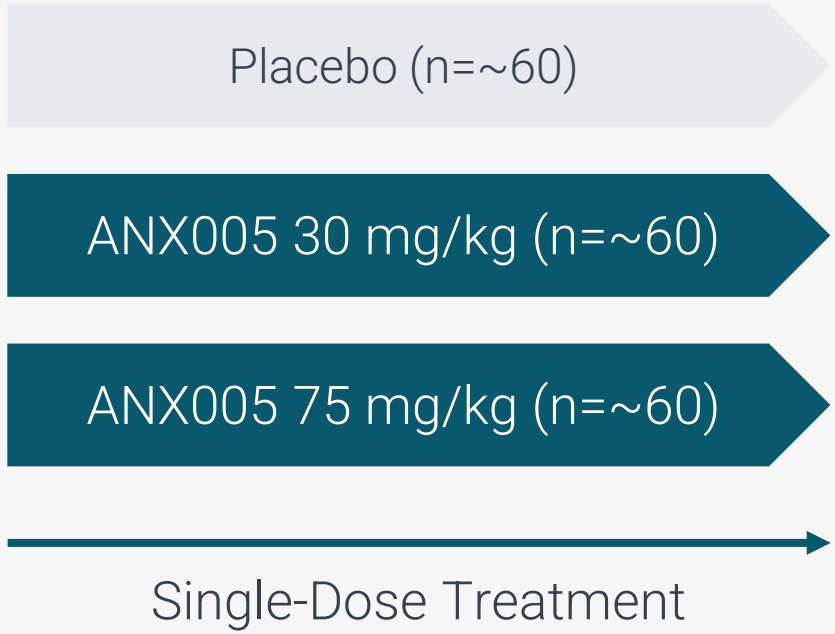
Placebo-Controlled Phase 2/3 GBS Trial Ongoing with Data Anticipated in 2023

Trial Design



Specifications

- Randomized, double-blind trial (N=180)
- Primary endpoint: GBS Disability Scale
- Patients stratified for baseline muscle strength and time from symptom onset
- Phase 2/3 data anticipated 2023

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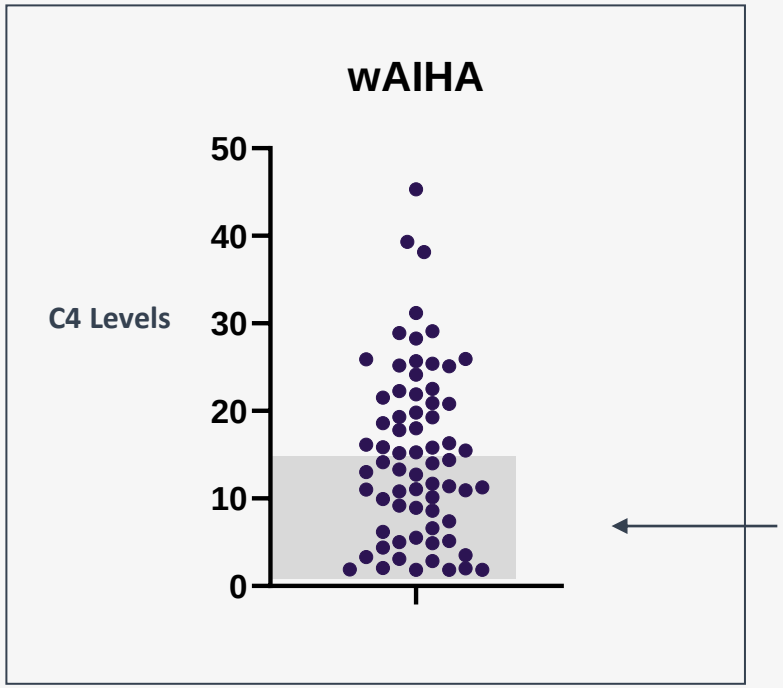
ANX005 for wAIHA
Inhibiting Upstream to Stop Complement-Mediated Disease

Data from ongoing Phase 2 trial anticipated in 2H22

Warm Autoimmune Hemolytic Anemia Overview[1]

- **C1q binds to red blood cells on autoantibody coated surface causing anemia**
  - IgG and IgM antibodies
- **C1q and complement activation amplify RBC destruction in ~25-50% of patients**
  - Complement levels correlate with level of hemolysis
- **Selecting patients with high baseline complement activity** who have a similar profile to CAD patients
- **ANX005 achieves full suppression of complement-mediated hemolysis**

Patients with Ongoing Complement Activity (consumption)



[1]Affects ~47,000 US patients per year

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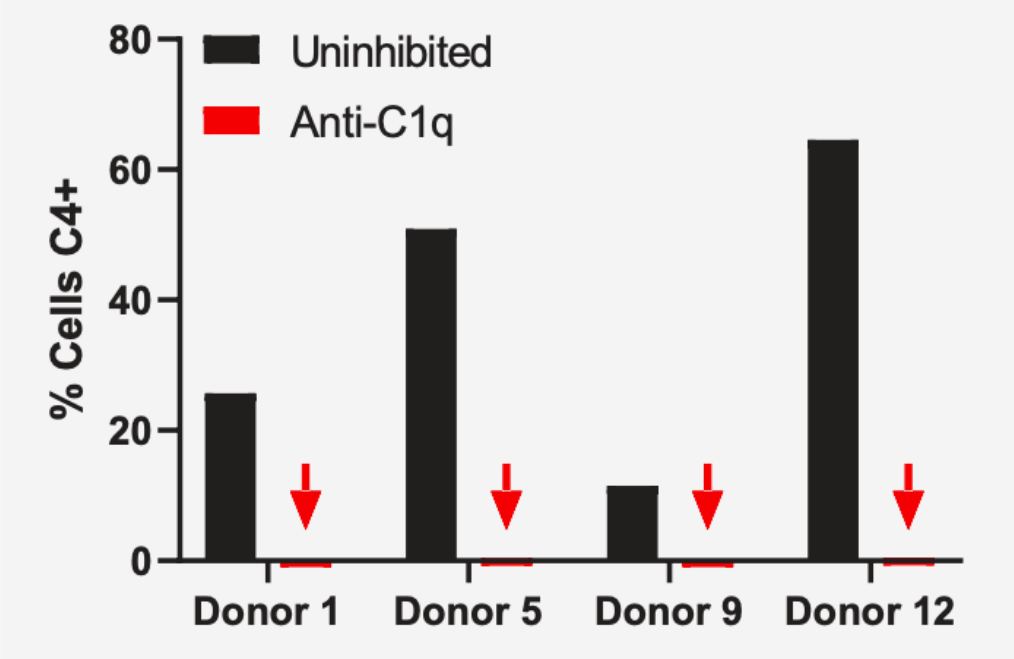
ANX005 Blocks Complement Activity in wAIHA Patient Sera

Ex Vivo Blockade of C4 Deposition on RBC in wAIHA Patient Sera with ANX005



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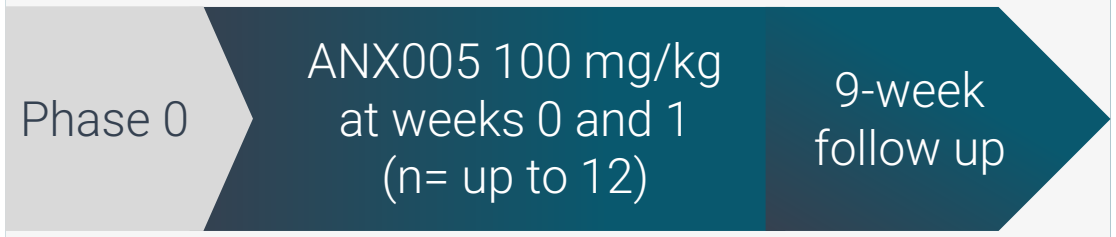
ANX005 Phase 2 Trial for wAIHA Underway; Clinical Data Anticipated in 2H 2022

Trial Design



Specifications

- Open label trial (n= up to 12); on and off treatment assessments
- Using Phase 0 'feeder' study to identify/select patients for Phase 2
- **Objective endpoints: safety, PK/PD, hemolysis markers, improvement in hemoglobin**
- Phase 2 data anticipated 2H 2022

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ANX005 for Huntington's Disease (HD)
Inhibiting Upstream to Protect Functioning Synapses

No approved therapies that slow or stop disease progression

- **Inherited, fatal disease** that causes progressive synapse damage and loss associated with neuroinflammation and loss of neurons
- **Affects ~80K people globally**, with ~300K at risk of inheriting the disease-causing HD gene[1]
- Life expectancy after diagnosis estimated to be **15-20 years**
- **Progressive decline** in motor, cognitive and psychiatric function

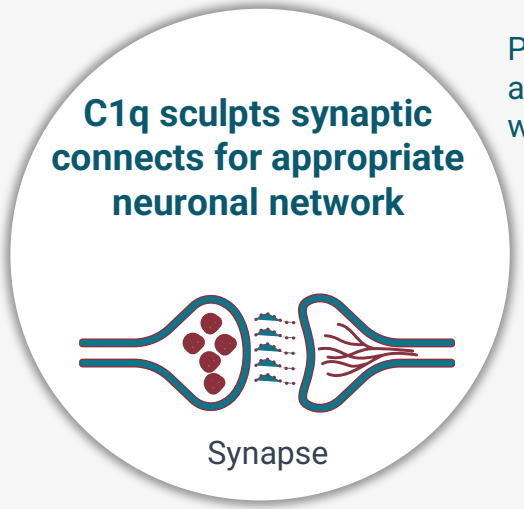
[1]GlobalData and market research reports

Loss of Functioning Synapses Results in Neurodegeneration

Blocking C1q protects functioning synapses, prevents loss and decreases disability[2]

C1q's Normal Role In Development

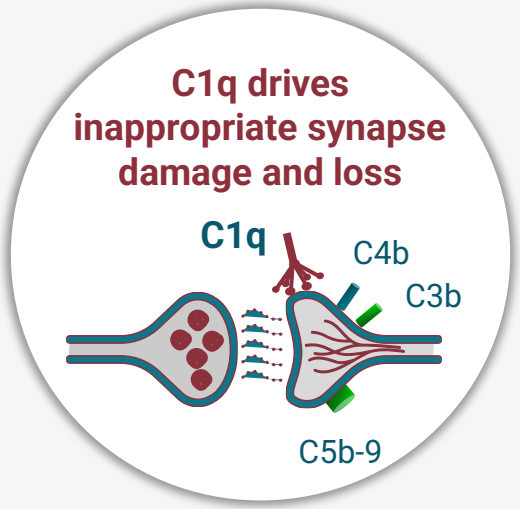
- C1q selectively recognizes, tags & drives removal of excess synapses
- Strong synapses remain to form appropriate circuits and normal brain health



Pathway turns off after developmental window

C1q's Role In Neurodegenerative Disease

- C1q recognizes, tags & drives removal of functioning synapses
- Triggers inappropriate synapse damage and loss, neuroinflammation and degeneration



Chronic pathway activation and functional decline

[1]Selkoe, 2002 *Science* **298**:789 | [2]Wilton 2021 doi.org/10.1101/2021.12.03.471180; Hong 2016 *Science* 10.1126/science.aad8373; Stevens 2007 *Cell* DOI 10.1016/j.cell.2007.10.036; Fonseca, 2004, *J Neurosci*; Dejanovic, 2018, *Neuron*; Vukojicic, 2019, *Cell Reports*; Howell, 2011, *J Clin Inves*; Williams, 2016, *Mol Neurodegen*; Jiao, 2018, *Mol Neurodeg*; Lui, 2016, Cell **165**:921; Krukowski, 2018, Int.*J Mol Sci*; Holden, 2021, *Science*; Annexon NfL reduction in SOD1 model, unpublished; Absinta, *Nature*, 2021

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Phase 2 Open-label Clinical Trial of ANX005 in Patients With, or at Risk for, Manifest Huntington's Disease

6-month treatment period (n=28) | **3-month follow-up**

Induction dosing of ANX005 administered by IV infusion on Days 1 and 5 or 6, followed by maintenance dosing every 2 weeks through Week 22

Follow up visits on Weeks 24, 28, and 36

Study Population

- Adults with, or at risk for, manifest HD ("Early HD")
- Total CAP score >400
- UHDRS independence score ≥ 80%

Primary & Secondary Endpoints

- Safety and tolerability of ANX005
- PK of ANX005 in serum & cerebrospinal fluid (CSF)
- PD as measured by C1q, NfL, and C4a serum and CSF concentrations

Exploratory Endpoints

- Composite UHDRS and its components

Final Clinical Study Findings Include:

- Target engagement, clinical measures and NfL in patients who completed both treatment and follow-up periods (n=23)
- Clinical measures in patients with high vs. low C4a complement (n=23)
- Safety and tolerability (n=28)

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ANX005 Phase 2 Trial Patient Baseline Demographics

Study Participant Characteristics	All Patients % (N=28)	Treatment Completers % (N=23)	TRACK-HD* % (N=123)
Age, mean SD, years	49.7 (12.5)	48.5 (13.3)	48.8 (9.8)
Female, %	42.9	34.8	45
CAG repeat length mean (SD)	44.6 (3.5)	45.1 (3.7)	43.7 (3.0)
CAP score mean, (SD)	505.7 (57.9)	512.2 (60.4)	NR
Manifest HD, n (%)	25 (89.3)	21 (91.3)	123 (100)
CSF C4a, mean (SD) ng/mL	13.9 (8.2)	15.0 (7.0)	NR
Baseline plasma NfL, mean (SD) pg/mL	40.1 (13.7)	41.3 (13.3)	NR
Baseline CSF NfL, mean (SD) pg/mL	3104.1 (810.8)	3236.0 (816.8)	NR
cUHDRS, mean (SD)	10.4 (3.2)	10.1 (2.9)	11.7 (2.9)
Total Functional Capacity , mean (SD)	10.6 (2.2)	10.4 (2.3)	10.9 (2.0)
Total Motor Score, mean (SD)	21.6 (12.6)	22.3 (11.4)	23.7 (10.8)
Symbol Digit Modalities Test, mean (SD)	29.7 (11.3)	28.8 (11.0)	33.6 (10.2)
Stroop Word Reading Test, mean (SD)	59.0 (18.7)	56.7 (16.7)	78.3 (19.5)

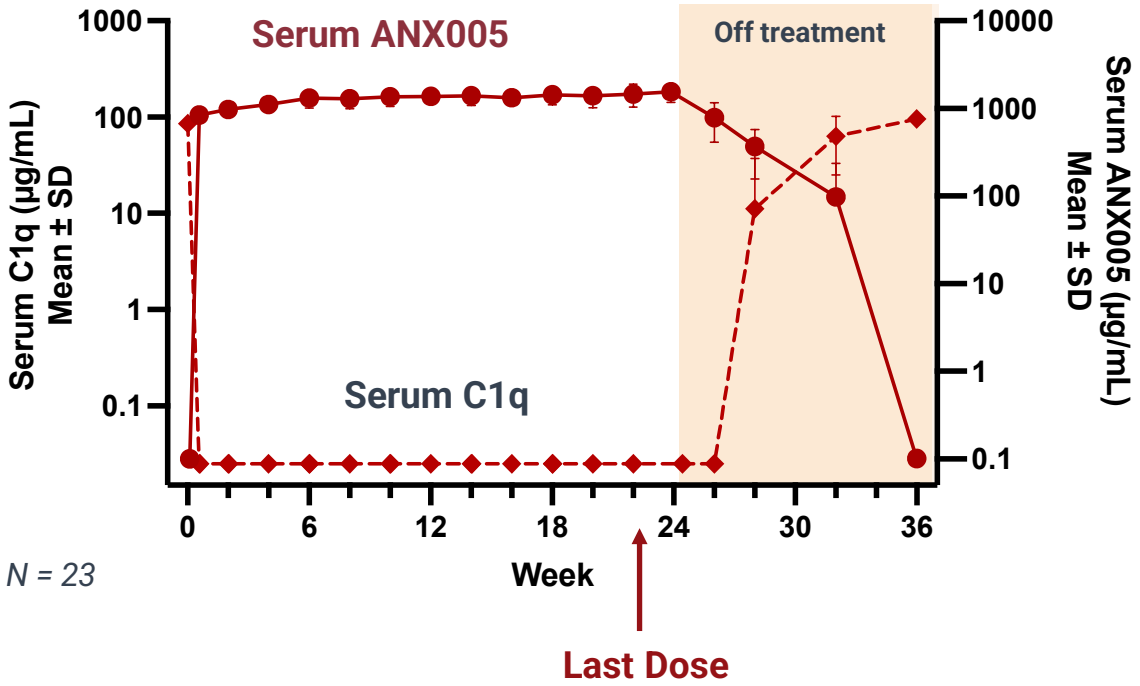
- ~ 90% of patients had “early manifest HD”
- Demographics consistent with prior HD natural history study cohorts (e.g., TRACK-HD*)

* TRACK-HD, HD natural history study. For illustrative purposes only - differences exist between patient demographics, study designs, and other factors and caution should be exercised when comparing data across studies.
NR=not reported

ANX005 Demonstrated Complete & Durable C1q Inhibition in Blood and CSF

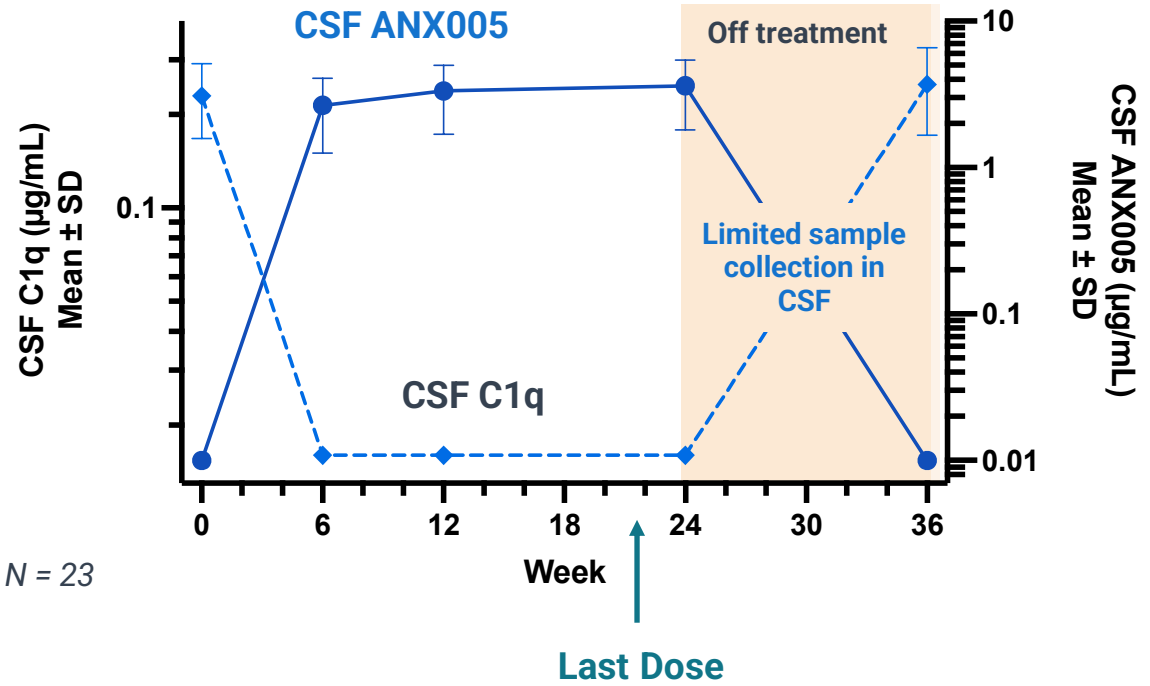
Target engagement 4-10 weeks in serum post last dose; may support less frequent dosing

Full Target Engagement in Serum



N = 23

Full Target Engagement in CSF



N = 23

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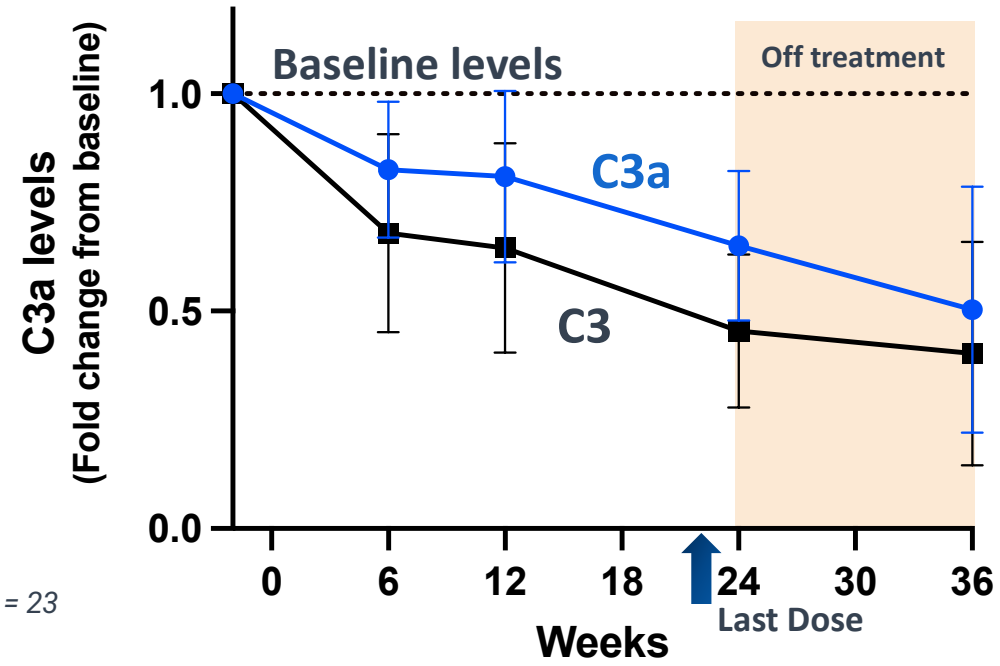
Evidence of Reduced Downstream Complement Activation & Neuroinflammation Through Entire 9 Month Study

Drug Effects Continue into Off-treatment Period



N = 23

ANX005 showed:

- Reduction of downstream complement activation (C3a)
- Reduction of neuroinflammation (C3)
  - C3 is produced by activated, neurotoxic astrocytes in the brain[1]

[1]Liddelow, Barres, 2017 *Nature* **541**: 481–487

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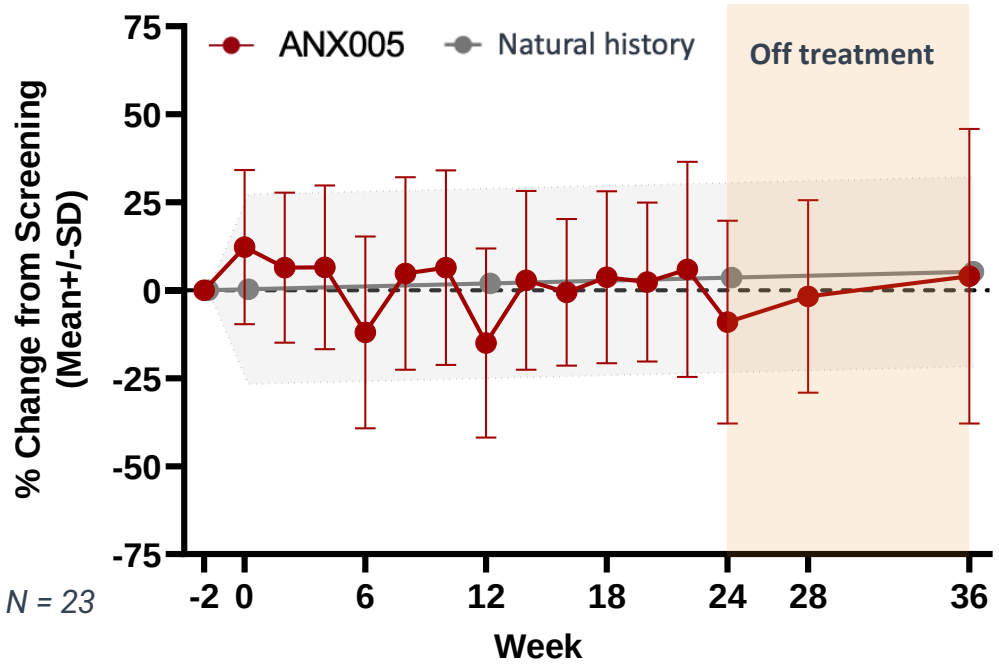
NfL Changes Stable and Consistent with Natural History Through Treatment and Follow-up Periods

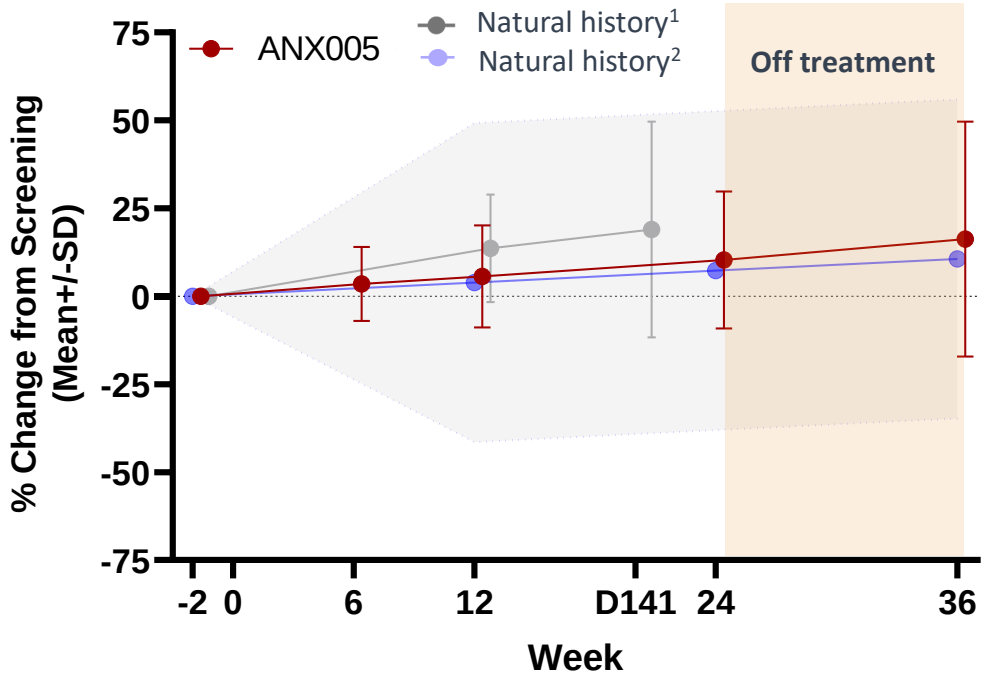
Plasma NfL



N = 23

[1]Interpolated data for manifest cohort; Rodriguez, et al., Sci Transl Med. 2020 12 16;12(574)

CSF NfL



[1]Tabrizi, NEJM 2019, 38:2307
[2]Interpolated from Rodrigues, et al., Sci Transl Med. 2020 12 16;12(574)

Results independent of baseline complement activity

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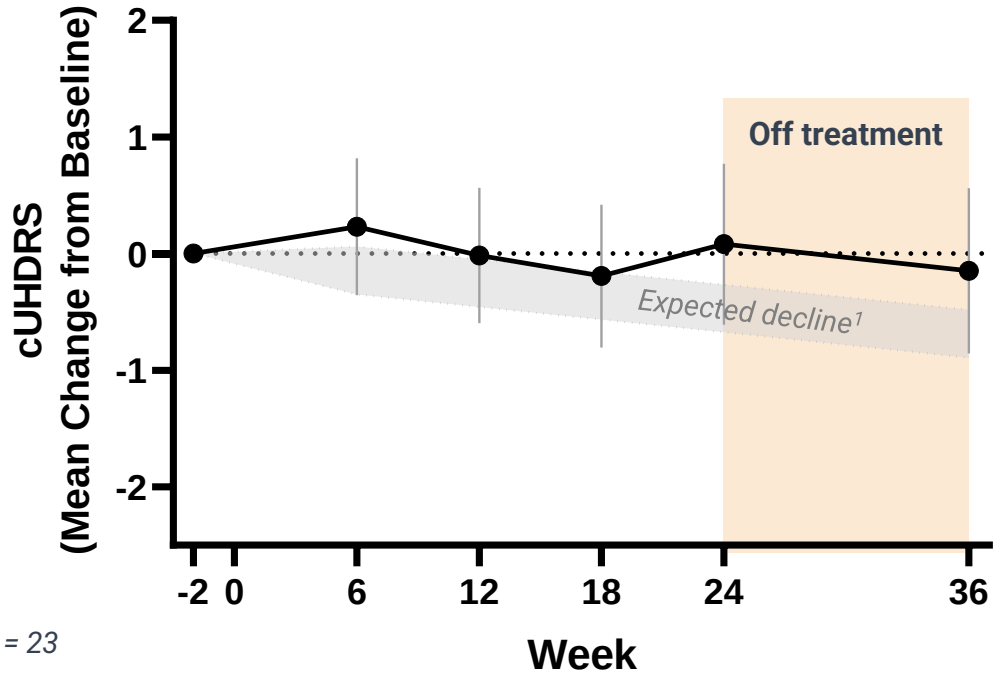
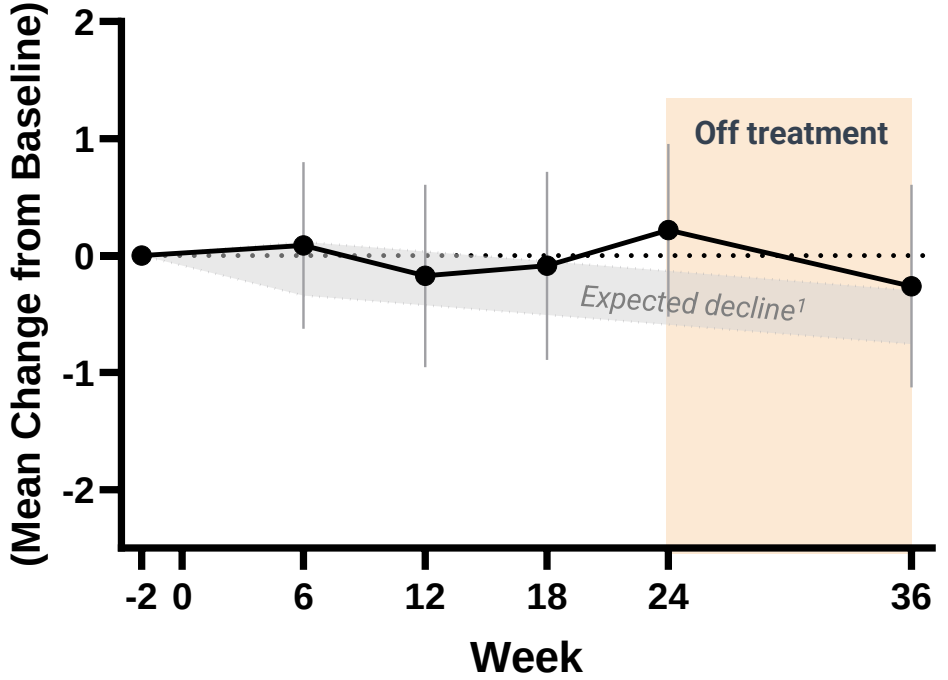
Clinical Disease Progression Stable in Overall Patient Population Through Entire 9-month Study

cUHDRS Stable Over 9 Months



N = 23

TFC Stable Over 9 Months



MMRM; LS means +/- 95% CI

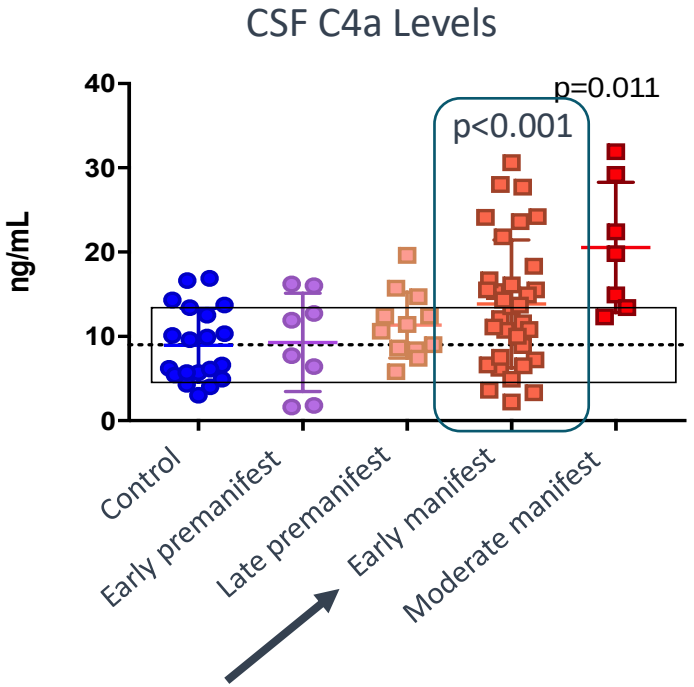
[1] Expected decline = interpolated natural history from Schobel 2017 (TRACK-HD)

ANNEXON biosciences

Complement Activation Correlates with Disease and Functional Decline in HD

Patients with higher baseline complement activity may be more likely to respond to anti-C1q therapy

CSF C4a Elevated and Increase with HD Progression*



Early manifest consistent with patients enrolled in current study

CSF C4a Activation Correlate with HD Functional Decline

Clinical endpoints	p-value	
Total functional score (TFC)	0.0333	Motor & Function
Total motor score (TMS)	0.0181	
Disease burden score (DBS)	0.1310	
Symbol digit mod. Test (SDMT)	0.0324	Cognitive Scales
Verbal fluency	0.0255	
Stroop color naming (SCN)	0.0454	
Stroop word recall (SWR)	0.0710	

*Higher complement activity in CSF (C4a) of HD Patients associated with disease severity & functional decline
Presented at HSG, November 2021; Annexon Collaboration with Ed Wild UCL

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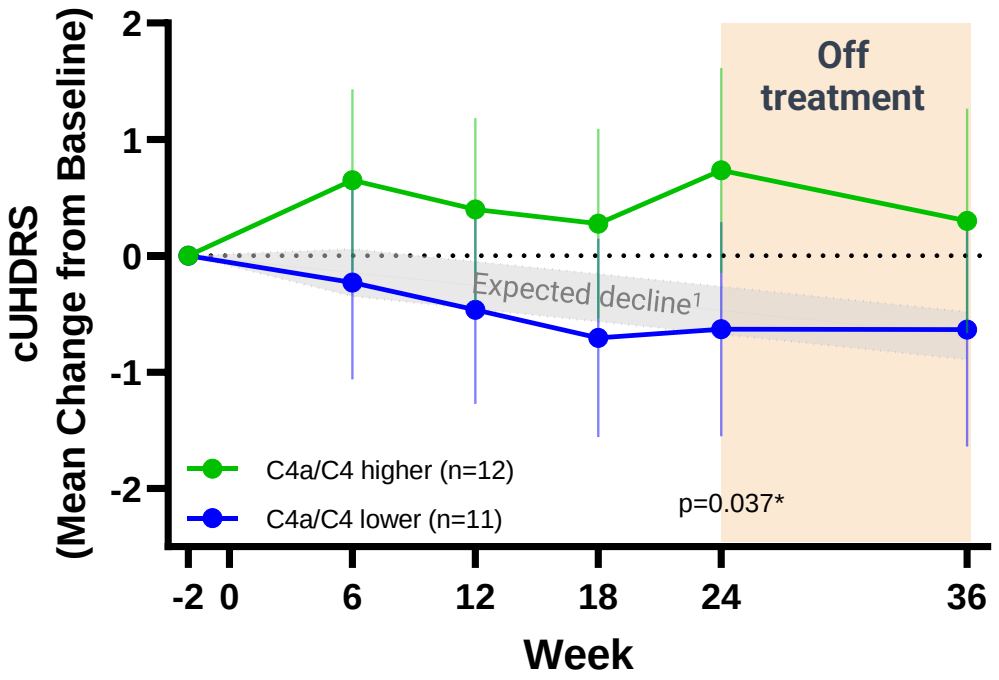
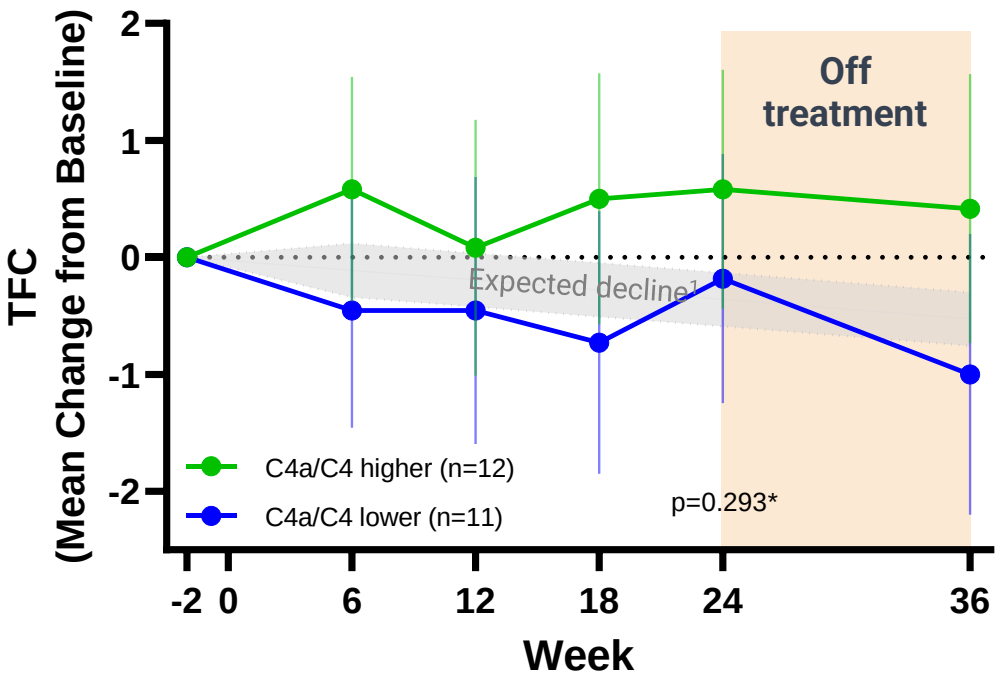
Rapid Benefit Maintained in Patients with High Baseline Complement Activity Through Treatment and Follow-up Periods

Benefit at All Time Points in High Complement Group (cUHDRS)



Benefit at All Time Points in High Complement Group (TFC)



*Comparing higher vs lower C4a/C4 groups at week 2; MMRM; LS means +/- 95% CI; N=23
[1]Expected decline = interpolated natural history from Schobel 2017 (TRACK-HD)

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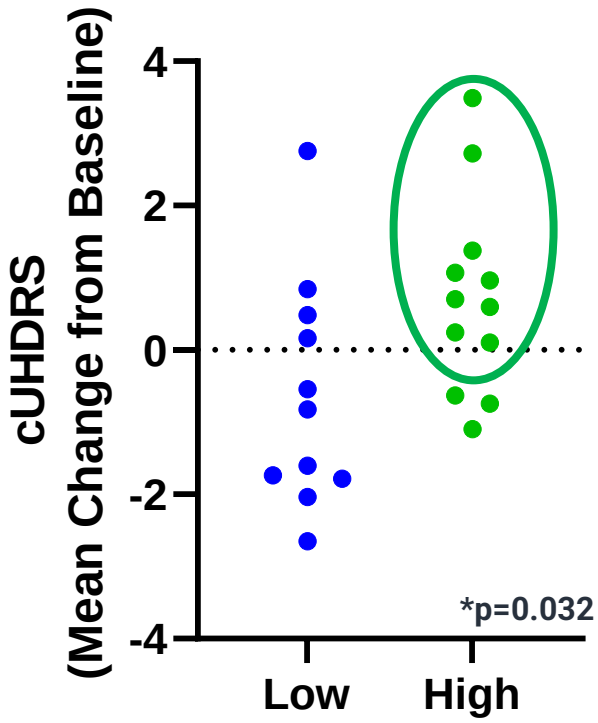
75% of Patients with High Baseline Complement Levels Showed Improvement at Week 24, Maintained at Week 36

Twice as many patients with high complement improved compared to patients with low complement

Week 24 cUHDRS
Change from Baseline

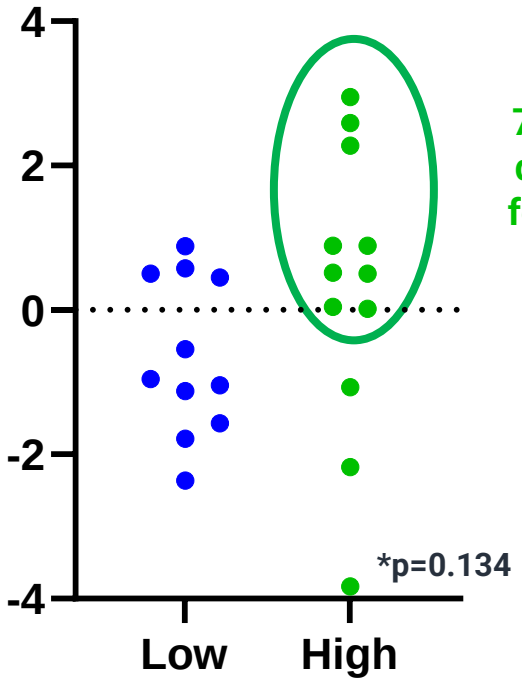


75% patients with elevated baseline complement improved (9/12)

vs.

36% with lower baseline complement (4/11)

Week 36 cUHDRS
Change from Baseline



75% maintained during 3-month follow-up period

Baseline Complement Activity (C4a/C4)

Baseline demographics evenly matched between patients with higher and lower CSF complement activation
*Wilcoxon-Mann-Whitney Test

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Chronic Dosing of ANX005 Generally Well-Tolerated with Favorable Benefit-Risk Profile Demonstrated in HD

Treatment Emergent Adverse Events	Safety Population (N=28)	
	All Grades, N (%)	Grade 3, N (%)
Any reported TEAEs	28 (100.0)	12 (42.9)
Most Common TEAE		
Infusion Related Reactions (IRR)	28 (100.0)	8 (28.6)
Most Common TEAEs (non-IRR)	25 (89.3)	6 (21.4)
Dizziness	5 (17.9)	0 (0)
Nausea	5 (17.9)	0 (0)
Headache	4 (14.3)	0 (0)
Vomiting	4 (14.3)	0 (0)
COVID-19	4 (14.3)	0 (0)
Rash	4 (14.3)	1 (3.6)
Serious TEAEs	2 (7.1)	2 (7.1)
Related to ANX005	2 (7.1)	2 (7.1)
Infections	0 (0)	0 (0)
TEAE with Fatal Outcome	0 (0)	0 (0)

No grade 4 TEAEs reported

- No change in safety results from interim analysis
- IRR primarily first dose effect – none after 2nd dose
- No deaths and no serious infections observed
- Two treatment discontinuations unrelated to drug (Covid-19, consent withdrawn)
- Three treatment discontinuations potentially related to drug: all improved/resolved after drug cessation
  - One event each: idiopathic pneumonitis (SAE), systemic lupus erythematosus (SAE), asymptomatic hemolytic anemia (AE)
- All cases of treatment discontinuation had elevated ANA titers at baseline; no patients with normal ANA titers developed SAE
- Enhanced screening of ANA autoantibody levels at baseline and additional monitoring incorporated into ongoing/future trial to reduce risk in chronic ANX005 dosing protocols

Promising ANX005 Phase 2 Results Support Continued Advancement in Huntington's Disease

Phase 2 HD Trial Learnings and Next Steps

- ✓ **Full C1q target engagement in blood and CSF into follow-up period**
- ✓ **Stabilized disease progression in full cohort over 9-month study**
- ✓ **Rapid clinical improvement maintained in patients with higher baseline complement activity over 9-month study**
- ✓ **Generally well-tolerated with favorable benefit-risk demonstrated in HD;** enhanced safety management approach implemented
- ✓ **First evaluation of widely-researched MOA in the clinic; increasing appreciation for preserving functioning synapses for brain health**
- ❑ Planning engagements with US and EU regulators
- ❑ Assessing opportunity to conduct well-controlled study in HD, leveraging precision medicine approach

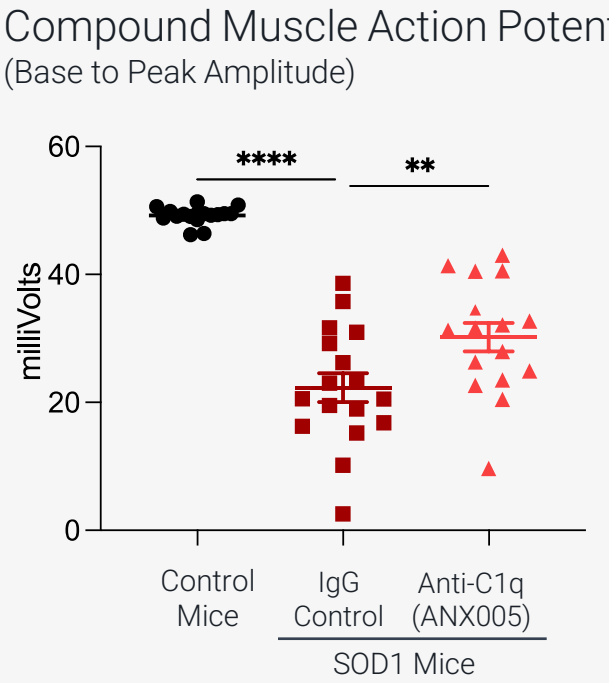
ANX005 for ALS
Inhibiting Upstream to Stop Neuroinflammation & Synaptic Loss

Data from ongoing Phase 2a trial anticipated in 2023

Amyotrophic Lateral Sclerosis Overview[1]

- **A fatal neurodegenerative disorder,** characterized by loss of upper (central) and lower (peripheral) motor neurons
- **C1q binds to synapses,** and amplifies complement-mediated synaptic loss and disability
- ANX005 demonstrated **target engagement in central and peripheral nervous systems** (HD & GBS)

Anti-C1q Protected Muscle Function in SOD1 ALS Mouse Model



[1]Affects ~19,000 US patients/year with no curative treatment

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ANX005 Phase 2a ALS Trial Ongoing; Data Anticipated in 2023

Trial Design

Specifications

- Open label trial (N= ~24)
- Target patients: Targeting all forms of ALS, onset of weakness within 3 years prior to enrollment, ALSFRS-R ≥ 30
- Objective endpoints: Safety, PK, C1q target engagement, and NfL concentrations in serum, Clinical outcomes (ALSFRS)
- Phase 2a data expected 2023

ANX007

for Ophthalmologic Diseases

- Geographic Atrophy (GA)

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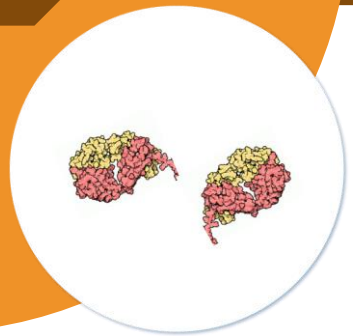
ANX007 Designed to Fully Inhibit Complement Activation in Neurodegenerative Diseases of the Eye

ANX007



ANX007 Therapeutic Approach

- ✓ Fab formulated for **intravitreal administration**
- ✓ **Localized inhibition of C1q** and the classical cascade in neurodegenerative diseases of the eye
- ✓ **Administered to >200 patients to date**
- ✓ Well-tolerated in Phase 1b, with **full target engagement** in the eye for at least four weeks
- ✓ Preclinical data demonstrating **protection of photoreceptor cells and retinal function**

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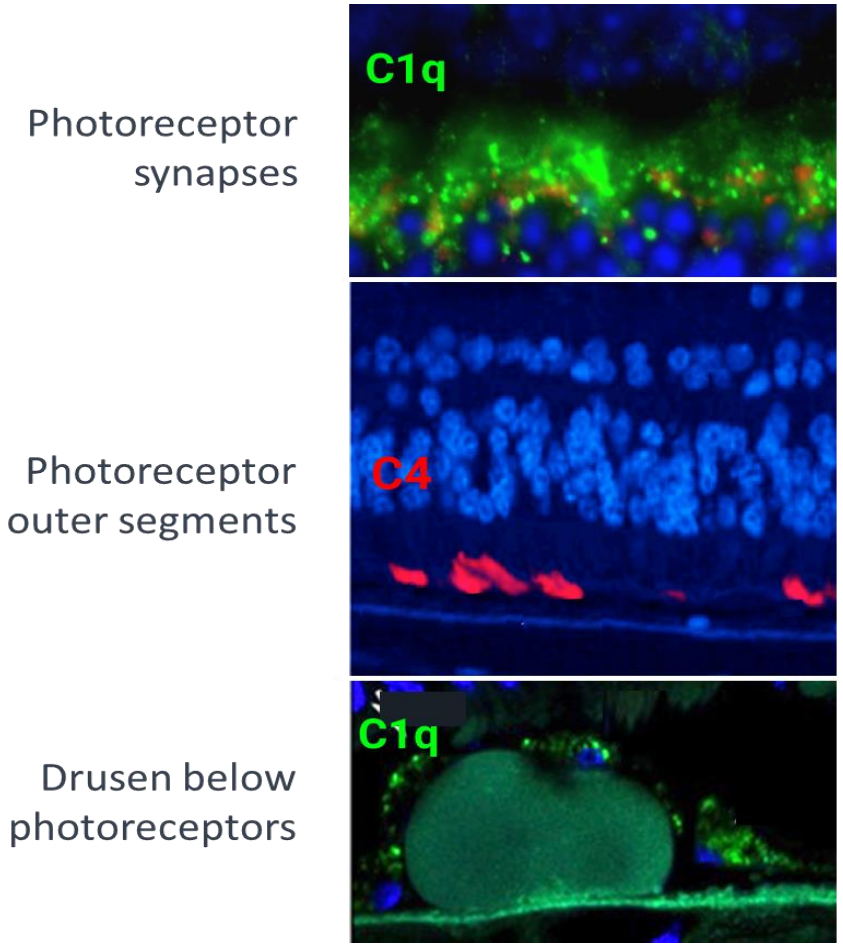
ANX007 for GA
C1q Inhibition Blocks All Downstream Tissue Damage

Data from ongoing Phase 2 trial anticipated in 1H 2023

ANX007 Therapeutic Approach

- GA is a leading cause of blindness[1]
- **Potential for enhanced efficacy by blocking upstream C1q and C4, *and* downstream C4, C3 and C5** activities that drive local immune response and destruction in retina
- **Blocking C1q retains homeostatic function of C3 and C5**



[1]Affects ~1M US patients, ~5M globally

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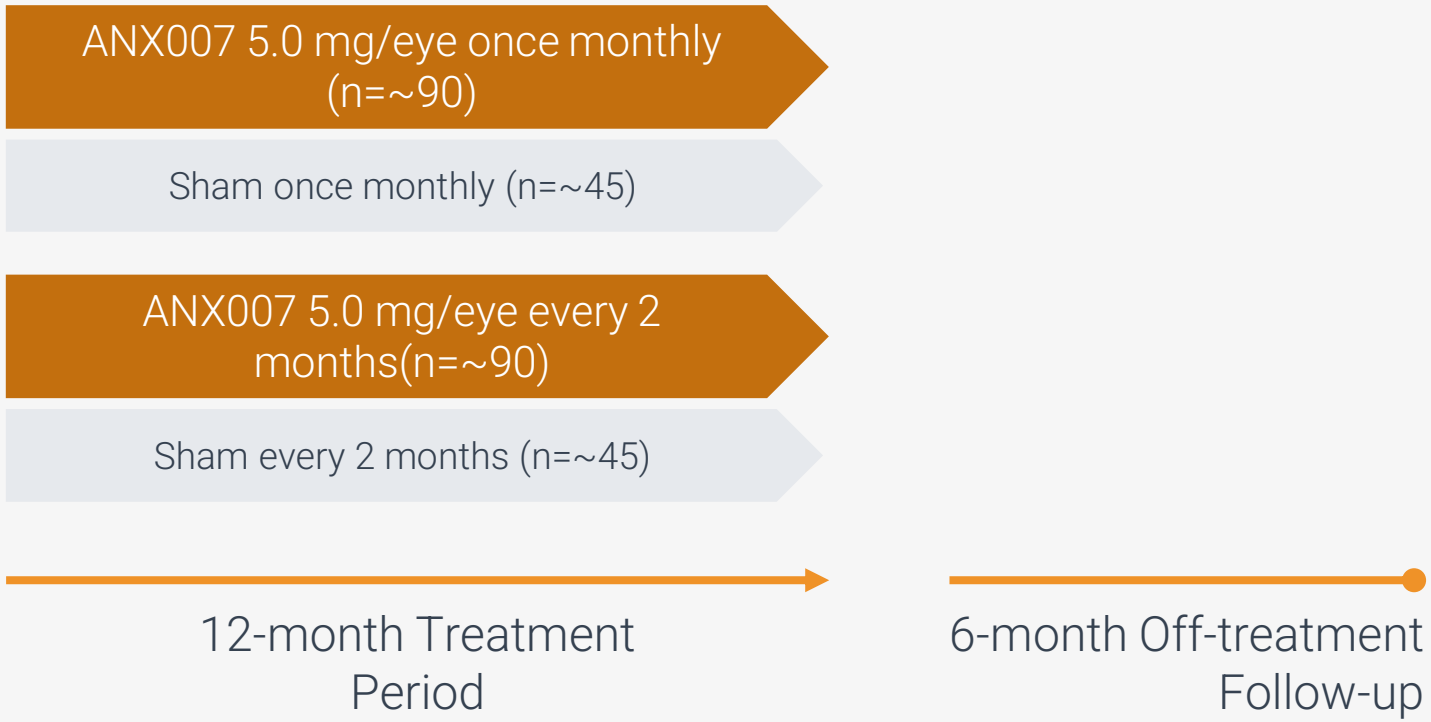
ANX007 Phase 2 Trial in GA Ongoing; Data Anticipated in 1H 2023

Trial Design



Specifications

- Randomized, double-masked trial (N=~270)
- **Primary endpoint: Change in area of geographic atrophy** on fundus autofluorescence
- Leveraging experience from related complement trials - **patients stratified for lesion location and lesion size**
- Topline Phase 2 data anticipated in 1H 2023

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ANX009

Subcutaneous for Autoimmune Diseases

- Lupus Nephritis (LN) and additional disorders

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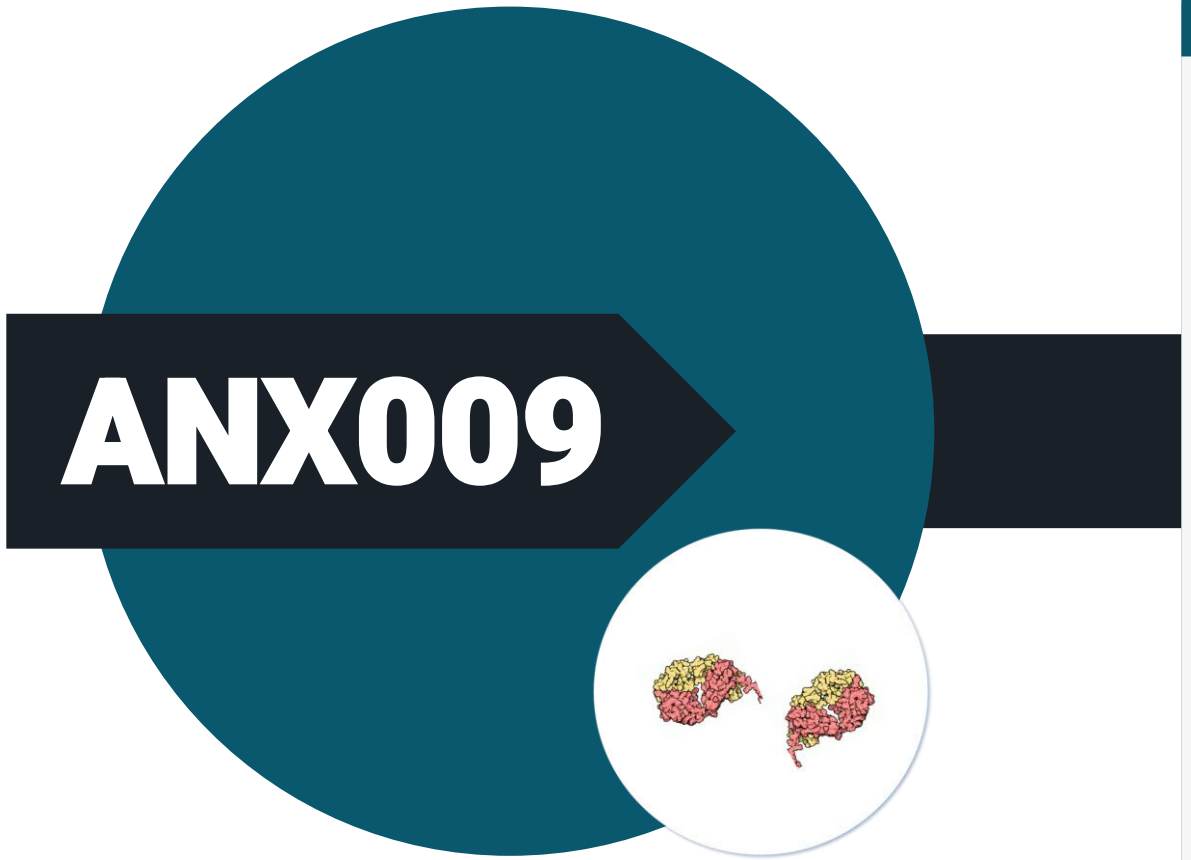
ANX009 Selectively Inhibits Complement Activation Only in Vascular Space



ANX009 Therapeutic Approach

- ✓ **Subcutaneous formulation** of an antigen-binding fragment (Fab)
- ✓ **Selectively inhibits C1q *in the vascular space***
- ✓ **Well-tolerated in Phase 1 trial, twice weekly dosing provided sustained C1q inhibition in the circulation**
- ✓ Designed to **enable chronic dosing** for use in future trials of autoimmune indications

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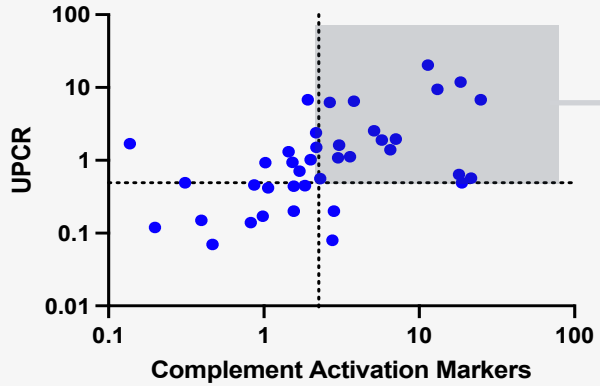
ANX009 for Lupus Nephritis (LN)
Selectively Inhibiting C1q to Stop Complement-Mediated Disease

Lupus Nephritis (LN) Overview[1]

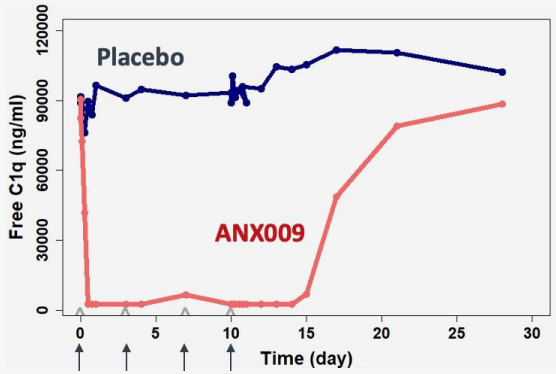
- **Autoantibodies against C1q enhance its activity and uniquely amplify kidney inflammation and damage**
- Precision medicine: **Targeting patients with high baseline complement activity** (top graph)
  - Approximately 1/3 of the population
- In a Phase 1 study, **twice weekly subcutaneous dosing of ANX009 provided full serum inhibition** (bottom graph)

High baseline complement activity correlated with disease activity



Patients most likely to respond to ANX009

ANX009 Inhibits Serum C1q in Ph 1 Study



Healthy volunteers; Dosing on days 0, 3, 7 and 10

[1]Affects ~60,000 US patients/year

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ANX009 Phase 1b Trial in Lupus Nephritis Ongoing; Initial Data Anticipated in 2H 2022

Trial Design

Specifications

- Target patients: Classical complement activity, smoldering disease, proteinuria, stable background therapy
- Objective endpoints: Safety and tolerability, complement PD markers, exploratory markers of renal tissue damage and function
- Phase 1b initial data anticipated 2H 2022

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LOOKING AHEAD

2022 Priorities

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Robust Preclinical Pipeline with Two Named Candidates Advancing Toward Clinical Development in 2022

ANX105 (IV)

Whole antibody: Modified for enhanced systemic dosing

- Intravenous administration
- Chronic autoimmune and neurological disorders

Autoimmune/Neuro

ANX1502 (oral)

Small molecule: Novel oral inhibitor of classical cascade

- Oral administration
- Peripheral autoimmune indications

Autoimmune

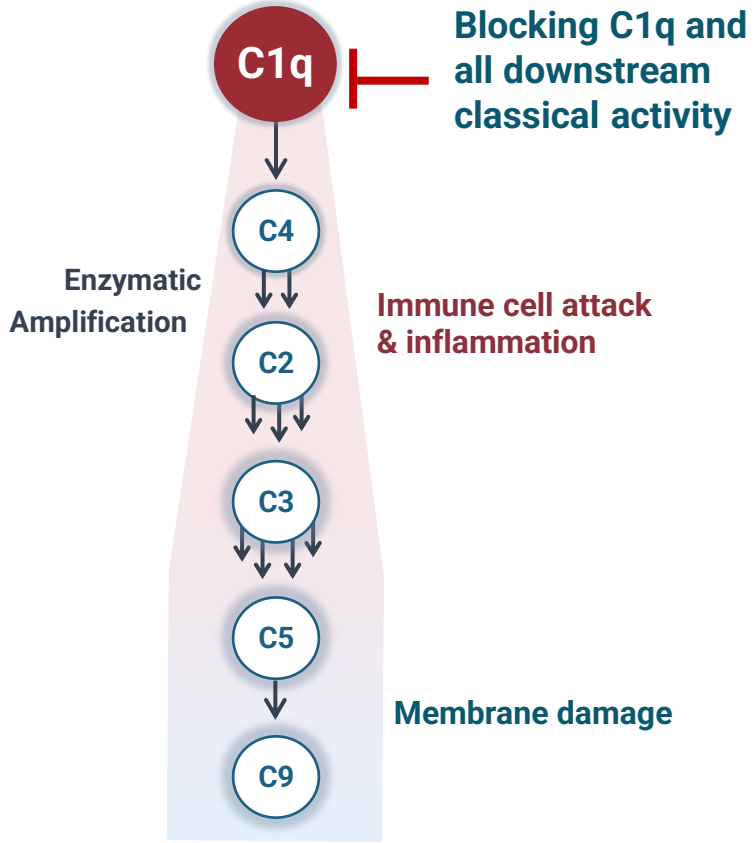
Platform

Wide range of additional opportunities in early pipeline

across mechanistically-related diseases

Autoimmune Neuro Ophthalmology

Classical Complement Pathway



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Five Key Clinical Priorities Driving Activities Through 2022

- Engage with US and EU regulators to discuss well-controlled study of ANX005 in HD, leveraging precision medicine approach
- Expect to report Ph2 data from ANX005 trial in wAIHA in 2H22
- Expect to report initial Ph1b data from ANX009 trial in LN in 2H22
- Prepare for multiple 2023 clinical readouts in GA, GBS and ALS
- Initiate clinical development of early-stage assets, ANX105 and ANX1502