# Fabry disease (mRNA-3630)

Last program update: February 26, 2020

<table>
<thead>
<tr>
<th>Modality</th>
<th>ID #</th>
<th>Program Indication</th>
<th>Preclinical development</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3 and commercial</th>
<th>Moderna rights</th>
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<td>mRNA-1944</td>
<td></td>
<td>Antibody against Chikungunya virus</td>
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<td>AZD7970</td>
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<td>Relaxin Heart failure</td>
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<td>50-50 U.S. profit sharing; AZ to pay royalties on ex-U.S. sales</td>
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<td>mRNA-3630</td>
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<td>α-GAL Fabry disease</td>
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<td>mRNA-6231</td>
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<td>IL-2 Autoimmune disorders</td>
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*mRNA-3630 is in IND-enabling GLP toxicology studies*
Fabry disease (Fabry) overview

Progressive, multi-organ disease with unmet need

• Fabry disease is an X-linked hereditary defect in glycosphingolipid metabolism caused by mutations in the GLA gene, which encodes for the lysosomal protein alpha galactosidase

• Disease burden: Progressive, multiorgan, lysosomal storage disorder (LSD) resulting in cellular and tissue dysfunction
  —Impact: vasculature, kidney, heart, GI, and neurological system
  —Childhood diagnosis; adult diagnosis post stroke or renal complications

• Target population: Annual incidence of ~1:80,000

• Unmet need despite approved treatments:
  —Recombinant ERT: Agalsidase beta (Sanofi/Genzyme); Agalsidase alpha (Shire/PLC)
  —Chaperone therapy: Migalastat (Amicus Therapeutics)

Moderna concept: IV-administered mRNA encoding α-GAL enzyme with native post-translational modifications to restore lysosomal enzyme activity for an extended duration
Fabry disease (mRNA-3630)
Preclinical data – sustained effect in multiple tissue types

Study Design:
- Species: Mouse
- Animals: GLA-/-
- Dose: 0.5 mpk
- Dosing Schedule: Single
- Injection Route: IV
- Sample Size: 3

With a single dose of our mRNA encoding for α-GAL we observed a sustained reduction in Lyso-Gb3 in pre-clinical studies.
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