Mouse Model for Inflammatory Bowel Diseases

**DESCRIPTION**
This is a transgenic double-knockout (DKO) mouse (Gpx1 and Gpx2). Both GPX1 and GPX2 belong to a selenium-dependent glutathione peroxidase (GPXs) family, which are enzymes that are efficient in the reduction of hydroperoxides. Gpx1 is expressed ubiquitously, and Gpx2 is highly expressed in the epithelium of the gastrointestinal (GI) tract. Gpx1/2-DKO mice have early-onset spontaneous ileocolitis beginning around weaning. These mice are an excellent model for human inflammatory bowel diseases (IBD) because they share similar disease etiology, which includes that the disease severity is highly influenced by genetic background, gut microflora, diet, and higher cancer risk in the distal GI tract. Unlike most mouse IBD models which have disrupted genes playing an important role in regulation of adaptive immunity, these Gpx1/2-DKO mice have intact immunity— which resembles human IBD.

**KEY ASPECTS**
- Homozygous disruption of both the endogenous Gpx1 and Gpx2 genes.
- Disease characteristics include ileitis, colitis, hypothermia, growth retardation, runting, perianal ulceration, diarrhea, wasting syndrome, IBD histopathology, tumors/cancer in the ileum or colon

**PUBLISHED DATA**
- Esworthy RS, Kim BW, Larson GP, Yip MLR, Smith DD, Li M, Chu F-F. A colitis locus on chromosome 2 impacting the severity of early-onset disease in mice deficient in GPx1 and GPx2. *Inflammatory Bowel Diseases*. In press, 2010

**INTELLECTUAL PROPERTY**

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<th>Title</th>
<th>US Patent Number</th>
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<td>Mice with Combined Disruption of GPX1 and GPX2 Gene have Growth Retardation, Hypothermia, and Colitis and Provide a Mouse Model for Cancer</td>
<td>6,762,343</td>
<td>7/14/2004</td>
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**CONTACT**
Matthew Grunseth, M.B.S.
Manager, Office of Technology Licensing
Telephone: (626) 471-7221 | Email: mgrunseth@coh.org

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