

The Gtp002 data product

Original number of samples	2,075
Number of samples (per 23.09.2026)	2,074
Number of unique participants	2,011
Biological sample type	DNA (whole blood)
Participant type(s)	MoBa mothers, MoBa children
Collection timepoint	17-18 weeks' gestation; birth
Selection criteria	Attention deficit hyperactivity disorder (ADHD)
Biomarker type(s)	Polymorphisms in genes related to xenobiotic metabolism
Original reference article	Engel et al. 2018
Analytical method(s)	Genotyping (Sequenom MassARRAY IPLEX®)
Related MoBaBIO product(s)	Mab015, Mab016, Mab017, Pro006
FHI Project number(s)	PDB1040

The project that generated these data

Prenatal Phthalate, Phenol and organophosphate (OPs) exposure and symptoms of ADHD in Norway

Project lead: Heidi Aase

The main purpose of this project was to investigate the link between exposures to pesticides and endocrine disruptors in pregnancy, and the development of ADHD during childhood. The specific aims of this project were to study organophosphate and phthalate exposure during pregnancy and the effect this has on risk for developing ADHD in childhood, to investigate thyroid hormone exposure during pregnancy and the effect this has on the risk for developing ADHD in childhood, as well as the effect of phthalate exposure during pregnancy and how this affects maternal and neonatal thyroid biomarkers. Genetic modification of the association between ADHD in childhood and organophosphate exposure was a further investigative aim, using genotype data on important loci involved in xenobiotic metabolism and specific mutations that are known to be linked.

Study population

The Gtp002 genotype data source is based on DNA samples from **1140 mothers** and **871 children**, and the study population comprises a case-control study design. 1098 of the maternal samples were samples collected from mothers at around 17 weeks' gestation, while 42 samples were collected from mothers after birth. Cases in this context were defined as mothers of children with attention deficit hyperactivity disorder (ADHD). Mothers were eligible for inclusion if they had been pregnant in 2003 or after, had completed the questionnaire administered when the child was 36 months of age, the child did not have a diagnosis of Down's syndrome or cerebral palsy, urine and blood samples from mid-pregnancy were available, and they lived in a geographic area that made them eligible for inclusion in the ADHD substudy. Children born in 2003 or after were identified as cases based on a clinical diagnosis of ADHD from the Norwegian Patient Registry (NPR). Cases were included based on a minimum of two registrations of "Hyperkinetic disorder" (ICD-10 codes F90, F90.0, F90.1, F90.8 or F90.9). Two registrations were a prerequisite for inclusion in order to avoid false diagnoses or registrations. Controls were randomly selected from the population of eligible mothers that had been defined for this study.

For more information on sample selection, refer to the original article describing the sampling and data collection by [Engel et al. 2018](#).

Available biomarker measures (variable names in bold)

Target loci

- Arginine-glutamic acid dipeptide repeats (RERE)

- Ataxin-2 (ATXN2)
- Broad complex-tramtrack-bric a brac and Cap'n'collar homology 2 (BACH2)
- Cytochrome P450 1A1 (CYP1A1)
- Cytochrome P450 1A2 (CYP1A2)
- Cytochrome P450 2A6 (CYP2A6)
- Cytochrome P450 3A4 (CYP3A4)
- Glutathione S-transferase mu 1 (GSTM1)
- Glutathione S-transferase pi 1 (GSTP1)
- Iodothyronine deiodinase 1 (DIO1)
- Iodothyronine deiodinase 2 (DIO2)
- N-acetyltransferase 1 (NAT1)
- N-acetyltransferase 2 (NAT2)
- Organic anion polypeptide C1 (OATP1C1)
- Paraoxonase 1 (PON1)
- Solute carrier family 16 member 10 (SLC16A10)
- Thyroid peroxidase (TPO)
- Thyroid-stimulating hormone receptor (TSHR)

Target Single Nucleotide Polymorphisms (variables in dataset)

- **rs662** (PON1)
- **rs1695** (GSTP1)
- **rs14399** (SLC16A10)
- **rs225011** (DIO2)
- **rs225012** (DIO2)
- **rs225014** (DIO2)
- **rs301799** (RERE)
- **rs366631** (GSTM1)
- **rs653178** (ATXN2)
- **rs854560** (PON1)
- **rs1801280** (NAT2)
- **rs2069521** (CYP1A2)
- **rs2069526** (CYP1A2)
- **rs2235544** (DIO1)
- **rs2470890** (CYP1A2)
- **rs4646421** (CYP1A1)
- **rs4986988** (NAT1)
- **rs10770704** (OATP1C1)
- **rs10944479** (BACH2)
- **rs11675434** (TPO)
- **rs12101255** (TSHR)
- **rs12720461** (CYP1A2)
- **rs28399433** (CYP2A6)
- **rs35599367** (CYP3A4)

Biological sampling and processing

Cord blood samples were drawn at the time of delivery or, in cases where cord blood was unavailable, a capillary sample was taken from the newborn child simultaneous to phenylketonuria (PKU) screening (approximately 3–4 days after birth). Cord blood samples were collected in two 7-ml ethylenediaminetetraacetic acid (EDTA) tubes. These were shipped from the collecting hospital overnight to MoBa's biobank at the Norwegian Institute of Public Health (NIPH). The samples usually arrived at the biobank within 1–2 days of blood donation, where whole blood was aliquoted into two polypropylene deep-well plates (930 µl in each, ABgene, Surrey, UK).

Whole blood samples were collected from mothers and fathers at 17–18 weeks of gestation, or after birth. Samples were collected in two 7-ml ethylenediaminetetraacetic acid (EDTA) tubes. These were shipped from the collecting hospital overnight to MoBa's biobank at the Norwegian Institute of Public Health (NIPH). The samples usually arrived at the biobank within 1–2 days of blood donation, where whole blood was aliquoted into two polypropylene deep-well plates (930 µl in each, ABgene, Surrey, UK).

DNA extraction was performed manually using a FlexiGene DNA extraction kit (Qiagen, Hilden, Germany), and DNA content was quality controlled using a spectrophotometer (Spectramax 190, Molecular Devices, Sunnyvale, CA). DNA is stored at –20 °C at NIPH's biobank.

For more information on biological sampling, processing and storage, please refer to the original reference articles for NIPH's biobank by [Rønningen et al. 2006](#) and [Paltiel et al. 2014](#)

Analytical methodology

Genotyping of the single nucleotide polymorphisms included in this study was conducted using the **Sequenom MassARRAY IPLEX® platform** (iPlex, Adelaide, SA, Australia). For more information, refer to the original methodology article by [Gabriel et al. 2009](#).

Published articles using Gtp002

This section also includes articles related to study design, sampling, and data collection.

- ❖ Manley CK, Villanger GD, Thomsen C, Cequier E, Sakhi AK, Reichborn-Kjennerud T, Herring AH, Øvergaard KR, Zeiner P, Roell KR, Engel LS, Kamai EM, Thistle J, Hall A, Aase H, Engel SM. Prenatal Exposure to Organophosphorus Pesticides and Preschool ADHD in the Norwegian Mother, Father and Child Cohort Study. *Int J Environ Res Public Health*. 2022 Jul 2;19(13):8148.
- ❖ Choi G, Keil AP, Richardson DB, Daniels JL, Hoffman K, Villanger GD, Sakhi AK, Thomsen C, Reichborn-Kjennerud T, Aase H, Engel SM. Pregnancy exposure to

organophosphate esters and the risk of attention-deficit hyperactivity disorder in the Norwegian mother, father and child cohort study. *Environ Int.* 2021 Sep;154:106549.

- ❖ Engel SM, Villanger GD, Nethery RC, Thomsen C, Sakhi AK, Drover SSM, Hoppin JA, Zeiner P, Knudsen GP, Reichborn-Kjennerud T, Herring AH, Aase H. Prenatal Phthalates, Maternal Thyroid Function, and Risk of Attention-Deficit Hyperactivity Disorder in the Norwegian Mother and Child Cohort. *Environ Health Perspect.* 2018 May 10;126(5):057004.

Restrictions for use

None currently known.

Acknowledgements recommended for use

We recommend that any use of these data in analyses that are presented in peer-review publications acknowledges the original article describing sampling and data collection:

Engel SM, Villanger GD, Nethery RC, Thomsen C, Sakhi AK, Drover SSM, Hoppin JA, Zeiner P, Knudsen GP, Reichborn-Kjennerud T, Herring AH, Aase H. Prenatal Phthalates, Maternal Thyroid Function, and Risk of Attention-Deficit Hyperactivity Disorder in the Norwegian Mother and Child Cohort. Environ Health Perspect. 2018 May 10;126(5):057004.

Disclaimer

The data in Gtp002 that are available for use are provided on an *as is* basis, and FHI does not provide any guarantees related to data quality and assurance of the original dataset. MoBa participants can retract their consent to participate in the MoBa study at any time, and results generated using biological samples will be removed from MoBa datasets accordingly. The descriptive information regarding sample sizes and data content in this product sheet is correct and up-to-date as of **23.09.2026**.