



Data Article

Proteomics dataset of liver tissue from spinal muscular atrophy, heterozygous, and wild-type mice, enabling pathway identification

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ABSTRACT

We present a label-free quantitative proteomics dataset from liver tissue of wild-type (WT), heterozygous (HET), and spinal muscular atrophy (SMA) mice at postnatal day 10 (P10). Proteins were extracted using urea lysis, digested with trypsin, and analyzed by LC-MS/MS on an Orbitrap Exploris 480 mass spectrometer. DIA-NN and Perseus software were used for data processing and statistical analysis, including principal component analysis (PCA) and differential expression analysis for comparisons between WT and SMA, and HET and SMA. One mis-genotyped SMA sample was identified and excluded based on Western blot validation of the survival motor neuron (SMN) protein levels. The dataset provides complete protein identification and quantification tables, lists of significantly altered proteins, and Western blot validation for ferrochelatase (FECH) and survival motor neuron (SMN) protein in RIPA-extracted proteins from whole liver organ homogenates. All raw and processed data, including meta-data and statistical outputs, have been deposited to the ProteomeXchange Consortium with the identifier PXD070887. The dataset can be reused for comparative proteomic anal-

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yses, cross-study integration, and meta-analyses of murine liver proteome alterations in neuromuscular and metabolic disease models.

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Specifications Table

Subject	Biology
Specific subject area	Proteomics in spinal muscular atrophy murine liver.
Type of data	Figures, tables, raw and processed proteomics data
Data collection	Liver tissues from WT, HET, and SMA mice of postnatal day 10 (P10) were analyzed by label-free quantitative proteomics. Proteins were extracted with urea buffer, digested with trypsin, and peptides separated on a Vanquish neo nano HPLC system coupled to a Thermo Orbitrap Exploris 480 with FAIMSpro interface. Data was processed using DIA-NN (v1.8) and analyzed in Perseus (v1.6.15). One mis-genotyped sample was identified through principal component analysis (PCA) and Western blot and excluded from downstream analysis. Validation Western blots were performed on independent RIPA-extracted tissues.
Data source location	CECAD Research Center and University of Cologne, Cologne, Germany
Data accessibility	Repository name: PRIDE (ProteomeXchange) Data identification number: PXD070887Direct URL to data: https://www.ebi.ac.uk/pride/archive/projects/PXD070887
Related research article	S. Vrettou, S. Müller, B. Wirth, SMN deficiency disrupts hepatic mitochondrial iron homeostasis and NRF2-dependent redox control in spinal muscular atrophy, bioRxiv (2026), doi: https://doi.org/10.64898/2026.01.08.698518 [1].

1. Value of the Data

- This dataset offers a detailed proteomic profile of liver tissue from WT, HET, and SMA mice at P10, enabling direct comparison of molecular changes linked to disease and carrier status (which are frequently overlooked, as HET animals are widely used as controls).
- We provide both the raw dataset (with one mis-genotyped SMA sample retained for transparency) and the processed dataset used for statistical comparisons, which enable reproducibility.
- The provision of protein-level annotations and validation by Western blot allows for pathway enrichment, biomarker discovery, and comparative studies in liver diseases.

2. Background

Spinal muscular atrophy (SMA) is an autosomal recessive neuromuscular disorder that affects alpha lower motor neurons in the spinal cord and is caused by mutation or biallelic deletion of the *SMN1* gene, leading to decreased levels of the survival motor neuron (SMN) protein [2]. While initially described as a neurodegenerative disease and studied mainly for its effects on motor neurons, SMA has recently been re-identified as a multi-organ disorder due to the ubiquitous expression of SMN protein [3]. The liver, which harbors critical roles in metabolism [4], constitutes a key organ in disease progression [5], but is understudied in SMA research. This dataset provides a comprehensive proteomic profile of liver tissue from P10 SMA, heterozygous, and wild-type mice. These data compilations allow deeper exploration of liver molecular pathways and enable cross-comparisons with other tissues in murine SMA datasets or other disease models. These data complement and support the findings of our associated research article [1].

3. Data Description

This dataset reports label-free data-independent acquisition (DIA) proteomics of liver tissue from P10 Taiwanese SMA mice ($\text{Smn}^{-/-}$; $\text{SMN2}^{\text{tg}/0}$) [6], heterozygous carriers (HET, $\text{Smn}^{+/-}$; $\text{SMN2}^{\text{tg}/0}$), and wild-type controls (WT, $\text{Smn}^{+/+}$; $\text{SMN2}^{0/0}$). SMA and HET animals were obtained as littermates from a mixed FVB/N $\times$ C57BL/6 colony, whereas WT controls were age-matched C57BL/6 mice.

Whole-liver lysates were digested in-solution and analyzed by DIA LC-MS/MS on a Thermo Orbitrap Exploris 480 mass spectrometer equipped with a FAIMS Pro interface and coupled to a Vanquish Neo nano-HPLC system. Peptide identification and quantification were performed using DIA-NN v1.8.1 with a predicted spectral library generated from the *Mus musculus* UniProt canonical FASTA database (UP0000000589; downloaded 2024-01-04). DIA-NN outputs were filtered at 1 % precursor- and protein-level FDR, and additional quality filters were applied (≥ 4 fragment ions per precursor, library q-value ≤ 0.01 , unique peptides only). Downstream statistical analysis, including normalization, PCA and differential abundance testing, was performed in Perseus v1.6.15.

All RAW data files, together with DIA-NN search outputs, spectral libraries and FASTA files, are made publicly available through the PRIDE [7] repository (accession number PXD070887) associated with this manuscript. One SMA biological replicate (raw file E3_ColID_656_3632_U_165) was identified as an outlier based on PCA (Fig. 1) and confirmed as mis-genotyped via Western blot analysis (Fig. 2A) of the same urea-extracted liver lysates used for proteomics. Although excluded from differential expression analysis, its RAW file and corresponding search results remain accessible in PRIDE for transparency. Table 1 provides the correspondence between RAW file names and sample group assignments.

For full transparency, all protein identifications and quantifications –including those from the mis-genotyped sample– are provided in Supplementary Table 1.

Here, we additionally provide the PCA plots before (Fig. 1) and after removal of the mis-genotyped SMA sample (Fig. 2B), alongside the corresponding Western blot confirming its inter-

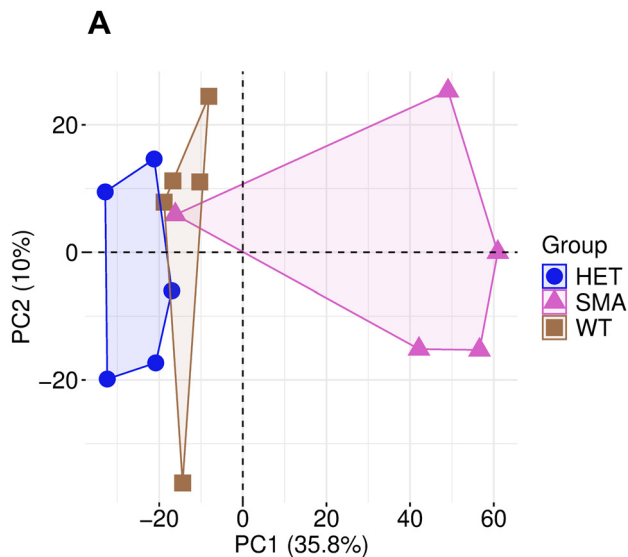


Fig. 1. Principal component analysis (PCA) of liver proteome data. PCA of WT, HET, and SMA postnatal day 10 (P10) liver samples, with each group represented by differently colored markers. One SMA sample (SMA-5) clustered separately from the remaining biological replicates, indicating inconsistency with its assigned genotype.

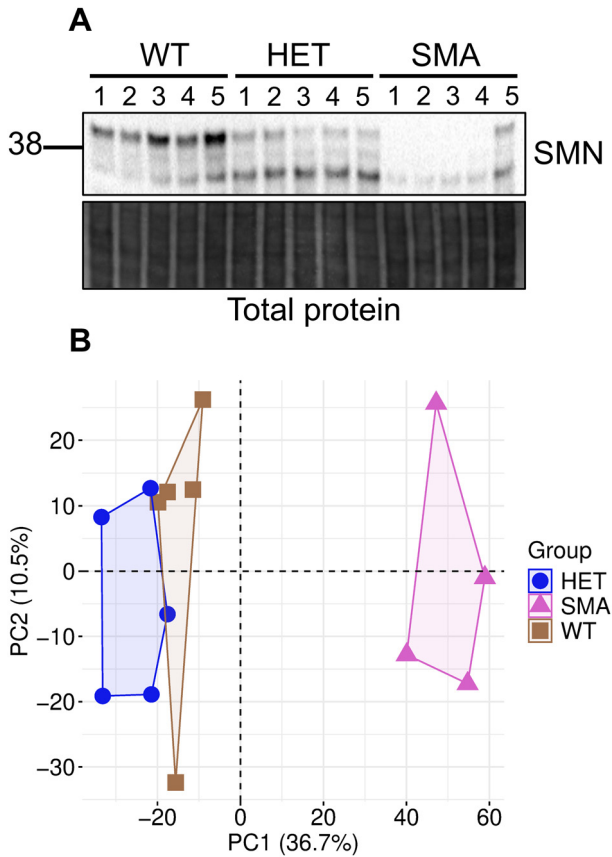


Fig. 2. Outlier validation and reassignment of biological groups. **(A)** Western blot analysis of the urea-extracted liver samples used for LC-MS/MS showing SMN protein levels across WT, HET, and SMA samples. Total protein reverse staining served as a loading control. The SMA sample (SMA-5) originally annotated as SMA displayed an SMN band pattern consistent with a HET genotype. **(B)** Principal component analysis (PCA) of the dataset following reassignment and removal of the mis-genotyped sample. WT, HET, and SMA groups are indicated by differently colored markers.

mediate SMN expression level relative to other SMA and WT or HET samples (Fig. 2A). These files allow readers to reproduce the rationale for sample exclusion.

To support biological interpretation of the dataset, we include volcano plots for the WT vs. SMA (Fig. 3A) and HET vs. SMA (Fig. 3B) comparisons, together with the corresponding Excel tables of significantly altered proteins derived from the Perseus analysis (Supplementary Tables 2, 3). Among the consistently altered proteins, ferrochelatase (FECH) was identified as upregulated in SMA relative to both WT and HET groups (Fig. 3A, and B). To independently confirm this upregulated target in SMA, we performed Western blot analysis on RIPA-extracted liver lysates (Fig. 4A), which validated the increased FECH expression (Fig. 4B) and strengthened the reliability of the DIA-based quantification.

Together, these materials provide a structured overview of the data quality assessment, statistical outputs and validation supporting the proteomics analysis. The full proteomics dataset (RAW files and DIA-NN output files) is available through the PRIDE [7] repository under the dataset identifier PXD070887, while here we provide in addition all figures and tables required to reproduce the PCA plots, volcano plots, and Western blot validation.

Table 1

PRIDE raw file names and sample group assignment.

Group	Biological replicate #	Raw file name	Comments
WT	WT-1	E3_ColID_656_3632_LI_151	
WT	WT-2	E3_ColID_656_3632_LI_152	
WT	WT-3	E3_ColID_656_3632_LI_153	
WT	WT-4	E3_ColID_656_3632_LI_154	
WT	WT-5	E3_ColID_656_3632_LI_155	
HET	HET-1	E3_ColID_656_3632_LI_156	
HET	HET-2	E3_ColID_656_3632_LI_157	
HET	HET-3	E3_ColID_656_3632_LI_158	
HET	HET-4	E3_ColID_656_3632_LI_159	
HET	HET-5	E3_ColID_656_3632_LI_160	
SMA	SMA-1	E3_ColID_656_3632_LI_161	
SMA	SMA-2	E3_ColID_656_3632_LI_162	
SMA	SMA-3	E3_ColID_656_3632_LI_163	
SMA	SMA-4	E3_ColID_656_3632_LI_164	
SMA	SMA-5	E3_ColID_656_3632_LI_165	*Excluded

* Sample E3_ColID_656_3632_LI_165 was excluded from downstream analysis after PCA and Western blot confirmed mis-genotyping.

4. Experimental Design, Materials and Methods

4.1. Animal model and tissue collection

The severe Taiwanese mouse model exhibits an early-onset phenotype resulting from homozygous deletion of the murine *Smn* gene, combined with two copies of the human *SMN2* transgene [6]. The animal cohorts used for the acquisition of the proteomics dataset correspond to: SMA (*Smn*^{-/-}; *SMN2*^{tg/0}), heterozygous (HET, *Smn*^{+/-}; *SMN2*^{tg/0}), wild-type (WT, *Smn*^{+/+}; without the human *SMN2* transgene). At P10, mice were rapidly decapitated, and organs were dissected under aseptic conditions. Livers were immediately snap-frozen on dry ice and stored at -80 °C until proteomic and biochemical analyses. At sacrifice, tails were collected and genotyping was performed on the extracted tail DNA using these primers: *Smn*^{KO} forward: 5'-ATAACACCACCCTCTACTC-3'; *Smn*^{KO} reverse 1: 5'-AGCTGAAGAACGATCAGC-3'; *Smn*^{KO} reverse 2: 5'-TAGCCGTGATGCCATTGTCA-3' [8].

4.2. Protein extraction and sample preparation

Approximately 10 mg of liver tissue was used for protein extraction and digestion according to the CECAD core facility urea digest protocol for tissues, with minor adjustments. Briefly, frozen tissue was homogenized in 8 M urea in 50 mM triethylammonium bicarbonate (TEAB; pH 8.0), supplemented with a 1x protease inhibitor cocktail (Roche, complete, EDTA-free). Homogenization was performed using a Precellys homogenizer (Bertin technologies) at 2 × 20 sec, 6500 rpm, followed by incubation on ice for 10 min and then centrifugation at 18,000 × g for 20 min at 4 °C. Following centrifugation, the supernatants were transferred to 1.5 mL Eppendorf tubes and sonicated in a Bioruptor Plus (Diagenode; 30 cycles of 30 sec at 4 °C) for 7 min at 4 °C. Liver lysates were then centrifuged at 18,000 × g for 20 min at 4 °C to further remove debris, and supernatants were collected into new 1.5 mL Eppendorf tubes. The liver homogenates' protein concentration was measured with a NanoDropTM One Spectrophotometer & QubitTM 4 Fluorometer (ThermoFischer, catalog number: A38189). For each sample, 50 µg protein was reduced with 10 mM DTT (30 min, RT), alkylated with 40 mM chloroacetamide (30 min, RT), and digested with Lys-C (enzyme:substrate 1:100, 2–4 h, 25 °C). Samples were then diluted with 50

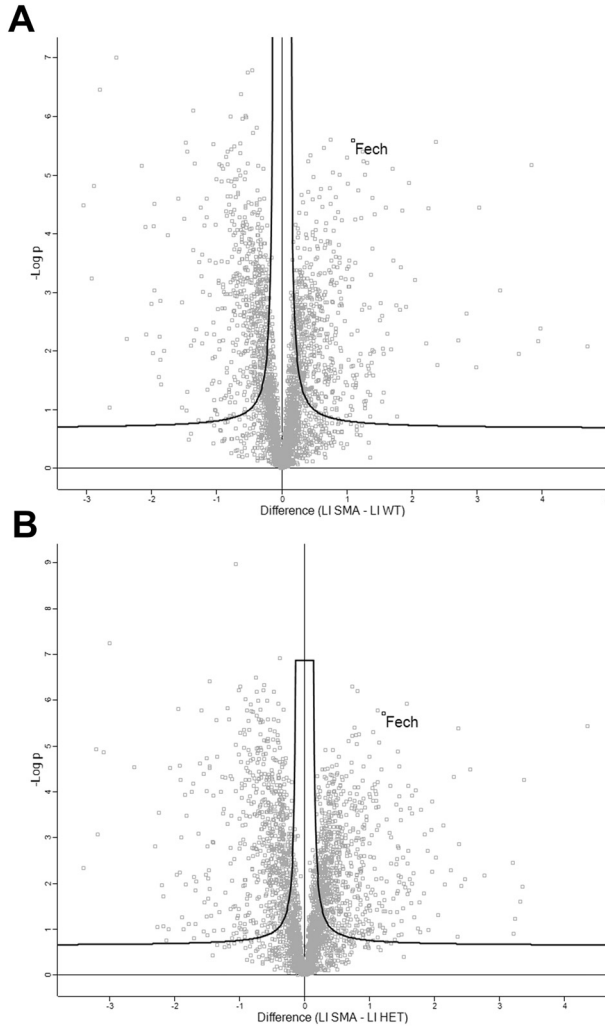


Fig. 3. Volcano plots of differentially abundant proteins. **(A)** Volcano plot showing proteins differentially abundant between WT and SMA liver samples. **(B)** Volcano plot showing proteins differentially abundant between HET and SMA liver samples. The ferrochelatase (FECH) protein highlighted in bold, at both comparisons, to illustrate its consistent differential abundance across comparisons.

mM TEAB to ≤ 2 M urea and subjected to overnight trypsin digestion (enzyme:substrate 1:50, 16 h, 25 °C). Digests were acidified to 1 % (v/v) formic acid and desalted using mixed-mode SDB-RPS StageTips according to the CECAD protocol (methanol and buffer A (0.1 % (v/v) formic acid in water) /buffer B (0.1 % (v/v) formic acid in 80 % acetonitrile) equilibration, loading ≤ 15 μ g digest, sequential washes in buffer A and B).

4.3. Data acquisition

Samples were analyzed by the CECAD Proteomics Facility (University of Cologne) on an Orbitrap Exploris 480 mass spectrometer that was equipped with a FAIMSpro differential ion mo-

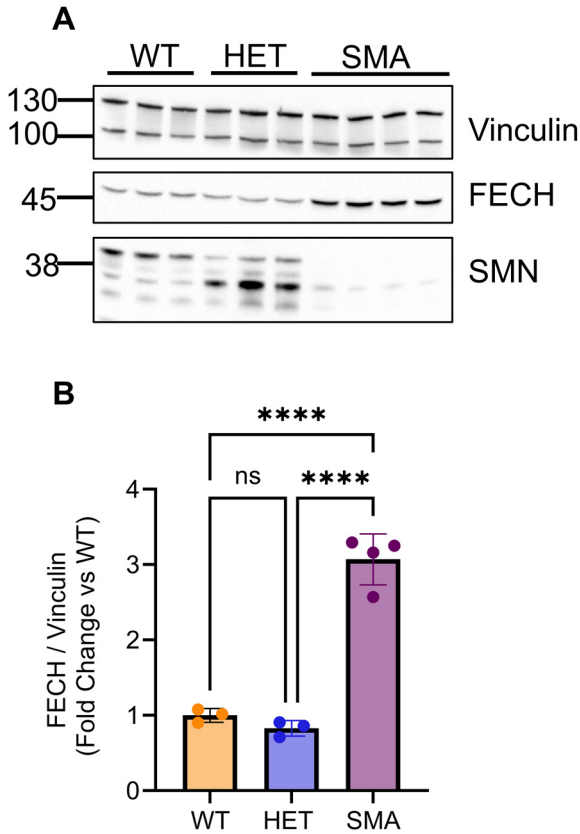


Fig. 4. Validation of proteomics-identified protein target with Western blot. **(A)** Western blot analysis of RIPA-extracted WT, HET, and SMA liver samples ($n = 3$ per group), using antibodies against ferrochelatase (FECH), survival motor neuron (SMN) protein and Vinculin (which served as loading control). **(B)** Quantification of FECH normalized to Vinculin, expressed as fold change relative to WT. Bars represent $\pm$ SD. Statistical analysis was performed using one-way ANOVA with Tukey's post hoc test (ns = not significant, **** $p < 0.0001$).

bility device (Thermo Scientific, granted by the German Research Foundation under E2 INST 1856/71-1 FUGG and INST 216/1163-1 FUGG). A Vanquish neo nano HPLC system (Thermo Scientific) was coupled to the mass spectrometer. The HPLC system was operated in trap and elute setup using a precolumn with back flush elution (Acclaim 5 μ m PepMap 300 μ m Cartridge) and an in-house packed analytical column (30 cm length, 75 μ m inner diameter, filled with 2.7 μ m Poroshell EC120 C18, Agilent). Peptides were chromatographically separated with an initial flow rate of 400 nL/min and the following gradient: initial 2 % B (0.1 % (v/v) formic acid in 80 % acetonitrile), up to 6 % in 4 min. Then, flow was reduced to 300 nL/min and B increased to 20 % in 50 min, up to 35 % B within 27 min and up to 95 % solvent B within 1.0 min while again increasing the flow to 400 nL/min, followed by column wash with 95 % solvent B and re-equilibration to initial condition. The FAIMS settings were -50V compensation voltage and electrode temperatures of 99.5 $^{\circ}$ C for the inner and 85 $^{\circ}$ C for the outer electrode. MS1 scans were acquired from 399 m/z to 1001 m/z at 15 k resolution. Maximum injection time was set to 22 ms and the AGC target to 100 %. MS2 scans ranged from 400 m/z to 1000 m/z and were acquired at 15 k resolution with a maximum injection time of 22 ms and an AGC target of 100 %. DIA scans covering the precursor range from 400 - 1000 m/z and were acquired in 60 $\times$ 10 m/z windows with an overlap of 1 m/z . All scans were stored as centroid.

4.4. Sample processing in DIA-NN

Samples were analyzed in DIA-NN 1.8.1 [9]. First a predicted spectral library was generated from a mouse canonical fasta file (UP000000589.fasta, downloaded from Uniprot on 2024-01-04). DIA-NN library prediction parameters were precursor mass range 400 m/z- 1000 m/z, precursor charge range 2-4, fragment mass range 250 m/z - 1500 m/z and N-terminal methionine excision enabled, maximum number of missed cleavages set to 1, min peptide length set to 7, max peptide length set to 30 and cysteine carbamidomethylation enabled as a fixed modification. Sample files were processed with the predicted library and the additional command line prompts “-report-lib-info” and “-relaxed-prot-inf”. The output was filtered at 0.01 FDR. Afterwards, DIA-NN output was further filtered on ≥ 4 fragment ions per precursor, library q-value, global q-value ≤ 0.01 and unique peptides using the dplyr package in R. Finally, LFQ values calculated using the DIA-NN R-package. Statistical analysis of results was performed in Perseus 1.6.15 [10]. The mass spectrometry data have been deposited to the ProteomeXchange Consortium via the PRIDE [7] partner repository with the dataset identifier PXD070887.

4.5. Data processing and analysis

Downstream analysis was performed in Perseus 1.6.15. The protein matrix was filtered to remove contaminants and entries identified only by site, log2-transformed, and normalized. Missing values were imputed from a normal distribution (width: 0.3, down-shift: 1.8). PCA and two-sample t-tests were used to identify genotype-specific proteomic changes. PCA coordinates were exported from Perseus and visualized using SRplot (Fig. 1, 2) [11], while volcano plots were directly exported from Perseus with significance threshold of FRD: 0.05 and S0: 0.1 (Fig. 3, Supplementary Tables 2, 3).

Functional enrichment of significantly altered proteins was assessed through 1D annotation enrichment using Gene Ontology (GO) and KEGG categories integrated in Perseus (Supplementary Tables 4, 5). Validation of proteomics targets by Western blot (Fig. 4) was performed using ImageJ for densitometric quantification, and statistical analysis was carried out by one-way ANOVA using GraphPad Prism 10.6.1.

4.6. Western blot analysis

For the validation of the SMA outlier that was identified by PCA (Fig. 1), we performed a Western blot on the same urea samples used for proteomics (5 WT, 5 HET, 5 SMA) (Fig. 2A). The blot was probed for SMN (mouse, Cat#: 610647, BD Biosciences Transduction Laboratories™, dilution 1:1000), with total protein staining (Thermo Scientific™ Pierce™ Reversible Protein Stain Kit for PVDF Membranes, Ca#: 24585) used as loading control.

Additionally, to validate the upregulation of ferrochelatase (FECH) detected in the proteomics comparisons of both WT versus SMA and HET versus SMA, we performed Western blot on different liver tissues homogenized in RIPA buffer from independent WT, HET, and SMA animals (Fig. 4A and B). The blot was incubated for SMN (same as above), FECH (rabbit, proteintech, Cat#: 14466-1-AP, dilution 1:1000), and vinculin (rabbit, Cat#:129002, Abcam, dilution 1:1000) served as the loading control.

Limitations

In this dataset, the liver tissues collected for proteomics analysis correspond to the late symptomatic stage of SMA (P10), and thus, the data compilation is exclusive to liver tissue and does

not reliably represent proteome alterations in other SMA organs or other stages of disease progression. Due to mis-genotyping, one SMA sample was excluded from the statistical analysis between the groups tested; nevertheless, it was retained in the raw dataset.

Ethics Statement

All mice were housed under a 12-hour light/dark cycle with access to food and water *ad libitum*. Breeding, housing, and experimental use of animals were performed in a specific pathogen-free environment. All animal experiments were approved by the Landesamt für Natur, Umwelt und Verbraucherschutz Nordrhein-Westfalen (LANUV) under the application numbers: 81-02.04. 2020.A196, 81-02.04. 2019.A017, 81-02.04. 2019.A138, §4.23.008, §4.22.002.

CRediT Author Statement

Sofia Vrettou: Conceptualization, Methodology, Software, Visualization, Validation, Writing, Original draft preparation. **Stefan Müller:** Methodology, Software, Data Curation, Resources, Writing, Funding Acquisition. **Brunhilde Wirth:** Conceptualization, Supervision, Writing-Reviewing and Editing, Funding Acquisition.

Data Availability

Label-free DIA proteomics of WT, HET, and SMA mouse liver at postnatal day 10 (P10) (Original data) (PRIDE).

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Declaration of Competing Interest

The authors declare no competing interests.

Supplementary Materials

Supplementary material associated with this article can be found, in the online version, at [doi:10.1016/j.dib.2026.112632](https://doi.org/10.1016/j.dib.2026.112632).

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