Data Article

Mitochondrial lipid profiling data of a traumatic optic neuropathy model

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Abstract

Traumatic optic neuropathy (TON) is a degenerative process that occurs in a subset of patients following blunt force trauma to the head. This condition is characterized by retinal ganglion cell (RGC) death and axon degeneration within the optic nerve [1]. At the cellular level, mitochondrial changes are associated with many optic neuropathies [2, 3]. Here, we provide a dataset demonstrating changes in the optic nerve mitochondrial lipid profile of a sonication-induced traumatic optic neuropathy (SI-TON) mouse model at 1, 7, and 14 days after injury. 32 C57BL/6J mice were separated into 4 groups (control, 1, 7, and 14 days) of 8, with 4 males and 4 females in each. Mice were exposed to sonication-induced trauma as described previously (by Tao et al) and optic nerves were harvested at 1, 7, or 14 days following injury [4]. Mitochondria were isolated from homogenized optic nerves and lipids were extracted. Extracted mitochondrial lipids were analysed with a Q-Exactive Orbitrap Liquid Chromatography-Mass Spectrometer (LC MS-MS). Further analysis of raw data was conducted with LipidSearch 4.1.3 and Metaboanalyst 4.0. This data is publicly available at the Metabolomics Workbench, http://www.metabolomicsworkbench.org (Project ID: PR000905).

Keywords:
Traumatic Optic Neuropathy
Neurodegeneration
Mitochondrial lipids
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Specifications Table

| Subject                  | Cell Biology                  |
|--------------------------|-------------------------------|
| Specific subject area    | Mitochondria, Lipids          |
| Type of data             | Table                         |
| How data were acquired   | Liquid Chromatography Q-Exactive Orbitrap Mass Spectrometry, LipidSearch 4.1.3, Metaboanalyst 4.0 |
| Data format              | Raw                           |
|                          | Analyzed                      |
|                          | Filtered                      |
| Parameters for data      | Optic nerve, age, survival    |
| collection               |                               |
| Description of data      | Sonication-induced trauma to optic nerves, optic nerves collected and mitochondria isolated. Mitochondrial lipids extracted with butanol-methanol method and analysed with mass spectrometry, further analysed with Metaboanalyst 4.0 and LipidSearch 4.1.3 |
| collection               |                               |
| Data source location     | Bascom Palmer Eye Institute, Miller School of Medicine at University of Miami, Miami, FL 33136, USA |
| Data accessibility       | Repository name: Metabolomics Workbench- Project ID PR000905 |
|                          | Data identification number: 10.21228/M8Q69W |
|                          | Direct URL to data: https://www.metabolomicsworkbench.org/data/DRCCMetadata.php?Mode=Project&ProjectID=PR000905 |
| Related research article | Tao, W., et al., A Novel Mouse Model of Traumatic Optic Neuropathy Using External Ultrasound Energy to Achieve Focal, Indirect Optic Nerve Injury. Sci Rep, 2017. 7(1): p. 11779. |

Value of the Data

- This data is useful for exploring metabolomic differences at the cellular level in the neurodegenerative disease process and as potential areas for further investigation in both diagnostic and therapeutic targets clinically.
- This data benefits investigators studying mitochondrial changes in the settings of optic neuropathies, neuroprotection, and neuro-regeneration.
- This data will serve as a starting point for investigation into mitochondrial lipidomic as well as multi-OMIC approaches in the study of traumatic optic neuropathy and other neurodegenerative diseases. Additional experiments can be done to further correlate mitochondrial dysfunction with traumatic optic neuropathy, an association that is not well established in the literature.
- This data can be used to examine changes at the cellular level in other optic neuropathies and general neurodegeneration.

1. Data Description

We present here the changes that occur in the mitochondrial lipidome from the optic nerve of two-month-old C57BL/6J mice after sonication-induced TON (SI-TON) at 1, 7, and 14 days post-injury (Figure 1); time points were selected based on previous data of RGC cell loss [4, 5]. 32 mice were randomly assigned into control (uninjured, n=8) and three experimental groups (n=8 each) with even distribution of males and females (Table 1). Following SI-TON, optic nerves were harvested from all mice at their respective time points. The control group nerves were collected on day 0 (at time of sonication exposure of the other groups). Tissue was homogenized and the Mitochondrial Isolation Kit for Tissue (PIERCE, Rockford, IL, USA) was used to isolate
Figure 1. Schematic diagram of mitochondrial isolation and lipid extraction following sonication-induced traumatic injury. 500msec pulses were transmitted at the supraorbital rim of C57BL/6j mice. Optic nerves were harvested at three time points to assess changes following exposure. Mitochondria were isolated and their lipids were extracted. Lipids were analyzed with mass spectrometry.

Table 1
Sample Identification. 32 optic nerves with an even distribution of males and females in each group. All samples were run in positive mode and negative mode two times.

| Sample | Treatment | Days Post Exposure | Sex | ESI Mode |
|--------|-----------|--------------------|-----|----------|
| C1     | No Sonication | N/A     | M   | Pos + Neg |
| C2     | No Sonication | N/A     | M   | Pos + Neg |
| C3     | No Sonication | N/A     | M   | Pos + Neg |
| C4     | No Sonication | N/A     | M   | Pos + Neg |
| C5     | No Sonication | N/A     | F   | Pos + Neg |
| C6     | No Sonication | N/A     | F   | Pos + Neg |
| C7     | No Sonication | N/A     | F   | Pos + Neg |
| C8     | No Sonication | N/A     | F   | Pos + Neg |
| D1_1   | Sonication  | 1       | M   | Pos + Neg |
| D1_2   | Sonication  | 1       | M   | Pos + Neg |
| D1_3   | Sonication  | 1       | M   | Pos + Neg |
| D1_4   | Sonication  | 1       | M   | Pos + Neg |
| D1_5   | Sonication  | 1       | F   | Pos + Neg |
| D1_6   | Sonication  | 1       | F   | Pos + Neg |
| D1_7   | Sonication  | 1       | F   | Pos + Neg |
| D1_8   | Sonication  | 1       | F   | Pos + Neg |
| D7_1   | Sonication  | 7       | F   | Pos + Neg |
| D7_2   | Sonication  | 7       | F   | Pos + Neg |
| D7_3   | Sonication  | 7       | F   | Pos + Neg |
| D7_4   | Sonication  | 7       | F   | Pos + Neg |
| D7_5   | Sonication  | 7       | M   | Pos + Neg |
| D7_6   | Sonication  | 7       | M   | Pos + Neg |
| D7_7   | Sonication  | 7       | M   | Pos + Neg |
| D7_8   | Sonication  | 7       | M   | Pos + Neg |
| D14_1  | Sonication  | 14      | M   | Pos + Neg |
| D14_2  | Sonication  | 14      | M   | Pos + Neg |
| D14_3  | Sonication  | 14      | M   | Pos + Neg |
| D14_4  | Sonication  | 14      | M   | Pos + Neg |
| D14_5  | Sonication  | 14      | F   | Pos + Neg |
| D14_6  | Sonication  | 14      | F   | Pos + Neg |
| D14_7  | Sonication  | 14      | F   | Pos + Neg |
| D14_8  | Sonication  | 14      | F   | Pos + Neg |


The mitochondria. We opted for a butanol-methanol (BUME) lipid extraction instead of the gold standard Bligh and Dyer method [6] after determining that the butanol-methanol method [7, 8] extracted more lipid species as well as higher grades of lipids in the small size of our samples (~ 1 mg) (unpublished data).

We used a Q-Exactive Orbitrap Mass Spectrometer to generate the raw data for each sample. All raw data were uploaded and processed with Lipidsearch 4.1.3. We provide the lipid nomenclature in Table 2 and the identified lipids based on Lipidsearch 4.1.3 in Table 3. Finally, all data were normalized and exported for bioinformatics analysis with Metaboanalyst 4.0. The lipid species heatmap (Figure 2), principal component analysis (PCA) and one-way ANOVA analysis were generated using Metaboanalyst 4.0 (Figure 3).
Table 2
LipidSearch 4.1.3 Nomenclature.

| Group | Abbreviations | Lipid Name |
|-------|---------------|------------|
| P- Choline | LPC | lysophosphatidylcholine |
|         | PAF | platelet-activating factor |
|         | PC | phosphatidylcholine |
|         | MePC | Methyl phosphatidylcholine |
| Ethanol | LPE | lysophosphatidylyethanolamine |
| Amine | PE | phosphatidylethanolamine |
|         | BisMePE | Bis-methyl phosphatidylethanolamine |
|         | dMePE | dimethylphosphatidylethanolamine |
| P- Serine | LPS | lysophosphatidylserine |
|         | PS | phosphatidylserine |
|         | BisMePS | Bis-methyl phosphatidylserine |
| P- Glycerol | LPG | lysophosphatidylglycerol |
|         | PG | phosphatidylglycerol |
|         | BisMePG | Bis-methyl phosphatidylglycerol |
| P- Inositol | LPI | lysophosphatidylinositol |
|         | PI | phosphatidylinositol |
|         | PIP | phosphatidylinositol |
|         | PIP2 | phosphatidylinositol |
|         | PIP3 | phosphatidylinositol |
| P- Ethanol | LPEt | lysophosphatidylethanolamine |
| Methanol | PET | phosphatidylethanolamine |
| P- Acid | LPA | lysophosphatic acid |
|         | BisMeLPA | Bis-methyl lysophosphatic acid |
|         | PA | phosphatic acid |
|         | BisMePA | Bis-methyl phosphatic acid |
|         | cPA | cyclic phosphatic acid |
| Methanol | LPMe | Lysophosphatidylmethanol |
| Sphingolipids | SM | sphingomyelin |
|         | LSM | lysosphingomyelin |
|         | phSM | sphingomyelin(phytosphingosine) |
| Neutral glycerolipid | MG | monoglyceride |
|         | DG | diglyceride |
|         | TG | triglyceride |
| Fatty Acid | FA | fatty acid |
| Cardiolipin | CL | Cardiolipin |
| Sphingoid base | So | Sphingosine |
|         | SoP | Sphingosine phosphate |
| Neutral Glycosphingolipids | CerG1 | Simple Glc series |
|         | CerG2 | Simple Glc series |
|         | CerG3 | Simple Glc series |
|         | CerG2GNAc1 | Simple Glc series |
|         | CerG3GNAc1 | Simple Glc series |
|         | CerG3GNAc2 ST | Simple Glc series Sulfatide |
| Glycosphingolipids | Cer | Ceramides |
|         | CerP | Ceramides phosphate |
|         | GM3 | Gangliosides |
|         | GM2 | Gangliosides |
|         | GM1 | Gangliosides |
|         | GD1a | Gangliosides |
|         | GD1b | Gangliosides |
|         | GD2 | Gangliosides |
|         | GD3 | Gangliosides |
|         | GT1a | Gangliosides |
|         | GT1b | Gangliosides |
|         | GT1c | Gangliosides |
|         | GT2 | Gangliosides |
|         | GT3 | Gangliosides |
|         | GQ1c | Gangliosides |
|         | GQ1b | Gangliosides |
(continued on next page)
Table 2 (continued)

| Group                      | Abbreviations | Lipid Name                                      |
|----------------------------|---------------|------------------------------------------------|
| Steroid                    | ChE           | Cholesterol Ester                               |
|                            | ZyE           | Zymosterol                                      |
|                            | SeE           | Stigmasterol ester                              |
|                            | SE            | Sitosterol ester                                |
|                            | AGlcSeE       | AcylGlcSitosterol ester                         |
|                            | D7ChE         | Deuterated Cholesterol Ester                    |
| Coenzyme                   | Co            | Coenzyme                                        |
| Fatty                      | OAFA          | (O-acyl)-1-hydroxy fatty acid                    |
| Ester                      | WE            | wax exter                                       |
|                            | AcCa          | Acyl Carnitine                                  |
| Glycoglycerolipid          | MGDG          | Monogalactosylinmonoacylglycerol                 |
|                            | DGDG          | Digalactosylmonoacylglycerol                     |
|                            | SQMG          | Sulfoquinovosylinmonoacylglycerol                |
|                            | SQDG          | Sulfoquinovosyldiacylglycerol                    |
| Neutral glycerolipid (deuterated) | D5DG  | Deuterated diglyceride                          |
|                            | D5TG          | Deuterated triglyceride                         |

Table 3
Identified LipidSearch 4.1.3 Lipids. All lipids as identified with base retention times and calculated masses displayed.

| Lipid Class | Lipid Species | Calculated Mass | Formula | Base Retention Time |
|-------------|---------------|-----------------|---------|---------------------|
| BisMePA     | BisMePA(18:2p/20:1) | 738.5563 | C43 H79 O7 N0 P1 | 14.204 |
| CerG1       | CerG1(18:0+p/0/12:1) | 799.6537 | C46 H89 O9 N1 | 14.055 |
|             | CerG1(18:0+p/0/24:1) | 827.685 | C48 H93 O9 N1 | 14.356 |
|             | CerG1(18:1/22:0/0) | 799.6537 | C46 H89 O9 N1 | 13.382 |
|             | CerG1(18:1/22:1) | 781.6432 | C48 H87 O8 N1 | 13.39 |
|             | CerG1(18:1/24:1) | 809.6745 | C48 H91 O8 N1 | 14.391 |
|             | CerG1(18:1/24:2) | 807.6588 | C48 H89 O8 N1 | 13.457 |
|             | CerG1(40:0+p/0/0) | 817.6643 | C46 H91 O10 N1 | 13.378 |
|             | CerG1(42:1+p/0) | 827.685 | C48 H93 O9 N1 | 13.892 |
| LPC         | LPC(16:0) | 495.3325 | C24 H50 O7 N1 P1 | 4.906 |
|             | LPC(18:0) | 523.3638 | C26 H54 O7 N1 P1 | 6.07 |
|             | LPC(18:1) | 521.3481 | C26 H52 O7 N1 P1 | 5.281 |
|             | LPC(18:2) | 519.3325 | C26 H50 O7 N1 P1 | 4.625 |
|             | LPC(20:4) | 543.3325 | C28 H50 O7 N1 P1 | 4.791 |
| PA          | PA(16:0/18:1) | 674.4887 | C37 H71 O8 N0 P1 | 34.197 |
|             | PA(18:0/18:1) | 702.52 | C39 H75 O8 N0 P1 | 35.43 |
| PC          | PC(16:0/18:1) | 759.5778 | C42 H82 O8 N1 P1 | 33.78 |
|             | PC(18:0/18:1) | 787.6091 | C44 H86 O8 N1 P1 | 34.914 |
|             | PC(32:0) | 733.5622 | C40 H80 O8 N1 P1 | 12.275 |
|             | PC(34:1) | 759.5778 | C42 H82 O8 N1 P1 | 12.665 |
|             | PC(36:1) | 787.6091 | C44 H86 O8 N1 P1 | 13.996 |
| PE          | PE(18:0/18:1) | 745.5622 | C41 H80 O8 N1 P1 | 14.41 |
|             | PE(18:0/18:1) | 729.5672 | C41 H80 O7 N1 P1 | 14.078 |
|             | PE(18:0/20:1) | 757.5985 | C43 H84 O7 N1 P1 | 14.93 |
|             | PE(18:1p/18:1) | 727.5516 | C41 H78 O7 N1 P1 | 13.052 |
| PET         | PET(16:0/18:1) | 702.52 | C39 H75 O8 N0 P1 | 35.43 |
| PS          | PS(18:0/18:1) | 789.552 | C42 H80 O10 N1 P1 | 14.155 |
|             | PS(18:0/20:1) | 817.5833 | C44 H84 O10 N1 P1 | 14.981 |
| SM          | SM(d42:2) | 812.6771 | C47 H93 O6 N2 P1 | 13.849 |
| TG          | TG(16:0/16:0/18:1) | 832.752 | C53 H100 O6 | 13.847 |
|             | TG(16:0/18:1/18:1) | 858.7676 | C55 H102 O6 | 14.736 |
|             | TG(16:0/18:1/18:2) | 856.752 | C55 H100 O6 | 14.254 |
|             | TG(18:0/16:0/18:1) | 860.7833 | C55 H104 O6 | 15.378 |
|             | TG(18:1/18:1/18:1) | 884.7833 | C57 H104 O6 | 15.135 |
|             | TG(18:1/18:1/18:2) | 882.7676 | C57 H102 O6 | 14.778 |
|             | TG(18:1/18:2/18:2) | 880.752 | C57 H100 O6 | 15.476 |
| dMePE       | dMePE(34:2p) | 727.5516 | C41 H78 O7 N1 P1 | 34.147 |
Figure 2. Heat map of mitochondrial lipid species. Lipid species changes depicted at control (uninjured), 1-day, 7-days, and 14-days post-sonication.

2. Experimental Design, Materials, and Methods

2.1. Animals

Animals were treated in accordance with the National Research Council’s Guide for the Care and Use of Laboratory Animals and the Association for Research in Vision and Ophthalmology (ARVO) Statement for the Use of Animals in Ophthalmic and Vision Research. All procedures involving mice were approved by the Institutional Animal Care and Use Committee (IACUC) at the University of Miami. C57BL/6J mice from Jackson Laboratory (Bar Harbor, ME, USA) were
Figure 3. One-Way Analysis of Variance and Principal Component Analysis. A. ANOVA displayed 18 statistically significant lipids out of 37 lipids identified. Only statistically significant lipids listed (in order from left to right). B. PCA with 95% confidence intervals

maintained in a temperature-regulated environment with a 12-hour light, 12-hour dark cycle. All mice were fed ad libitum. Two-month-old mice were used.

2.2. Sonication-Induced Traumatic Optic Neuropathy (SI-TON) Model

SI-TON was performed as described previously [4]. TON was induced in C57BL/6J mice with a Branson Digital Sonifier 450 (Branson Ultrasonics, Danbury, CT, USA) by a 3mm microtip probe in a soundproof chamber. Mice were anesthetized with vaporized isoflurane supplied with oxygen. Supraorbital fur was shaved, and the microtip probe was placed directly above the insertion point of the optic nerve into the optic canal. Left optic nerves were injured with a 500 msec shock at a 35% amplitude providing 20 kHz ultrasound waves. Following sonication, mice were placed in a new cage with thermal support until fully recovered.

2.3. Sample Preparation and Mitochondrial Isolation

Optic nerve samples were collected at 1 day, 7 days, and 14 days post-sonication. The mice brain was carefully removed to expose the underlying optic nerves and tracts. Optic nerves were harvested starting at the optic nerve head and dissecting at the optic chiasm. Optic nerves were dried and weighed to approximately 1 mg. Mitochondria were isolated using the Mitochondrial Isolation Kit for Tissue (PIERCE, Rockford, IL, USA). The protocol was modified for use in our
small samples. Briefly, optic nerves were washed twice in 1x Phosphate Buffered Saline (PBS) to remove all fat and debris. Samples were then resuspended in 200 μl of BupH PBS and carefully dounced 15 times. Homogenate was centrifuged at 1,000 x g at 4°C for 3 minutes and supernatant was discarded. The pellet was resuspended in 200μl of Reagent A and vortexed at maximum speed for 5 seconds. Samples were then incubated for 2 minutes on ice. 2.5 μl of Reagent B were added and incubated on ice for 5 minutes with 5 seconds of vortexing at every minute of incubation. 200 μl of Reagent C were added, the tube was inverted for mixing, and then centrifuged at 700 x g for 10 minutes. The pellet was discarded and supernatant was again centrifuged at 3,000 x g for 15 minutes. This resultant mitochondrial pellet was kept and supernatant was stored for cytosolic analysis. The mitochondrial pellet was washed with wash buffer and placed on ice for subsequent lipid extraction. Mitochondrial isolation was validated by dot blot targeting for TOM20, a mitochondrial membrane protein.

2.4. Butanol-Methanol (BUME) Lipid Extraction

Lipids were extracted using a modified BUME lipid extraction as performed by Cruz and Lofgren [7, 8]. Briefly, mitochondrial pellets were resuspended in 300 μl n-butanol: methanol 3:1 (v/v) and vortexed for one minute. 150 μl of heptane: ethyl acetate 3:1 (v/v) was then added to the test tube and vortexed for one minute followed by another 150 μl of heptane/ ethyl Acetate 3:1 (v/v) and another minute of vortexing. Phase separation was induced by adding 300 μl of 50 mM LiCl followed by vortexing for one minute. Here, with two layers visible, the samples were centrifuged at 2,700 x g for 10 minutes. The upper layer (organic) was collected and the bottom layer (aqueous) was re-extracted by phase separation two more times. The organic layers were combined and dried using a SpeedVac Concentrator. Samples were stored dry in -80°C for further processing. A bichinonic acid (BCA) protein assay was performed on the aqueous protein layer to normalize the lipid concentrations in each sample using the Pierce Micro BCA Protein Assay Kit (PIERCE, Rockford, IL, USA).

2.5. High Performance Liquid Chromatography and Mass Spectrometry

Lipid samples were resuspended in 50μl of chloroform: methanol 2:1 (v/v), placed in an ultrasonic water bath for 20 minutes, and then vortexed for 2 minutes. 30 μl were loaded into vials for four runs (two positive, two negative) of 5 μl each. Lipids were analyzed with liquid chromatography electrospray tandem mass spectrometry (LC-MS/ MS) using the Accela HPLC system and an orbitrap mass spectrometer (Q-Exactive, Thermo Scientific, Waltham, MA). An Acclaim 120 C18 3μm column (Thermo Scientific) was used with LC-MS grade methanol: water 60:40 (v/v) with 10mM ammonium acetate and methanol chloroform 60:40 (v/v) with 10mM ammonium acetate, as solvent A and B, respectively. A Heated Electrospray Ionization Source (HESI) was operated at a spray voltage of 4,415 V, a HESI vaporization temperature of 275°C, and an auxiliary gas flow of 15 arbitrary units. The scan range was set at 150-1500 m/z. The gradient ran at 35% to 100% Solvent B for 13 minutes, held at 35% solvent B for 2 minutes and finally brought up to 100% solvent A for 3 minutes and held for 2 minutes. The LC-MS/ MS method was validated for mitochondrial lipid analysis using external standards with known concentrations of cardiolipin.

2.6. Lipid Identification and Bioinformatics Analysis

Raw data produced by LC-MS/MS was uploaded to LipidSearch 4.1.3. The parameters were set to an M-score of 5.0, Productsearch, precursor (5/5) ppm, intensity threshold of 1.0% and quantitation and TopRank filter were turned on. ID quality were graded from A-D. All target classes
were selected. All adducts for both negative and positive modes were selected with the exception of \((\text{CH3CH2})_3\text{NH}^+\) and \((\text{CH3})_2\text{NH2}\). Once all peaks were identified, samples were aligned in positive mode, negative mode, and also all trial runs in one. Lipid identification was graded from A-C with an M-Score of 5. Any false positive peaks were rejected. All aligned data was exported and formatted for further analysis in Metaboanalyst 4.0. The data was normalized by pooling the control group, log transforming, scaling by mean-centering and dividing by the square root of the standard deviation (pareto scaling). A heat map (Figure 2) was generated along with PCA and one-way ANOVA (Figure 3) analyses. 18 of the 37 lipids were statistically significant (p-value set to 0.05).

**Declaration of Competing Interest**

The authors declare that they have no known competing financial interests or personal relationships which have, or could be perceived to have, influenced the work reported in this article.

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