HEARING LOSS IN ALZHEIMER DISEASE IS ASSOCIATED WITH ALTERED SERUM LIPIDOMIC BIOMARKER PROFILES

Daniel A. Llano[1,2], Lina K. Issa[1], Priya Devanarayan[3], Viswanath Devanarayan[4,5], Alzheimer's Disease Neuroimaging Initiative (ADNI).

1. Molecular and Cellular Biology, University of Illinois at Urbana-Champaign, Urbana, IL, USA
2. Carle Neuroscience Institute, Urbana, IL, USA
3. Department of Biology and Schreyer Honors College, Pennsylvania State University, University Park, PA, USA
4. GlaxoSmithKline, Collegeville, PA, USA
5. Department of Mathematics, Statistics and Computer Science, University of Illinois at Chicago, Chicago, IL, USA

Corresponding Author: Daniel A. Llano MD, PhD, d-llano@illinois.edu

Acknowledgment:

The authors thank Dr. Dinesh Barupal for useful discussions regarding the ADNI lipidomics dataset.

Abstract:

Recent data have found that aging-related hearing loss (ARHL) is associated with the development of Alzheimer Disease (AD). However, the nature of the relationship between these two disorders is not clear. There are multiple potential factors that link ARHL and AD, and previous groups have speculated that common metabolic dysregulation may underlie the propensity to develop both disorders. Here, we investigate the distribution of serum lipidomic biomarkers in AD subjects with or without hearing loss in a publicly available dataset. Serum levels of 349 known lipids from 16 lipid classes were measured in 185 AD patients. Using previously defined co-regulated sets of lipids, both age- and sex-adjusted, we found that lipid sets enriched in phosphatidylcholine and phosphatidylethanolamine showed a strong association with hearing loss. Examination of biochemical classes confirmed these relationships and found that serum phosphatidylcholine levels were significantly lower in AD subjects with hearing loss. A similar relationship was not found in normal subjects. These data suggest that a

synergistic relationship may exist between AD, hearing loss and metabolic biomarkers, such that in the context of a pathological state such as AD, alterations in serum metabolic profiles are also shared with hearing loss hearing loss. These data also point to a potential role for phosphatidylcholine, a molecule with antioxidant properties, in the underlying pathophysiology of ARHL in the context of AD, which has implications for our understanding and potential treatment of both disorders.

Introduction:

Aging-related hearing loss (ARHL) and Alzheimer Disease (AD) are common disabling disorders in the elderly. Over the age of 65, approximately 10% of individuals develop AD, while approximately 40% develop ARHL (Hebert, Weuve, Scherr, & Evans, 2013; Nash et al., 2011), Both are rising in prevalence as the population ages, with an estimated 83 million individuals over the age of 65 in the U.S. by the year 2050 (Ortman, Velkoff, & Hogan, 2014). Recent data have revealed an association between these two disorders, such that the likelihood of developing cognitive impairment, and ultimately AD, is increased in individuals with ARHL (Ford et al., 2018; Golub et al., 2017; Lin, Ferrucci, et al., 2011; Lin, Metter, et al., 2011; Lin, Thorpe, Gordon-Salant, & Ferrucci, 2011; Lin et al., 2013; Panza, Solfrizzi, & Logroscino, 2015; Thomson, Auduong, Miller, & Gurgel, 2017). This relationship holds true even when adjusting for age, sex and multiple other potentially confounding variables such as comorbid illness. A causal association has not been identified, though multiple mechanisms by which hearing loss may lead to AD have been proposed (reviewed in (Nadhimi & Llano, 2020)).

ARHL and AD do share several potential biological substrates. Both are associated with metabolic stress and diminished mitochondrial function (Fujimoto & Yamasoba, 2014; Wang et al., 2014). ARHL is also associated with more traditional markers of AD such as increases in CSF tau or diminished hippocampal and entorhinal cortical volume (Xu et al., 2019). Recently, it has been suggested that AD may be associated with widespread dysregulation of lipid metabolism (Kao, Ho, Tu, Jou, & Tsai, 2020) and plasma lipid profiles have been shown to correlate with multiple AD-related biomarkers (Barupal et al., 2019). Lipid dysregulation may also play a role in the development of hearing loss (Campbell, Rybak, & Khardori, 1996). It is therefore possible that an underlying process of metabolic dysregulation, including altered lipid homeostasis, may account for the relationship between AD and ARHL.

Lipids are a major component of biological membranes and integral for neuronal function. The term lipids encompasses all fatty acids and their derivatives; the three main classes of lipids are triglycerides, phospholipids, and sterols. Lipid metabolism is the catabolic and synthetic

mechanism through which energy is extracted from fats and fats are produced, respectively. The availability of lipids is derived from three sources: our diet, adipose tissue storage, and the liver's synthetic capacity. Fats ingested in the diet enter the gastrointestinal tract, digested by pancreatic lipases in the small intestine and then imported across the intestinal mucosa where they are resynthesized. The resynthesized fats are then packaged along with cholesterol into chylomicrons which allow for nonpolar substances to move within the aqueous environment of our lymphatic and circulatory systems. These fats are then oxidized through β-oxidation for energy production or re-esterized for storage in adipose tissue. Alternatively, the resynthesized fats in the small intestine can be distributed to the liver through portal circulation or to adipose tissue. Lipids derived from endogenous synthesis in the liver are packaged into very-low-density lipoproteins that are transported to tissue or stored in adipose tissue. Fat stores in adipose tissue are mobilized for energy production by the action of hormone-sensitive lipase as needed.

Given the potential roles for lipids dysregulation in both AD and ARHL, in the current study, we examined the distribution of serum lipids in subjects with AD, with and without hearing loss, using a publicly-available dataset (Alzheimer Disease Neuroimaging Initiative, ADNI).

Methods:

Database:

The ADNI database (adni.loni.usc.edu) utilized in this research was launched in 2003 as a public-private partnership, led by Principal Investigator Michael W. Weiner, MD. The primary goal of ADNI has been to test whether serial MRI, PET, other biological markers, and clinical and neuropsychological assessments can be combined to measure the progression of MCI and early AD. For up-to-date information, see www.adni-info.org. This study was conducted across multiple clinical sites and was approved by the Institutional Review Boards of all of the participating institutions. Informed written consent was obtained from all participants at each site. Data used for the analyses presented here were accessed on June 25, 2020.

Lipid analysis:

Details of lipid extraction and measurement as well as quality control measures have been previously described (Barupal et al., 2018). In brief, fasting serum samples were obtained from subjects during the baseline visit. Lipids were extracted using organic solvents. Serum extracts were then analyzed using liquid chromatography with mass spectrometry. After quality control measures, we have data from a total of 349 known lipids from 16 classes (see Table 1 for listing of lipid classes). The lipid subclasses in the ADNI serum lipidomics data set used in this study

include acylcarnitine, fatty acid, cholesteryl ester, lysophosphatidylcholine, lysophosphatidylethanolamine, phosphatidylcholine, phosphatidylethanolamine, phosphatidylinositol, plasmalogen phosphatidylcholine, plasmalogen phosphatidylethanolamine, ceramide, glucosylceramide, sphingomyelin, diacylglycerol, and triacylglycerol (see Table 1 for listing of lipid classes).

Hearing loss assessment:

Hearing was not systematically measured in the ADNI database. Similar to a previous report (Xu et al., 2019), we used subjective hearing loss complaints found in the following datasheets (downloaded June 25, 2020): ADSXLIST.csv, BLSCHECK.csv, INITHEALTH.csv, MEDHIST.csv, NEUROEXM.csv, PHYSICAL.csv, RECBLLOG.csv, RECMHIST.csv. We used the search terms “hear”, “auditory”, “ear”, “deaf”, “presbycusis”, and “HOH (hard of hearing)” and eliminated those reports that were clearly not related to aging-related hearing loss (e.g., skin cancer on ear, earwax, etc.) and eliminated duplicates. Subjects having a hearing complaint are labeled in this study as “hearing loss” or HL. Other subjects are listed as “non-hearing loss” or NHL, notwithstanding the fact that hearing was not objectively measured (see below).

Statistical Methods:

The effect of each individual lipid species on hearing loss in AD subjects was assessed via analysis of covariance (ANCOVA) after adjusting for gender and age as covariates, and log transforming he lipid expression values. Samples with absolute value of studentized residuals from this model exceeding 3 were identified as outliers and excluded from further analysis. The summary measures reported from this analysis include the area under the Receiver Operating Characteristic curve (ROC AUC), covariates-adjusted significance (p-value), and false discovery rate (Benjamini & Hochberg, 1995).

The effect of each of the 16 known lipid classes and 28 empirically derived lipid sets (Barupal et al., 2019) on hearing loss in AD subjects was assessed via "lipid set analysis" (LSA). See supplementary Table 1 for a listing of the lipids in each of the 28 sets. This LSA analysis on the lipid classes and lipid sets was based on the maxmean statistic of the gene-set-analysis algorithm (Efron & Tibshirani, 2007). which was applied on the residuals from the above ANCOVA model on the individual lipid species in order to adjust for the effects of age and gender. Individual subject level standardized composite scores were determined for each lipid class and each lipid set from this algorithm. These scores were then used to assess the effect of each of the lipid classes and lipid sets on hearing loss in AD subjects. The results were

summarized in terms of ROC-AUC, covariates-adjusted significance (p-value) and false discovery rate (q-value). Lipid sets with q-value < 0.05 were considered as statistically significant. The corresponding lipid classes and individual lipid specifies with Bonferroni adjusted p-value < 0.05 were highlighted and studied further in terms of their potential connections to hearing loss in AD subjects.

Results:

Demographics:

Data were obtained from 185 subjects with AD. Of the 185, 40 (21.6%) reported hearing loss (HL). HL subjects were not significantly different in age than NHL subjects (HL: 77.2 ± 5.8 years [SD], NHL: 74.8 ± 7.7 years [SD], $p > 0.05$). HL subjects were more likely to be men than control subjects (NHL = 47% men, HL = 68% men, $p < 0.05$, Chi-Square). HL and control subjects did not differ significantly in average ADAS13 scores (HL: 30.4 ± 8.0 [SD], NHL: 28.8 ± 7.6 [SD], $p > 0.05$) or body mass index (HL: 26.0 ± 4.1 kg/m^2 [SD], NHL: 25.3 ± 3.8 kg/m^2 [SD], $p > 0.05$, see Table 2).

Lipidomic biomarker sets that separate HL from NHL subjects:

Levels of 349 lipids were measured across 16 classes. Because levels of many of the lipids are strongly correlated due to co-regulation, and because of the high potential for false discovery when comparing the levels of all 349 lipids, we attempted to reduce the data by grouping the lipids. A previous report measured correlations between all of the serum lipid biomarkers, and using a dynamic clustering algorithm known as dynamicTreeCut, determined that 28 co-regulated sets of lipids were present (Barupal et al., 2019). They also found that many of these lipid sets were associated with either AD diagnosis or AD biomarkers. Although most of the sets were homogeneous (or near-homogeneous) clusters of single lipid types, others comprised a mixture of lipids (see supplementary Table 1 for listing of lipids in each class).

Given the robust performance of these clusters to signal changes in AD biomarkers, we asked whether these same clusters were also associated with the presence of HL. The p- and q- values for the 28 groups of lipids are shown in Table 3. We found that two sets of lipids correlated with the presence of hearing loss: set 23 and set 4, both with p- and q-values below 0.05, with set 23 producing the best performance. We therefore focused on the lipids found in these two sets for subsequent analyses of lipid class and individual lipids.

Lipid classes and individual lipids that separate HL from NHL subjects:

Using the biomarker sets to narrow our hypotheses about which lipids exhibit signal changes in hearing, we attempted to determine which lipid classes were most significantly associated with HL. Within the two top sets identified above, 25 lipids in 7 classes were identified, with only the phosphatidylcholine class surviving correction for multiple comparisons (uncorrected p-value = 0.0057, Bonferroni corrected to 0.04). See Figure 1 for boxplots of the 7 biomarker classes comparing HL and NHL subjects See Table 4 for a listing of lipid classes found in sets 4 and 23 and their associated capacity to separate HL from NHL subjects.

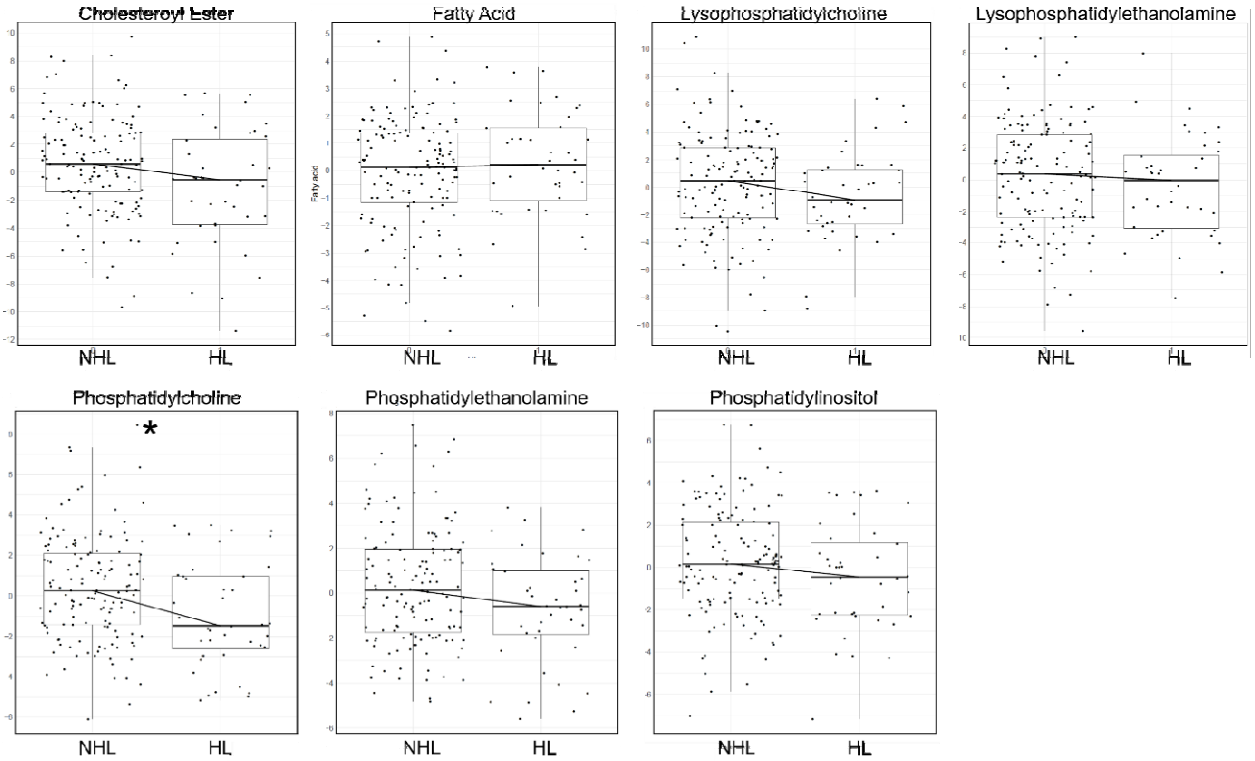

*Figure 1: Box plots showing the mean, first and third quartiles of the distributions, demonstrating differences in levels of lipids in the seven classes of lipids identified as parts of sets 4 and 23 from (Barupal et al., 2019), distinguishing between HL and NHL subjects. Shown are standardized values (centered by mean and divided by standard deviation), after adjusting for age and gender as covariates. * Bonferroni-corrected p-value of < 0.05.*

Using the 25 lipids identified above, the most commonly-appearing lipid class was phosphatidylcholine (14/25 lipids or 56%), which is significantly greater than the proportion of all tested lipids that were in the phosphatidylcholine class (82/349 lipids or 23.4%, $p<0.05$, Chi-Squared test). See Table 5 for a listing of individual lipids in sets 4 and 23 and their associated capacity to separate HL from NHL subjects. Both of these analyses point to phosphatidylcholine levels as the main factor distinguishing between HL and NHL subjects.

Analysis of non-AD subjects and APOE:

Similar analyses were done in subjects with mild cognitive impairment (MCI) and control subjects without memory loss. None of the lipid sets were found to differentiate HL from NHL subjects in either control or MCI cohorts. Subjects across all groups (control, MCI and AD) were also separated based on genotype (having at least one copy of APOE4 or none), and no association was found between genotype and likelihood of HL.

Discussion:

In the current study, 349 serum biomarkers were measured in 185 subjects with AD. Using previously-identified co-regulated sets of biomarkers (Barupal et al., 2019), we found two sets of lipids that were strongly associated with the presence of HL. Within these sets, the most common class of lipids was phosphatidylcholine, and as a class and as individual biomarkers, phosphatidylcholines were found to be significantly diminished in individuals with HL. Similar analyses in non-AD subjects (control and MCI) did not reveal significant associations between lipidomic biomarkers and HL

Weaknesses in the study:

Hearing loss in this study was assessed in a non-systematic way – via reports obtained from the subjects. Previous data have established concordance values between subjective and objective hearing loss ranging from 65-77% depending on demographic factors (Kamil, Genther, & Lin, 2015). Although there are several publicly-available databases that have measured hearing loss objectively (e.g., Baltimore Longitudinal Study of Aging or National Health and Nutrition Examination Survey), these did not systematically measure an extensive panel of lipid biomarkers. Conversely, despite the richness of biomarker data available in ADNI, hearing was not systematically measured. Thus, additional future work in subjects with objectively-measured hearing loss will be required to confirm the associations reported here.

In addition, as an observational study, the current work cannot be used to support the idea that supplementation of phosphatidylcholine can protect against ARHL in subjects with AD. It is possible that phosphatidylcholine levels and ARHL are related by a third, unmeasured, factor. Only a prospective, randomized and blinded trial can determine whether phosphatidylcholine can improve ARHL.

Phosphatidylcholine, Alzheimer Disease and hearing loss:

Phosphatidylcholine is one of the major phospholipids and a fundamental structural component of cell membranes. Previous studies have shown that polyunsaturated phosphatidylcholine in the plasma membrane serves more than just a structural role; it also plays a rate-limiting role in the activation of enzymatic antioxidants situated in the cell membrane. These antioxidants serve to protect the cell membrane from damage by reactive oxygen species; they include superoxide dismutase and glutathione, catalase, glutathione S-transferase, and glutathione peroxidase (Seidman, Khan, Tang, & Quirk, 2002). The process of lipid peroxidation has previously been linked to the presence of free radicals (Su et al., 2019); meaning that reactive oxygen species have also been implicated in injury to polyunsaturated fatty acids in the cell membrane ultimately resulting in cell membrane damage (Seidman et al., 2002). The effects of reactive oxygen species on cell death are well established and it is well known that these radicals can lead to either apoptosis or oncotic necrosis depending on whether there's significant ATP depletion in the cell or if an executioner caspase is activated, respectively. Reactive oxygen species arise from normal mitochondrial respiration and their generation is found to be increased during periods of prolonged hypoperfusion. As an implication of aging, one can expect to observe decreased blood flow within the circulatory system, specifically in the inner ear (Seidman et al., 2002). The cochlea is further vulnerable to oxidative stress due to increased mitochondrial respiration from the high metabolic demands of their mechanosensory hair cells. These increased levels of reactive oxygen species leading to oxidative stress can ultimately lead to permanent cochlear degeneration as is seen in patients with hearing loss (Gonzalez-Gonzalez, 2017).

The lipids measured in this study were extracted from blood samples which brings about the question of how lipids enter circulation. Fats ingested through our diet reach the small intestine where short- and medium-chain fatty acids are absorbed directly into the blood via intestine capillaries and travel through the portal vein. In the liver, fat is packaged into very low density lipoproteins for transport in the bloodstream to extrahepatic tissues. On the contrary, long-chain fatty acids are absorbed intact in the small intestine, incorporated into chylomicrons, and secreted into lymphatic circulation. Additionally, adipocytes can release stored fatty acids into the blood as lipid levels in the blood decrease. Increasing evidence suggests that some fatty acids can be synthesized in the brain, but that essential fatty acids still have to be transported across the blood-brain barrier (Bruce, Zsombok, & Eckel, 2017). Additional studies further suggest that this is a dynamic process with active daily turnover (Rapoport, Chang, & Spector, 2001). The exact mechanism behind how fats enter the brain is still unclear. One study performed on cholesterol homeostasis and hearing loss indicates that since the blood-brain

barrier prevents the uptake of this lipoprotein from circulation, all brain cholesterol is synthesized in astrocytes; further, excess cholesterol is metabolized into 24 (S)-hydroxycholesterol before secretion from the blood-brain barrier to the liver (Malgrange, Varela-Nieto, de Medina, & Paillasse, 2015).

Regarding phospholipids like phosphatidylcholine, the rat brain has been shown to be capable of synthesizing phosphatidylcholine via the phosphatidylethanolamine N - methyltransferase pathway (Crews, Hirata, & Axelrod, 1980) and CDP-choline pathway (Paoletti, Elena, Domizi, & Banchio, 2011). In addition to the synthetic capacity of brain tissue, some believe that although circulating phosphatidylcholine cannot cross the blood-brain barrier, its choline component has the ability to do so through the action of choline transporters; once this choline enters the brain, it can then be incorporated into the brain's own PC (Allen & Lockman, 2003). Increasing evidence has also shown that there is a net efflux of choline from the brain via a carrier-mediated efflux transporter system; this system plays an important role in choline turnover in the brain (Lee & Kang, 2008).

There has also been evidence of altered phosphatidylcholine metabolism in AD (Whiley et al., 2014; Zamroziewicz, Zwilling, & Barbey, 2016). PCs interact with ApoE as part of the HDL group, and are implicated in cholesterol transport. The enzymes that catalyze the breakdown of phosphatidylcholine to phosphatidate, or glycerophosphocholine and free fatty acids have been directly associated with AD. Alterations in reaction cascades of phospholipase D enzymes has been linked to neurodegenerative processes, with activation of phospholipase A2 family enzymes by Aβ peptide in neurons, in turn releasing secondary lipid messengers (Sanchez-Mejia & Mucke, 2010). A 2014 study concluded that phosphatidylcholine regulation was affected in AD, and that it could be linked to the roles of phospholipase A2 and phospholipase D1 in Aβ activation (Whiley et al., 2014). A 2019 study reports that plasma levels of phosphatidylcholine-DHA have been found to predict the occurrence of dementia (Patrick, 2019). Phosphatidylcholine-DHA levels in the highest quartile had 47% lower risk of dementia than those with levels in lower 3 quartiles, independent of ApoE4 allele. Low levels of lysoPC predicted diagnosis of mild dementia and AD within 2-3 year with 90% accuracy (Patrick, 2019). Low plasma phosphatidylcholine levels as well as their components (long-chain polyunsaturated fatty acid docosahexaenoic acid and choline) have been found are predictive of cognitive decline (Zamroziewicz et al., 2016).

Conclusion:

Thus, in the current study, we observed that in the context of AD, lower serum levels of phosphatidylcholine were associated with ARHL. The fact that this association was found in AD subjects, but not in non-AD subjects, suggests that there is an interaction between the presence of AD and the relationship between phosphatidylcholine and ARHL. Given that AD is associated with diminished brain mitochondrial function and increased levels of lipid peroxidation, it is possible that individuals with AD may not have the metabolic reserve to withstand additional metabolic stressors, such as declining levels of antioxidant molecules such as phosphatidylcholine. These data also suggest that normalizing phosphatidylcholine levels in AD subjects, but not in non-AD subjects, may have a role in for the treatment or prevention of ARHL. Future studies will need to be done to investigate the potential therapeutic role of phosphatidylcholine in this context.

Tables:

Table 1: Listing of lipid classes in the current study

Lipid Classes	Lipid Count
Acylcarnitine	9
Ceramide	19
Cholesterol	1
Cholesteroyl ester	8
Diacylglycerol	13
Fatty acid	29
Glactoylceramide	1
Glucosylceramide	6
Lactosylceramide	1
Lysophosphatidylcholine	22
Lysophosphatidylethanolamine	4
Phosphatidylcholine	82
Phosphatidylethanolamine	25
Phosphatidylinositol	11
Sphingomyelin	34
Triacylglycerol	84

309 Table 2: Demographic variables. *p<0.05

		NHL	HL
n (# of AD subjects)		145	40
Gender* (n)	**F**	77	13
	M	68	27
Age in years (Mean +/- SD)		74.8 (7.7)	77.2 (5.8)
BMI in kg/m^2 (Mean +/- SD)		25.32 (3.8)	26 (4.1)
ADAS13 (Mean +/- SD)		28.6 (7.6)	30.4 (8)

Table 3: Table of lipid sets derived from from Barupal et al. 2019 and their performance in distinguishing HL from NHL subjects. ROC AUC = receiver-operator characteristic area under the curve.

Lipid Set	Median (HN)	Median (HL)	ROC AUC	p-value (unadj.)	q-value (FDR-BH)
Set.23	0.54	-2.1	0.66	0.0006	0.0175
Set.4	0.16	-1.06	0.64	0.0032	0.0447
Set.6	-0.05	0.35	0.62	0.0148	0.1332
Set.25	1	-1.3	0.62	0.019	0.1332
Set.3	0.37	-0.74	0.6	0.0297	0.1664
Set.16	-0.16	0.27	0.59	0.0529	0.2171
Set.10	0.26	-1.18	0.6	0.0543	0.2171
Set.14	0.51	-1.16	0.6	0.0705	0.2467
Set.11	0.55	-1.21	0.61	0.0802	0.2495
Set.27	0.6	-1.61	0.58	0.1025	0.2871
Set.19	0.2	-0.35	0.58	0.1238	0.3014
Set.7	0.06	-0.74	0.58	0.1292	0.3014
Set.8	0.31	-0.26	0.57	0.1694	0.3467
Set.28	0.4	-0.12	0.56	0.1788	0.3467
Set.15	0.71	-0.31	0.56	0.1857	0.3467
Set.20	0.22	-0.45	0.56	0.2013	0.3523
Set.13	-0.67	-1.3	0.56	0.2554	0.4206
Set.17	0.34	0.2	0.47	0.2836	0.4387
Set.24	-0.4	-1.43	0.56	0.3135	0.4387
Set.2	-0.2	0.25	0.47	0.3153	0.4387
Set.9	0.54	-0.33	0.55	0.329	0.4387
Set.1	-0.3	-1.07	0.55	0.3879	0.4937
Set.26	0.02	0.58	0.48	0.4248	0.5171
Set.22	-0.18	-0.35	0.53	0.4606	0.5359
Set.18	-0.1	-0.68	0.54	0.4785	0.5359
Set.5	0.54	-0.55	0.53	0.6265	0.6747
Set.21	-0.21	0.32	0.5	0.8784	0.9109
Set.12	0.09	0.07	0.51	0.9827	0.9827

318 Table 4 Table of lipid classes derived from sets 4 and 23 from Barupal et al. 2019 and their
319 performance in distinguishing HL from NHL subjects.

Lipid Class	Median (NHL)	Median (HL)	ROC AUC	p-value
Phosphatidylcholine	0.25	-1.48	0.63	0.0057
Phosphatidylethanolamine	0.13	-0.6	0.59	0.0216
Cholesteroyl.ester	0.58	-0.53	0.62	0.0239
Phosphatidylinositol	0.16	-0.45	0.58	0.1142
Lysophosphatidylcholine	0.48	-0.93	0.58	0.1255
Lysophosphatidylethanolamine	0.35	-0.02	0.55	0.3185
Fatty.acid	0.12	0.22	0.54	0.3344

320

Table 5: Table of lipids derived from sets 4 and 23 from Barupal et al. 2019 and their performance in distinguishing HL from NHL subjects.

Lipid ID	Lipid Class	Lipid Set	Median (NHL)	Median (HL)	Fold Change (HL/HN)	p-value
UCD.Lipid.162	Phosphatidylcholine	Set-23	63671	52397	0.82	0.0003
UCD.Lipid.163	Phosphatidylcholine	Set-23	29367	24419.5	0.83	0.0006
UCD.Lipid.148	Phosphatidylcholine	Set-4	5056746	4380450.5	0.87	0.0010
UCD.Lipid.161	Phosphatidylcholine	Set-23	46893	39148.5	0.83	0.0014
UCD.Lipid.164	Phosphatidylcholine	Set-23	20525	16734.5	0.82	0.0033
UCD.Lipid.17	Cholesteroyl ester	Set-4	254617	198944.5	0.78	0.0055
UCD.Lipid.150	Phosphatidylcholine	Set-4	58706	49712	0.85	0.0069
UCD.Lipid.406	Phosphatidylcholine	Set-4	130587	116420	0.89	0.0079
UCD.Lipid.128	Lysophosphatidylcholine	Set-4	45190	37318.5	0.83	0.0130
UCD.Lipid.451	Phosphatidylethanolamine	Set-23	5726.33	4968	0.87	0.0149
UCD.Lipid.143	Phosphatidylcholine	Set-4	23780	19794	0.83	0.0150
UCD.Lipid.409	Phosphatidylcholine	Set-4	69527	57894	0.83	0.0163
UCD.Lipid.149	Phosphatidylcholine	Set-4	35516.5	27777	0.78	0.0166
UCD.Lipid.462	Phosphatidylinositol	Set-4	9408	8256	0.88	0.0183
UCD.Lipid.447	Phosphatidylethanolamine	Set-23	12761	11301.5	0.89	0.0197
UCD.Lipid.450	Phosphatidylethanolamine	Set-23	12513.79	10888.5	0.87	0.0217
UCD.Lipid.410	Phosphatidylcholine	Set-4	7910	7552	0.95	0.0310
UCD.Lipid.145	Phosphatidylcholine	Set-4	22891	19311	0.84	0.0329
UCD.Lipid.126	Lysophosphatidylcholine	Set-4	14172.5	12585	0.89	0.0428
UCD.Lipid.399	Phosphatidylcholine	Set-4	98283	79011	0.80	0.0661
UCD.Lipid.16	Cholesteroyl ester	Set-4	179126	125757	0.70	0.0846
UCD.Lipid.442	Phosphatidylethanolamine	Set-4	1817.5	1492	0.82	0.1016
UCD.Lipid.381	Lysophosphatidylethanolamine	Set-4	5577.5	5206	0.93	0.2087
UCD.Lipid.517	Fatty acid	Set-4	111966	100323	0.90	0.5032
UCD.Lipid.513	Fatty acid	Set-4	22307	21665	0.97	0.7021

References:

Allen, D., & Lockman, P. (2003). The blood-brain barrier choline transporter as a brain drug delivery vector. *Life sciences, 73*(13), 1609-1615.

Barupal, D. K., Baillie, R., Fan, S., Saykin, A. J., Meikle, P. J., Arnold, M., . . . ADNI. (2019). Sets of coregulated serum lipids are associated with Alzheimer's disease pathophysiology. *Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring, 11*, 619-627.

Barupal, D. K., Fan, S., Wancewicz, B., Cajka, T., Sa, M., Showalter, M. R., . . . Kaddurah-Daouk, R. (2018). Generation and quality control of lipidomics data for the alzheimer's disease neuroimaging initiative cohort. *Scientific data, 5*, 180263.

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: a practical and powerful approach to multiple testing. *Journal of the Royal statistical society: series B (Methodological), 57*(1), 289-300.

Bruce, K. D., Zsombok, A., & Eckel, R. H. (2017). Lipid processing in the brain: a key regulator of systemic metabolism. *Frontiers in endocrinology, 8*, 60.

Campbell, K. C., Rybak, L. P., & Khardori, R. (1996). Sensorineural hearing loss and dyslipidemia. *American journal of audiology, 5*(3), 11-14.

Crews, F. T., Hirata, F., & Axelrod, J. (1980). Identification and properties of methyltransferases that synthesize phosphatidylcholine in rat brain synaptosomes. *Journal of neurochemistry, 34*(6), 1491-1498.

Efron, B., & Tibshirani, R. (2007). On testing the significance of sets of genes. *The annals of applied statistics, 1*(1), 107-129.

Ford, A. H., Hankey, G. J., Yeap, B. B., Golledge, J., Flicker, L., & Almeida, O. P. (2018). Hearing loss and the risk of dementia in later life. *Maturitas, 112*, 1-11.

Fujimoto, C., & Yamasoba, T. (2014). Oxidative stresses and mitochondrial dysfunction in age-related hearing loss. *Oxidative medicine and cellular longevity, 2014*.

Golub, J. S., Luchsinger, J. A., Manly, J. J., Stern, Y., Mayeux, R., & Schupf, N. (2017). Observed hearing loss and incident dementia in a multiethnic cohort. *Journal of the American Geriatrics Society, 65*(8), 1691-1697.

Gonzalez-Gonzalez, S. (2017). The role of mitochondrial oxidative stress in hearing loss. *Neurol. Disord. Ther, 1*, 1-5.

Hebert, L. E., Weuve, J., Scherr, P. A., & Evans, D. A. (2013). Alzheimer disease in the United States (2010–2050) estimated using the 2010 census. *Neurology, 80*(19), 1778-1783.

Kamil, R. J., Genther, D. J., & Lin, F. R. (2015). Factors associated with the accuracy of subjective assessments of hearing impairment. *Ear and hearing, 36*(1), 164.

Kao, Y.-C., Ho, P.-C., Tu, Y.-K., Jou, I., & Tsai, K.-J. (2020). Lipids and Alzheimer's Disease. *International journal of molecular sciences, 21*(4), 1505.

Lee, N.-Y., & Kang, Y.-S. (2008). The efflux transport of choline through blood-brain barrier is inhibited by Alzheimer's disease therapeutics. *Biomolecules & Therapeutics, 16*(3), 179-183.

Lin, F. R., Ferrucci, L., Metter, E. J., An, Y., Zonderman, A. B., & Resnick, S. M. (2011). Hearing loss and cognition in the Baltimore Longitudinal Study of Aging. *Neuropsychology, 25*(6), 763.

Lin, F. R., Metter, E. J., O'Brien, R. J., Resnick, S. M., Zonderman, A. B., & Ferrucci, L. (2011). Hearing loss and incident dementia. *Archives of Neurology, 68*(2), 214-220.

Lin, F. R., Thorpe, R., Gordon-Salant, S., & Ferrucci, L. (2011). Hearing loss prevalence and risk factors among older adults in the United States. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 66*(5), 582-590.

Lin, F. R., Yaffe, K., Xia, J., Xue, Q.-L., Harris, T. B., Purchase-Helzner, E., . . . Simonsick, E. M. (2013). Hearing loss and cognitive decline in older adults. *JAMA internal medicine, 173*(4), 293-299.

Malgrange, B., Varela-Nieto, I., de Medina, P., & Paillasse, M. R. (2015). Targeting cholesterol homeostasis to fight hearing loss: a new perspective. *Frontiers in aging neuroscience, 7*, 3.

Nadhimi, Y., & Llano, D. A. (2020). Does hearing loss lead to dementia? A review of the literature. *Hearing research*, 108038.

Nash, S. D., Cruickshanks, K. J., Klein, R., Klein, B. E., Nieto, F. J., Huang, G. H., . . . Tweed, T. S. (2011). The prevalence of hearing impairment and associated risk factors: the Beaver Dam Offspring Study. *Archives of Otolaryngology–Head & Neck Surgery, 137*(5), 432-439.

Ortman, J. M., Velkoff, V. A., & Hogan, H. (2014). *An aging nation: the older population in the United States*: United States Census Bureau, Economics and Statistics Administration, US ….

Panza, F., Solfrizzi, V., & Logroscino, G. (2015). Age-related hearing impairment—a risk factor and frailty marker for dementia and AD. *Nature Reviews Neurology, 11*(3), 166.

Paoletti, L., Elena, C., Domizi, P., & Banchio, C. (2011). Role of phosphatidylcholine during neuronal differentiation. *IUBMB life, 63*(9), 714-720.

Patrick, R. P. (2019). Role of phosphatidylcholine-DHA in preventing APOE4-associated Alzheimer's disease. *The FASEB Journal, 33*(2), 1554-1564.

Rapoport, S. I., Chang, M. C., & Spector, A. A. (2001). Delivery and turnover of plasma-derived essential PUFAs in mammalian brain. *Journal of lipid research, 42*(5), 678-685.

Sanchez-Mejia, R. O., & Mucke, L. (2010). Phospholipase A2 and arachidonic acid in Alzheimer's disease. *Biochim Biophys Acta, 1801*(8), 784-790. doi:10.1016/j.bbalip.2010.05.013

Seidman, M. D., Khan, M. J., Tang, W. X., & Quirk, W. S. (2002). Influence of lecithin on mitochondrial DNA and age-related hearing loss. *Otolaryngology—Head and Neck Surgery, 127*(3), 138-144.

Su, L.-J., Zhang, J.-H., Gomez, H., Murugan, R., Hong, X., Xu, D., . . . Peng, Z.-Y. (2019). Reactive oxygen species-induced lipid peroxidation in apoptosis, autophagy, and ferroptosis. *Oxidative medicine and cellular longevity, 2019*.

Thomson, R. S., Auduong, P., Miller, A. T., & Gurgel, R. K. (2017). Hearing loss as a risk factor for dementia: a systematic review. *Laryngoscope investigative otolaryngology, 2*(2), 69-79.

Wang, X., Wang, W., Li, L., Perry, G., Lee, H.-g., & Zhu, X. (2014). Oxidative stress and mitochondrial dysfunction in Alzheimer's disease. *Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease, 1842*(8), 1240-1247.

Whiley, L., Sen, A., Heaton, J., Proitsi, P., García-Gómez, D., Leung, R., . . . Mecocci, P. (2014). Evidence of altered phosphatidylcholine metabolism in Alzheimer's disease. *Neurobiology of aging, 35*(2), 271-278.

Xu, W., Zhang, C., Li, J.-Q., Tan, C.-C., Cao, X.-P., Tan, L., . . . Initiative, A. s. D. N. (2019). Age-related hearing loss accelerates cerebrospinal fluid tau levels and brain atrophy: a longitudinal study. *Aging (Albany NY), 11*(10), 3156.

Zamroziewicz, M. K., Zwilling, C. E., & Barbey, A. K. (2016). Inferior prefrontal cortex mediates the relationship between phosphatidylcholine and executive functions in healthy, older adults. *Frontiers in aging neuroscience, 8*, 226.

422 Supplemental Table 1: Listing of lipids in all empirically-derived sets from Barupal et al. 2019

Lipid Set	Lipid Class	Lipid ID	Network Label	FullName	AcylChainInformation	Adduct	RT	MZ
Set-1	Triacylglycerol	UCD.Lipid.173	TG 42:0	TG (42:0)	TG (14:0_14:0_14:0)	[M+NH4]+	9.43	740.6763
Set-1	Triacylglycerol	UCD.Lipid.174	TG 40:0	TG (40:0)	TG(12:0_12:0_16:0)	[M+NH4]+	9.1	712.6419
Set-1	Triacylglycerol	UCD.Lipid.177	TG 42:2	TG (42:2)	TG(12:0_12:0_18:2)	[M+NH4]+	8.467	736.6446
Set-1	Triacylglycerol	UCD.Lipid.179	TG 44:0	TG (44:0)	TG(14:0_14:0_16:0)	[M+NH4]+	10.13	768.7037
Set-1	Triacylglycerol	UCD.Lipid.180	TG 44:1	TG (44:1)	TG(12:0_14:0_18:1)	[M+NH4]+	9.455	766.6919
Set-1	Triacylglycerol	UCD.Lipid.181	TG 44:2	TG (44:2)		[M+Na]+	9.033	769.6316
Set-1	Triacylglycerol	UCD.Lipid.182	TG 46:0	TG (46:0)	TG(14:0_16:0_16:0)	[M+NH4]+	10.38	796.7389
Set-1	Triacylglycerol	UCD.Lipid.183	TG 46:1	TG (46:1)	TG(12:0_16:0_18:1)	[M+NH4]+	9.944	794.7232
Set-1	Triacylglycerol	UCD.Lipid.184	TG 46:2	TG (46:2)	TG(12:0_16:1_18:1)	[M+Na]+	9.512	797.663
Set-1	Triacylglycerol	UCD.Lipid.185	TG 46:3	TG (46:3)	TG(12:0_16:1_18:2)	[M+Na]+	9.065	795.6473
Set-1	Triacylglycerol	UCD.Lipid.186	TG 46:4 A	TG (46:4) A		[M+Na]+	8.8	793.6322
Set-1	Triacylglycerol	UCD.Lipid.187	TG 48:0	TG (48:0)	TG(16:0_16:0_16:0)	[M+NH4]+	10.81	824.7702
Set-1	Triacylglycerol	UCD.Lipid.188	TG 48:1	TG (48:1)	TG(14:0_16:0_18:1)	[M+NH4]+	10.4	822.7545
Set-1	Triacylglycerol	UCD.Lipid.189	TG 48:2	TG (48:2)	TG(14:0_16:0_18:2)	[M+NH4]+	9.993	820.7389
Set-1	Triacylglycerol	UCD.Lipid.192	TG 49:0	TG (49:0)	TG(16:0_16:0_17:0)	[M+NH4]+	10.94	838.7858
Set-1	Triacylglycerol	UCD.Lipid.193	TG 49:1	TG (49:1)	TG(15:0_16:0_18:1)	[M+NH4]+	10.6	836.7702
Set-1	Triacylglycerol	UCD.Lipid.194	TG 49:2	TG (49:2)	TG(15:0_16:0_18:2)	[M+NH4]+	10.18	834.7545
Set-1	Triacylglycerol	UCD.Lipid.196	TG 50:0	TG (50:0)	TG(16:0_16:0_18:0)	[M+NH4]+	11.21	852.8015
Set-1	Triacylglycerol	UCD.Lipid.197	TG 50:1	TG (50:1)	TG(16:0_16:0_18:1)	[M+NH4]+	10.82	850.7858
Set-1	Triacylglycerol	UCD.Lipid.198	TG 50:2	TG (50:2)	TG(16:0_16:1_18:1)	[M+NH4]+	10.42	848.7702
Set-1	Triacylglycerol	UCD.Lipid.202	TG 50:6	TG (50:6)	TG(14:0_16:1_20:5)	[M+Na]+	9.18	845.6599
Set-1	Triacylglycerol	UCD.Lipid.203	TG 51:1	TG (51:1)	TG(16:0_17:0_18:1)	[M+NH4]+	10.96	864.8015
Set-1	Triacylglycerol	UCD.Lipid.208	TG 52:0	TG (52:0)	TG(16:0_18:0_18:0)	[M+NH4]+	11.58	880.8328
Set-1	Triacylglycerol	UCD.Lipid.209	TG 52:1	TG (52:1)	TG(16:0_18:0_18:1)	[M+NH4]+	11.22	878.8171
Set-1	Triacylglycerol	UCD.Lipid.215	TG 53:1	TG (53:1)	TG(17:0_18:0_18:1)	[M+NH4]+	11.35	892.8328
Set-1	Triacylglycerol	UCD.Lipid.220	TG 54:0	TG (54:0)	TG(18:0_18:0_18:0)	[M+NH4]+	11.92	908.8576
Set-1	Triacylglycerol	UCD.Lipid.221	TG 54:1	TG (54:1)	TG(18:0_18:0_18:1)	[M+NH4]+	11.58	906.8484
Set-1	Triacylglycerol	UCD.Lipid.229	TG 56:2	TG (56:2)	TG(18:1_18:1_20:0)	[M+NH4]+	11.58	932.8641
Set-2	Cholesteroyl ester	UCD.Lipid.10	CE 16:1	CE (16:1)	16:1 Cholesteryl ester	[M+Na]+	10.33	645.5581
Set-2	Lysophosphatidylcholine	UCD.Lipid.119	LPC 14:0	LPC (14:0)	LysoPC 14:0	[M+H]+	0.981	468.3089
Set-2	Phosphatidylcholine	UCD.Lipid.133	PC 28:0	PC (28:0)		[M+H]+	4.239	678.5065

Set -2	Phosphatidylcholine	UCD.Lipid.134	PC 30:0	PC (30:0)	PC(14:0_16:0)	[M+H]+	4.811	706.5378
Set -2	Phosphatidylcholine	UCD.Lipid.135	PC 30:1	PC (30:1)		[M+H]+	4.322	704.5225
Set -2	Phosphatidylcholine	UCD.Lipid.136	PC 31:0	PC (31:0)		[M+H]+	5.11	720.5538
Set -2	Phosphatidylcholine	UCD.Lipid.137	PC 31:1	PC (31:1)		[M+H]+	4.82	718.5345
Set -2	Phosphatidylcholine	UCD.Lipid.138	PC 32:3	PC (32:3)		[M+H]+	4.811	728.5222
Set -2	Phosphatidylcholine	UCD.Lipid.139	PC 33:0	PC (33:0)		[M+H]+	5.64	748.5851
Set -2	Phosphatidylcholine	UCD.Lipid.383	PC 32:1	PC (32:1)	PC(16:0_16:1)	[M+Ac–H]–	5.033	790.56
Set -2	Phosphatidylcholine	UCD.Lipid.384	PC 32:2	PC (32:2)	PC(14:0_18:2)	[M+Ac–H]–	4.567	788.54
Set -2	Phosphatidylcholine	UCD.Lipid.385	PC 33:1	PC (33:1)	PC(15:0_18:1)	[M+Ac–H]–	5.34	804.58
Set -2	Phosphatidylcholine	UCD.Lipid.388	PC 34:1	PC (34:1)	PC(16:0_18:1)	[M+Ac–H]–	5.648	818.59
Set -2	Phosphatidylcholine	UCD.Lipid.390	PC 34:3	PC (34:3)	PC(16:0_18:3)	[M+Ac–H]–	4.75	814.56
Set -2	Phosphatidylcholine	UCD.Lipid.391	PC 34:4	PC (34:4)	PC(14:0_20:4)	[M+Ac–H]–	4.492	812.54
Set -2	Phosphatidylcholine	UCD.Lipid.392	PC 35:1	PC (35:1)	PC(17:0_18:1)	[M+Ac–H]–	5.973	832.61
Set -2	Phosphatidylcholine	UCD.Lipid.395	PC 36:1	PC (36:1)	PC(18:0_18:1)	[M+Ac–H]–	6.314	846.62
Set -2	Phosphatidylethanolamine	UCD.Lipid.431	PE 36:1	PE (36:1)	PE(18:0_18:1)	[M–H]–	5.648	744.55
Set -2	Phosphatidylethanolamine	UCD.Lipid.437	PE 40:6	PE (40:6)	PE(18:0_22:6)	[M–H]–	4.908	790.54
Set -2	Phosphatidylinositol	UCD.Lipid.452	PI 32:1	PI (32:1)	PI(16:0_16:1)	[M–H]–	4.025	807.5
Set -2	Phosphatidylinositol	UCD.Lipid.458	PI 36:4	PI (36:4)	PI(16:0_20:4)	[M–H]–	4.092	857.52
Set -3	Sphingomyelin	UCD.Lipid.169	SM d30:1	SM (d30:1)	SM (d18:1_12:0)	[M+H]+	3.634	647.5123
Set -3	Ceramide	UCD.Lipid.257	Cer d32:1	Ceramide (d32:1)	Cer[NS](d16:1_16:0)	[M+Ac–H]–	5.091	568.49
Set -3	Ceramide	UCD.Lipid.258	Cer d33:1	Ceramide (d33:1)	Cer[NS](d17:1_16:0)	[M+Cl]–	5.415	558.47
Set -3	Ceramide	UCD.Lipid.260	Cer d34:1	Ceramide (d34:1)	Cer[NS](d18:1_16:0)	[M+Ac–H]–	5.748	596.53
Set -3	Ceramide	UCD.Lipid.261	Cer d34:2	Ceramide (d34:2)		[M+Ac–H]–	5.182	594.51
Set -3	Ceramide	UCD.Lipid.262	Cer d36:1	Ceramide (d36:1)	Cer[NS](d18:1_18:0)	[M+Cl]–	6.455	600.51
Set -3	Ceramide	UCD.Lipid.272	Cer d43:1	Ceramide (d43:1)	Cer[NS](d19:1_24:0)	[M+Cl]–	8.682	698.62
Set -3	Sphingomyelin	UCD.Lipid.463	SM d32:0	SM (d32:0)		[M+Ac–H]–	4.467	735.56
Set -3	Sphingomyelin	UCD.Lipid.464	SM d32:1	SM (d32:1)	SM(d18:1_14:0)	[M+Ac–H]–	4.267	733.55
Set -3	Sphingomyelin	UCD.Lipid.465	SM d32:2	SM (d32:2)		[M+Ac–H]–	3.818	731.53
Set -3	Sphingomyelin	UCD.Lipid.466	SM d33:1	SM (d33:1)	SM(d17:1_16:0)	[M+Ac–H]–	4.558	747.57
Set -3	Sphingomyelin	UCD.Lipid.469	SM d36:0	SM (d36:0)		[M+Ac–H]–	5.765	791.63
Set -3	Sphingomyelin	UCD.Lipid.470	SM d36:1	SM (d36:1)	SM(d18:1_18:0)	[M+Ac–H]–	5.523	789.61
Set -3	Sphingomyelin	UCD.Lipid.473	SM d37:1	SM (d37:1)		[M+Ac–H]–	5.873	803.63
Set -3	Sphingomyelin	UCD.Lipid.474	SM d38:0	SM (d38:0)	SM(d18:0_20:0)	[M+Ac–H]–	6.455	819.66
Set -3	Sphingomyelin	UCD.Lipid.475	SM d38:1	SM (d38:1)	SM(d18:1_20:0)	[M+Ac–H]–	6.214	817.64

Set-3	Sphingomyelin	UCD.Lipid.477	SM d39:1	SM (d39:1)	SM(d16:1_23:0)	[M+Ac–H]–	6.55	831.66
Set-3	Sphingomyelin	UCD.Lipid.490	SM d43:1	SM (d43:1)		[M+Ac–H]–	7.77	887.72
Set-3	Sphingomyelin	UCD.Lipid.491	SM d43:2	SM (d43:2)		[M+Ac–H]–	7.079	885.71
Set-4	Lysophosphatidylcholine	UCD.Lipid.126	LPC 20:5	LPC (20:5)	LysoPC 20:5	[M+H]+	0.964	542.3239
Set-4	Lysophosphatidylcholine	UCD.Lipid.128	LPC 22:6	LPC (22:6)	LysoPC 22:6	[M+H]+	1.155	568.3404
Set-4	Phosphatidylcholine	UCD.Lipid.143	PC 36:6	PC (36:6)		[M+H]+	4.231	778.5378
Set-4	Phosphatidylcholine	UCD.Lipid.145	PC 37:6	PC (37:6)		[M+H]+	4.496	792.5538
Set-4	Phosphatidylcholine	UCD.Lipid.148	PC 38:6 A	PC (38:6) A	PC(16:0_22:6	[M+H]+	4.778	806.5691
Set-4	Phosphatidylcholine	UCD.Lipid.149	PC 38:7	PC (38:7)		[M+H]+	4.314	804.5538
Set-4	Phosphatidylcholine	UCD.Lipid.150	PC 39:6	PC (39:6)		[M+H]+	5.027	820.5851
Set-4	Cholesteroyl ester	UCD.Lipid.16	CE 20:5	CE (20:5)	20:5 Cholesteryl ester	[M+Na]+	9.711	693.5581
Set-4	Cholesteroyl ester	UCD.Lipid.17	CE 22:6	CE (22:6)	22:6 Cholesteryl ester	[M+Na]+	9.894	719.5738
Set-4	Lysophosphatidylethanolamine	UCD.Lipid.381	LPE 22:6	LPE (22:6)	LysoPC 22:6	[M–H]–	1.263	524.28
Set-4	Phosphatidylcholine	UCD.Lipid.399	PC 36:5 B	PC (36:5) B	PC(16:1_20:4)	[M+Ac–H]–	4.683	838.56
Set-4	Phosphatidylcholine	UCD.Lipid.406	PC 38:6	PC (38:6)	PC(18:1_20:5)_PC(18:2_20:4)	[M+Ac–H]–	4.908	864.58
Set-4	Phosphatidylcholine	UCD.Lipid.409	PC 40:6 B	PC (40:6) B	PC(18:0_22:6)	[M+Ac–H]–	5.532	892.61
Set-4	Phosphatidylcholine	UCD.Lipid.410	PC 40:7	PC (40:7)	PC(18:1_22:6)	[M+Ac–H]–	4.983	890.59
Set-4	Phosphatidylethanolamine	UCD.Lipid.442	PE p-36:5	PE (p-36:5) or PE (o-36:6)	PE(P-16:0_20:5)	[M–H]–	5.099	720.5
Set-4	Phosphatidylinositol	UCD.Lipid.462	PI 40:6	PI (40:6)	PI(18:0_22:6)	[M–H]–	4.508	909.55
Set-4	Fatty acid	UCD.Lipid.513	FA 20:5	FA (20:5)	FA (20:5)	[M–H]–	1.53	301.22
Set-4	Fatty acid	UCD.Lipid.517	FA 22:6	FA (22:6)	FA (22:6)	[M–H]–	1.72	327.23
Set-5	Diacylglycerol	UCD.Lipid.104	DG 32:1	DG (32:1)	DG(16:0_16:1)	[M+Na]+	6.143	589.4812
Set-5	Diacylglycerol	UCD.Lipid.105	DG 34:1	DG (34:1)	DG(16:0_18:1)	[M+Na]+	6.892	617.5116
Set-5	Diacylglycerol	UCD.Lipid.106	DG 34:2	DG (34:2)	DG(16:0_18:2)	[M+Na]+	6.27	615.4965
Set-5	Diacylglycerol	UCD.Lipid.107	DG 34:3	DG (34:3)	DG(16:1_18:2)	[M+NH4]+	5.686	608.5246
Set-5	Diacylglycerol	UCD.Lipid.109	DG 36:2	DG (36:2)	DG(18:1_18:1)	[M+NH4]+	6.95	638.5718
Set-5	Diacylglycerol	UCD.Lipid.110	DG 36:3	DG (36:3)	DG(18:1_18:2)	[M+NH4]+	6.42	636.5562
Set-5	Triacylglycerol	UCD.Lipid.190	TG 48:3	TG (48:3)	TG(12:0_18:1_18:2)	[M+Na]+	9.546	823.6789
Set-5	Triacylglycerol	UCD.Lipid.191	TG 48:4	TG (48:4)	TG(12:0_18:2_18:2)	[M+Na]+	9.34	821.6597
Set-5	Triacylglycerol	UCD.Lipid.195	TG 49:3	TG (49:3)	TG(15:0_16:1_18:2)	[M+NH4]+	9.794	832.7389
Set-5	Triacylglycerol	UCD.Lipid.199	TG 50:3	TG (50:3)	TG(16:1_16:1_18:1)	[M+NH4]+	10.02	846.7545
Set-5	Triacylglycerol	UCD.Lipid.200	TG 50:4	TG (50:4)	TG(16:1_16:1_18:2)	[M+Na]+	9.629	849.6943
Set-5	Triacylglycerol	UCD.Lipid.201	TG 50:5	TG (50:5)	TG(14:0_18:2_18:3)	[M+Na]+	9.24	847.6786
Set-5	Triacylglycerol	UCD.Lipid.204	TG 51:2	TG (51:2)	TG(16:0_17:0_18:2)	[M+NH4]+	10.62	862.7858

Set-5	Triacylglycerol	UCD.Lipid.205	TG 51:3	TG (51:3)	TG(15:0_18:1_18:2)	[M+NH4]+	10.25	860.7702
Set-5	Triacylglycerol	UCD.Lipid.210	TG 52:2	TG (52:2)	TG(16:0_18:1_18:1)	[M+NH4]+	10.83	876.8015
Set-5	Triacylglycerol	UCD.Lipid.211	TG 52:3	TG (52:3)	TG(16:0_18:1_18:2)	[M+NH4]+	10.47	874.7858
Set-5	Triacylglycerol	UCD.Lipid.216	TG 53:2	TG (53:2)	TG(17:0_18:1_18:1)	[M+NH4]+	11.01	890.8171
Set-5	Triacylglycerol	UCD.Lipid.217	TG 53:3	TG (53:3)	TG(17:0_18:1_18:2)	[M+NH4]+	10.65	888.8015
Set-6	Diacylglycerol	UCD.Lipid.108	DG 36:1	DG (36:1)	DG(18:0_18:1)	[M+NH4]+	7.47	640.5588
Set-6	Diacylglycerol	UCD.Lipid.113	DG 38:0	DG (38:0)		[M+NH4]+	7.94	670.6086
Set-6	Diacylglycerol	UCD.Lipid.114	DG 38:3	DG (38:3)	DG(20:1_18:2)	[M+Na]+	6.974	669.5653
Set-6	Diacylglycerol	UCD.Lipid.116	DG 38:6	DG (38:6)	DG(18:2_20:4)	[M+Na]+	5.732	663.4959
Set-6	Lysophosphatidylcholine	UCD.Lipid.127	LPC 22:4	LPC (22:4)	LysoPC 22:4	[M+H]+	1.661	572.3711
Set-6	Lysophosphatidylcholine	UCD.Lipid.129	LPC o-16:0	LPC (o-16:0)	LPC (o-16:0)	[M+H]+	1.752	482.3606
Set-6	Lysophosphatidylcholine	UCD.Lipid.130	LPC p-16:0	LPC (p-16:0) or LPC (o-16:1)	LPC (o-16:1)	[M+H]+	1.694	480.3448
Set-6	Lysophosphatidylcholine	UCD.Lipid.131	LPC p-18:0	LPC (p-18:0) or LPC (o-18:1)	LPC (o-18:1)	[M+H]+	1.909	508.3762
Set-6	Triacylglycerol	UCD.Lipid.175	TG 40:1	TG (40:1)		[M+NH4]+	8.384	710.6298
Set-6	Triacylglycerol	UCD.Lipid.178	TG 42:3	TG (42:3)		[M+NH4]+	8.24	734.6265
Set-6	Ceramide	UCD.Lipid.19	Cer d42:2	Ceramide (d42:2)	Cer[NS](d18:2_24:0)	[M+H]+	7.615	648.6289
Set-6	Triacylglycerol	UCD.Lipid.253	TG 60:6	TG (60:6)		[M+Na]+	11.27	985.8105
Set-6	Ceramide	UCD.Lipid.259	Cer d34:0	Ceramide (d34:0)	Cer[NDS](d18:0_16:0)	[M+Ac–H]–	5.998	598.54
Set-6	Phosphatidylethanolamine	UCD.Lipid.439	PE p-36:1	PE (p-36:1) or PE (o-36:2)	PE(P-18:0_18:1)	[M–H]–	6.838	728.56
Set-6	Fatty acid	UCD.Lipid.511	FA 20:3	FA (20:3)	FA (20:3)	[M–H]–	2.5	305.25
Set-6	Fatty acid	UCD.Lipid.512	FA 20:4	FA (20:4)	FA (20:4)	[M–H]–	1.93	303.23
Set-6	Fatty acid	UCD.Lipid.515	FA 22:1	FA (22:1)	FA (22:1)	[M–H]–	3.9	337.31
Set-6	Acylcarnitine	UCD.Lipid.9	AC C18:3	Acylcarnitine C18:3	Acylcarnitine 18:3	[M+H]+	1.13	422.3265
Set-7	Phosphatidylcholine	UCD.Lipid.142	PC 36:4 A	PC (36:4) A	PC(18:2_18:2)	[M+H]+	4.637	782.5694
Set-7	Glucosylceramide	UCD.Lipid.368	GlcCerd 24:1-2OH	GlcCer(d14:1(4E)/20:0(2OH))		[M+Cl]–	4.966	750.53
Set-7	Phosphatidylcholine	UCD.Lipid.382	PC 32:0	PC (32:0)	PC(16:0_16:0)	[M+Ac–H]–	5.573	792.58
Set-7	Phosphatidylcholine	UCD.Lipid.386	PC 33:2	PC (33:2)	PC(16:0_18:2)	[M+Ac–H]–	4.858	802.56
Set-7	Phosphatidylcholine	UCD.Lipid.387	PC 34:0	PC (34:0)	PC(16:0_18:0)	[M+Ac–H]–	6.239	820.61
Set-7	Phosphatidylcholine	UCD.Lipid.389	PC 34:2	PC (34:2)	PC(16:0_18:2)	[M+Ac–H]–	5.166	816.58
Set-7	Phosphatidylcholine	UCD.Lipid.393	PC 35:2	PC (35:2)	PC(17:0_18:2)	[M+Ac–H]–	5.48	830.59
Set-7	Phosphatidylcholine	UCD.Lipid.396	PC 36:2	PC (36:2)	PC(18:0_18:2)	[M+Ac–H]–	5.806	844.61
Set-7	Phosphatidylcholine	UCD.Lipid.400	PC 37:2	PC (37:2)	PC(19:0_18:2)	[M+Ac–H]–	6.14	858.62
Set-7	Phosphatidylethanolamine	UCD.Lipid.434	PE 38:2	PE (38:2)	PE(20:0_18:2)	[M–H]–	5.806	770.57
Set-7	Phosphatidylethanolamine	UCD.Lipid.443	PE p-38:2	PE (p-38:2) or PE (o-38:3)		[M–H]–	6.98	754.58

Set-7	Phosphatidylethanolamine	UCD.Lipid.448	PE p-40:4	PE (p-40:4) or PE (o-40:5)	PE(P-20:0_20:4)	[M–H]–	6.87	778.58
Set-7	Phosphatidylinositol	UCD.Lipid.453	PI 34:1	PI (34:1)	PI(16:0_18:1)	[M–H]–	4.483	835.53
Set-7	Phosphatidylinositol	UCD.Lipid.454	PI 34:2	PI (34:2)	PI(16:0_18:2)	[M–H]–	4.158	833.52
Set-7	Phosphatidylinositol	UCD.Lipid.455	PI 36:1	PI (36:1)	PI(18:0_18:1)	[M–H]–	5.198	863.57
Set-7	Phosphatidylinositol	UCD.Lipid.456	PI 36:2	PI (36:2)	PI(18:0_18:2)	[M–H]–	4.732	861.55
Set-7	Phosphatidylinositol	UCD.Lipid.457	PI 36:3	PI (36:3)	PI(16:0_20:3)	[M–H]–	4.225	859.53
Set-8	Lysophosphatidylcholine	UCD.Lipid.120	LPC 15:0	LPC (15:0)	LysoPC 15:0	[M+H]+	1.206	482.3241
Set-8	Lysophosphatidylcholine	UCD.Lipid.121	LPC 17:1	LPC (17:1)	LysoPC 17:1	[M+H]+	1.345	508.3401
Set-8	Lysophosphatidylcholine	UCD.Lipid.122	LPC 18:0	LPC (18:0)	LysoPC 18:0	[M+H]+	2.258	524.3708
Set-8	Lysophosphatidylcholine	UCD.Lipid.123	LPC 18:3	LPC (18:3)	LysoPC 18:3	[M+H]+	0.981	518.3241
Set-8	Lysophosphatidylcholine	UCD.Lipid.124	LPC 20:0	LPC (20:0)	LysoPC 20:0	[M+H]+	3.062	552.4021
Set-8	Lysophosphatidylcholine	UCD.Lipid.369	LPC 16:0	LPC (16:0)	LysoPC 16:0	[M+Ac–H]–	1.563	554.35
Set-8	Lysophosphatidylcholine	UCD.Lipid.370	LPC 16:1	LPC (16:1)	LysoPC 16:1	[M+Ac–H]–	1.18	552.33
Set-8	Lysophosphatidylcholine	UCD.Lipid.371	LPC 18:0 A	LPC (18:0) A	LysoPC 18:0	[M+Ac–H]–	2.145	582.38
Set-8	Lysophosphatidylcholine	UCD.Lipid.372	LPC 18:1	LPC (18:1)	LysoPC 18:1	[M+Ac–H]–	1.713	580.36
Set-8	Lysophosphatidylcholine	UCD.Lipid.373	LPC 18:2	LPC (18:2)	LysoPC 18:2	[M+Ac–H]–	1.313	578.35
Set-8	Lysophosphatidylcholine	UCD.Lipid.374	LPC 20:1	LPC (20:1)	LysoPC 20:1	[M+Ac–H]–	2.47	608.39
Set-8	Lysophosphatidylcholine	UCD.Lipid.375	LPC 20:2	LPC (20:2)	LysoPC 20:2	[M+Ac–H]–	1.896	606.38
Set-8	Lysophosphatidylcholine	UCD.Lipid.376	LPC 20:3	LPC (20:3)	LysoPC 20:3	[M+Ac–H]–	1.505	604.36
Set-8	Lysophosphatidylcholine	UCD.Lipid.377	LPC 22:5	LPC (22:5)	LysoPC 22:5	[M+Ac–H]–	1.388	628.36
Set-8	Lysophosphatidylethanolamine	UCD.Lipid.378	LPE 16:0	LPE (16:0)	LysoPE 16:0	[M–H]–	1.596	452.28
Set-8	Lysophosphatidylethanolamine	UCD.Lipid.379	LPE 18:2	LPE (18:2)	LysoPE 18:2	[M–H]–	1.347	476.28
Set-9	Phosphatidylcholine	UCD.Lipid.132	PC 27:0-CHO	PC (16:0/9:0(CHO))	PC (16:0_9:0(CHO))	[M+H]+	2.374	650.4391
Set-9	Triacylglycerol	UCD.Lipid.176	TG 42:1	TG (42:1)	TG(12:0_12:0_18:1)	[M+Na]+	8.967	743.6162
Set-9	Fatty acid	UCD.Lipid.493	FA 11:0	FA (11:0)	FA (11:0)	[M–H]–	0.82	185.15
Set-9	Fatty acid	UCD.Lipid.494	FA 12:0	FA (12:0)	FA (12:0)	[M–H]–	1.06	199.17
Set-9	Fatty acid	UCD.Lipid.495	FA 13:0	FA (13:0)	FA (13:0)	[M–H]–	1.3	213.19
Set-9	Fatty acid	UCD.Lipid.496	FA 14:0	FA (14:0)	FA (14:0)	[M–H]–	1.63	227.2
Set-9	Fatty acid	UCD.Lipid.498	FA 15:0	FA (15:0)	FA (15:0)	[M–H]–	2.02	241.22
Set-9	Fatty acid	UCD.Lipid.499	FA 15:1	FA (15:1)	FA (15:1)	[M–H]–	1.5	239.2
Set-9	Fatty acid	UCD.Lipid.500	FA 16:0	FA (16:0)	FA (16:0)	[M–H]–	2.47	255.23
Set-9	Fatty acid	UCD.Lipid.502	FA 17:0	FA (17:0)	FA (17:0)	[M–H]–	2.96	269.25
Set-9	Fatty acid	UCD.Lipid.504	FA 18:0	FA (18:0)	FA (18:0)	[M–H]–	3.26	283.26
Set-9	Fatty acid	UCD.Lipid.508	FA 20:0	FA (20:0)	FA (20:0)	[M–H]–	3.85	311.3

Set -9	Fatty acid	UCD.Lipid.514	FA 22:0	FA (22:0)	FA (22:0)	[M–H]–	4.5	339.33
Set -9	Fatty acid	UCD.Lipid.518	FA 24:0	FA (24:0)	FA (24:0)	[M–H]–	5.23	367.36
Set -9	Fatty acid	UCD.Lipid.520	FA 26:0	FA (26:0)	FA (26:0)	[M–H]–	6.02	395.39
Set -9	Fatty acid	UCD.Lipid.521	FA 28:0	FA (28:0)	FA (28:0)	[M–H]–	6.82	423.42
Set -10	Sphingomyelin	UCD.Lipid.171	SM d40:2	SM (d40:2)		[M+H]+	6.038	785.6531
Set -10	Phosphatidylinositol	UCD.Lipid.461	PI 38:5	PI (38:5)	PI(18:1_20:4)	[M–H]–	4.142	883.53
Set -10	Sphingomyelin	UCD.Lipid.468	SM d34:2	SM (d34:2)	SM(d18:2_16:0)	[M+Ac–H]–	4.359	759.57
Set -10	Sphingomyelin	UCD.Lipid.471	SM d36:2	SM (d36:2)		[M+Ac–H]–	4.974	787.6
Set -10	Sphingomyelin	UCD.Lipid.472	SM d36:3	SM (d36:3)		[M+Ac–H]–	4.508	785.58
Set -10	Sphingomyelin	UCD.Lipid.476	SM d38:2	SM (d38:2)		[M+Ac–H]–	5.623	815.63
Set -10	Sphingomyelin	UCD.Lipid.478	SM d39:2	SM (d39:2)		[M+Ac–H]–	5.988	829.64
Set -10	Sphingomyelin	UCD.Lipid.479	SM d40:0	SM (d40:0)		[M+Ac–H]–	7.138	847.69
Set -10	Sphingomyelin	UCD.Lipid.480	SM d40:1	SM (d40:1)	SM(d18:1_22:0)	[M+Ac–H]–	6.888	845.67
Set -10	Sphingomyelin	UCD.Lipid.481	SM d40:2 A	SM (d40:2) A		[M+Ac–H]–	6.205	843.66
Set -10	Sphingomyelin	UCD.Lipid.482	SM d40:2 B	SM (d40:2) B	SM(d18:2_22:0)	[M+Ac–H]–	6.306	843.66
Set -10	Sphingomyelin	UCD.Lipid.483	SM d40:3	SM (d40:3)		[M+Ac–H]–	5.64	841.64
Set -10	Sphingomyelin	UCD.Lipid.484	SM d41:1	SM (d41:1)	SM(d18:1_23:0)	[M+Ac–H]–	7.229	859.69
Set -10	Sphingomyelin	UCD.Lipid.485	SM d41:2	SM (d41:2)	SM(d18:2_23:0)	[M+Ac–H]–	6.65	857.67
Set -10	Sphingomyelin	UCD.Lipid.486	SM d42:0	SM (d42:0)		[M+Ac–H]–	7.794	875.72
Set -10	Sphingomyelin	UCD.Lipid.487	SM d42:1	SM (d42:1)	SM(d18:1_24:0)	[M+Ac–H]–	7.562	873.71
Set -11	Glactoylceramide	UCD.Lipid.117	LacCer d34:1	Gal-Gal-Cer(d18:1/16:0) or Lactosylceramide(d18:1/16:0)		[M+H]+	4.795	862.625
Set -11	Lactosylceramide	UCD.Lipid.118	LacCer d32:2	Lactosylceramide (d18:1/24:1(15Z))		[M+H]+	6.685	972.7346
Set -11	Cholesteroyl ester	UCD.Lipid.12	CE 18:2	CE (18:2)	18:2 Cholesteryl ester	[M+NH4]+	10.4	666.6184
Set -11	Phosphatidylcholine	UCD.Lipid.155	PC o-34:0	PC (o-34:0)		[M+H]+	6.478	748.6204
Set -11	Phosphatidylcholine	UCD.Lipid.156	PC p-32:1	PC (p-32:1) or PC (o-32:2)		[M+H]+	4.91	716.5626
Set -11	Phosphatidylcholine	UCD.Lipid.157	PC p-34:1	PC (p-34:1) or PC (o-34:2) B	PC(P-16:0_18:1)	[M+H]+	5.79	744.5908
Set -11	Phosphatidylcholine	UCD.Lipid.160	PC p-40:1	PC (p-40:1) or PC (o-40:2)		[M+H]+	6.637	828.6842
Set -11	Sphingomyelin	UCD.Lipid.170	SM d34:0	SM (d34:0)		[M+H]+	4.96	705.5905
Set -11	Glucosylceramide	UCD.Lipid.363	GlcCer d38:1	GlcCer (d38:1)		[M+Ac–H]–	6.489	814.64
Set -11	Glucosylceramide	UCD.Lipid.364	GlcCer d40:1	GlcCer (d40:1)	GlcCer[NS](d18:1_22:0)	[M+Ac–H]–	7.163	842.67
Set -11	Glucosylceramide	UCD.Lipid.365	GlcCer d41:1	GlcCer (d41:1)		[M+Ac–H]–	7.495	856.69
Set -11	Glucosylceramide	UCD.Lipid.366	GlcCer d42:1	GlcCer (d42:1)	GlcCer[NS](d18:1_24:0)	[M+Ac–H]–	7.82	870.7
Set -11	Glucosylceramide	UCD.Lipid.367	GlcCer d42:2	GlcCer (d42:2)	GlcCer[NS](d18:1_24:1)	[M+Ac–H]–	7.138	868.69

Set-11	Phosphatidylcholine	UCD.Lipid.412	PC o-32:0	PC (o-32:0)		[M+Ac–H]–	5.973	778.59
Set-11	Phosphatidylcholine	UCD.Lipid.414	PC p-34:0	PC (p-34:0) or PC (o-34:1)	PC(P-18:0_16:0)	[M+Ac–H]–	6.039	804.61
Set-11	Sphingomyelin	UCD.Lipid.467	SM d34:1	SM (d34:1)	SM(d18:1_16:0)	[M+Ac–H]–	4.866	761.58
Set-12	Diacylglycerol	UCD.Lipid.111	DG 36:4	DG (36:4)	DG(18:2_18:2)	[M+NH4]+	5.815	634.5404
Set-12	Diacylglycerol	UCD.Lipid.112	DG 36:5	DG (36:5)	DG(18:2_18:3)	[M+NH4]+	5.65	632.5248
Set-12	Triacylglycerol	UCD.Lipid.206	TG 51:4	TG (51:4)	TG(15:0_18:2_18:2)	[M+Na]+	9.877	863.7099
Set-12	Triacylglycerol	UCD.Lipid.207	TG 51:5	TG (51:5)	TG(15:0_18:2_18:3)	[M+NH4]+	9.77	856.7347
Set-12	Triacylglycerol	UCD.Lipid.212	TG 52:4	TG (52:4)	TG(16:1_18:1_18:2)	[M+NH4]+	10.1	872.7702
Set-12	Triacylglycerol	UCD.Lipid.213	TG 52:5	TG (52:5)	TG(16:0_18:2_18:3)	[M+NH4]+	9.728	870.7545
Set-12	Triacylglycerol	UCD.Lipid.214	TG 52:6	TG (52:6)	TG(16:1_18:2_18:3)	[M+Na]+	9.35	873.6943
Set-12	Triacylglycerol	UCD.Lipid.218	TG 53:4	TG (53:4)	TG(17:1_18:1_18:2)	[M+NH4]+	10.32	886.7858
Set-12	Triacylglycerol	UCD.Lipid.219	TG 53:5	TG (53:5)	TG(17:1_18:2_18:2)	[M+NH4]+	9.902	884.7702
Set-12	Triacylglycerol	UCD.Lipid.223	TG 54:3	TG (54:3)	TG(18:0_18:1_18:2)	[M+NH4]+	10.86	902.8171
Set-12	Triacylglycerol	UCD.Lipid.224	TG 54:4	TG (54:4)	TG(18:1_18:1_18:2)	[M+NH4]+	10.49	900.8015
Set-12	Triacylglycerol	UCD.Lipid.225	TG 54:5	TG (54:5)	TG(18:1_18:2_18:2)	[M+Na]+	10.12	903.7412
Set-12	Triacylglycerol	UCD.Lipid.226	TG 54:6	TG (54:6)	TG(18:1_18:2_18:3)	[M+Na]+	9.745	901.7256
Set-12	Triacylglycerol	UCD.Lipid.227	TG 54:8	TG (54:8)	TG(18:2_18:3_18:3)	[M+Na]+	8.94	897.6943
Set-13	Triacylglycerol	UCD.Lipid.228	TG 56:1	TG (56:1)	TG(18:0_18:1_20:0)	[M+NH4]+	11.85	934.8797
Set-13	Triacylglycerol	UCD.Lipid.237	TG 57:1	TG (57:1)		[M+NH4]+	12	948.8863
Set-13	Triacylglycerol	UCD.Lipid.238	TG 57:2	TG (57:2)		[M+NH4]+	11.88	946.8718
Set-13	Triacylglycerol	UCD.Lipid.239	TG 58:1	TG (58:1)	TG(18:0_18:1_22:0)	[M+NH4]+	11.99	962.911
Set-13	Triacylglycerol	UCD.Lipid.241	TG 58:2	TG (58:2)	TG(18:1_18:1_22:0)	[M+NH4]+	11.85	960.8954
Set-13	Triacylglycerol	UCD.Lipid.242	TG 58:3	TG (58:3)	TG(18:1_20:1_20:1)	[M+NH4]+	11.6	958.8797
Set-13	Triacylglycerol	UCD.Lipid.247	TG 59:2	TG (59:2)		[M+NH4]+	12	974.9031
Set-13	Triacylglycerol	UCD.Lipid.248	TG 59:3	TG (59:3)		[M+NH4]+	11.89	972.8871
Set-13	Triacylglycerol	UCD.Lipid.250	TG 60:2	TG (60:2)	TG(18:1_20:1_22:0)	[M+NH4]+	11.99	988.9267
Set-13	Triacylglycerol	UCD.Lipid.251	TG 60:3	TG (60:3)	TG(18:1_20:1_22:1)	[M+NH4]+	11.95	986.9034
Set-13	Triacylglycerol	UCD.Lipid.252	TG 60:4	TG (60:4)	TG(18:1_20:2_22:1)	[M+NH4]+	11.78	984.8877
Set-13	Triacylglycerol	UCD.Lipid.254	TG 62:3	TG (62:3)		[M+NH4]+	12.04	1014.934
Set-13	Triacylglycerol	UCD.Lipid.255	TG 62:4	TG (62:4)		[M+NH4]+	11.94	1012.918
Set-13	Triacylglycerol	UCD.Lipid.256	TG 64:4	TG (64:4)		[M+NH4]+	12.04	1040.949
Set-14	Ceramide	UCD.Lipid.18	Cer d41:1	Ceramide (d18:1/23:0)	Cer[NS](d18:1_23:0)	[M+H]+	7.963	636.6289
Set-14	Ceramide	UCD.Lipid.263	Cer d38:1	Ceramide (d38:1)	Cer[NS](d18:1_20:0)	[M+Cl]–	7.163	628.54
Set-14	Ceramide	UCD.Lipid.264	Cer d39:1	Ceramide (d39:1)	Cer[NS](d18:1_22:0)	[M+Cl]–	7.512	642.56

Set-14	Ceramide	UCD.Lipid.265	Cer d40:0	Ceramide (d40:0)	Cer[NDS](d18:0_22:0)	[M+Ac–H]–	8.061	682.63
Set-14	Ceramide	UCD.Lipid.266	Cer d40:1	Ceramide (d40:1)	Cer[NS](d18:1_22:0)	[M+Cl]–	7.836	656.58
Set-14	Ceramide	UCD.Lipid.267	Cer d40:2	Ceramide (d40:2)	Cer[NS](d18:2_22:0)	[M+Cl]–	7.185	654.56
Set-14	Ceramide	UCD.Lipid.268	Cer d41:1	Ceramide (d41:1)	Cer[NS](d18:1_23:0)	[M+Cl]–	8.161	670.59
Set-14	Ceramide	UCD.Lipid.269	Cer d42:0	Ceramide (d42:0)	Cer[NDS](d18:0_24:0)	[M+Cl]–	8.685	686.62
Set-14	Ceramide	UCD.Lipid.270	Cer d42:1	Ceramide (d42:1)	Cer[NS](d18:1_24:0)	[M+Ac–H]–	8.469	708.65
Set-14	Ceramide	UCD.Lipid.271	Cer d42:2 A	Ceramide (d42:2) A	Cer[NS](d18:1_24:1)	[M+Cl]–	NA	NA
Set-14	Ceramide	UCD.Lipid.273	Cer d44:1	Ceramide (d44:1)		[M+Cl]–	9.068	712.64
Set-15	Phosphatidylcholine	UCD.Lipid.159	PC p-38:4	PC (p-38:4) or PC (o-38:5) B	PC(P-18:0_20:4)	[M+H]+	5.823	794.6054
Set-15	Phosphatidylethanolamine	UCD.Lipid.167	PE 38:4	PE (38:4)	PE(18:0_20:4)	[M+H]+	5.723	768.556
Set-15	Phosphatidylcholine	UCD.Lipid.413	PC p-32:0	PC (p-32:0) or PC (o-32:1)	PC(P-16:0_16:0)	[M+Ac–H]–	5.881	776.58
Set-15	Phosphatidylcholine	UCD.Lipid.415	PC p-34:1	PC (p-34:1) or PC (o-34:2) A	PC(P-16:0_18:1)	[M+Ac–H]–	5.54	802.6
Set-15	Phosphatidylcholine	UCD.Lipid.416	PC p-34:2	PC (p-34:2) or PC (o-34:3)	PC(P-16:0_18:2)	[M+Ac–H]–	5.44	800.58
Set-15	Phosphatidylcholine	UCD.Lipid.417	PC p-36:1	PC (p-36:1) or PC (o-36:2)	PC(P-18:0_18:1)	[M+Ac–H]–	6.2	830.63
Set-15	Phosphatidylcholine	UCD.Lipid.418	PC p-36:2	PC (p-36:2) or PC (o-36:3)	PC(P-18:0_18:2)	[M+Ac–H]–	5.59	828.61
Set-15	Phosphatidylcholine	UCD.Lipid.419	PC p-36:3	PC (p-36:3) or PC (o-36:4)	PC(P-16:0_20:3)	[M+Ac–H]–	5.44	826.6
Set-15	Phosphatidylcholine	UCD.Lipid.420	PC p-36:4	PC (p-36:4) or PC (o-36:5)	PC(P-16:0_20:4)	[M+Ac–H]–	5.332	824.58
Set-15	Phosphatidylcholine	UCD.Lipid.422	PC p-38:4	PC (p-38:4) or PC (o-38:5) A	PC(P-18:0_20:4)	[M+Ac–H]–	5.482	852.61
Set-15	Phosphatidylcholine	UCD.Lipid.423	PC p-38:5	PC (p-38:5) or PC (o-38:6)	PC(P-18:0_20:5)	[M+Ac–H]–	5.257	850.6
Set-16	Acylcarnitine	UCD.Lipid.1	AC C8:0	Acylcarnitine C8:0	Acylcarnitine 8:0	[M+H]+	0.55	288.2169
Set-16	Acylcarnitine	UCD.Lipid.2	AC C8:1	Acylcarnitine C8:1	Acylcarnitine 8:1	[M+H]+	0.52	286.2013
Set-16	Cholesterol	UCD.Lipid.20	Cholesterol	Cholesterol		[M–H2O+H]+	4.861	369.3516
Set-16	Acylcarnitine	UCD.Lipid.3	AC C10:1	Acylcarnitine C10:1	Acylcarnitine 10:1	[M+H]+	0.59	314.2326
Set-16	Acylcarnitine	UCD.Lipid.4	AC C12:0	Acylcarnitine C12:0	Acylcarnitine 12:0	[M+H]+	0.74	344.2801
Set-16	Acylcarnitine	UCD.Lipid.5	AC C16:0	Acylcarnitine C16:0	Acylcarnitine 16:0	[M+H]+	1.586	400.342
Set-16	Fatty acid	UCD.Lipid.519	FA 24:1	FA (24:1)	FA (24:1)	[M–H]–	4.52	365.34
Set-16	Acylcarnitine	UCD.Lipid.6	AC C18:0	Acylcarnitine C18:0	Acylcarnitine 18:0	[M+H]+	2.34	428.3727
Set-16	Acylcarnitine	UCD.Lipid.7	AC C18:1	Acylcarnitine C18:1	Acylcarnitine 18:1	[M+H]+	1.743	426.3578
Set-16	Acylcarnitine	UCD.Lipid.8	AC C18:2	Acylcarnitine C18:2	Acylcarnitine 18:2	[M+H]+	1.329	424.3425
Set-17	Fatty acid	UCD.Lipid.497	FA 14:1	FA (14:1)	FA (14:1)	[M–H]–	1.2	225.19
Set-17	Fatty acid	UCD.Lipid.501	FA 16:1	FA (16:1)	FA (16:1)	[M–H]–	1.82	253.22
Set-17	Fatty acid	UCD.Lipid.503	FA 17:1	FA (17:1)	FA (17:1)	[M–H]–	2.22	267.23
Set-17	Fatty acid	UCD.Lipid.505	FA 18:1	FA (18:1)	FA (18:1)	[M–H]–	2.7	281.25

Set-17	Fatty acid	UCD.Lipid.506	FA 18:2	FA (18:2)	FA (18:2)	[M–H]–	2.05	279.23
Set-17	Fatty acid	UCD.Lipid.507	FA 18:3	FA (18:3)	FA (18:3)	[M–H]–	1.623	277.22
Set-17	Fatty acid	UCD.Lipid.509	FA 20:1	FA (20:1)	FA (20:1)	[M–H]–	3.35	309.28
Set-17	Fatty acid	UCD.Lipid.510	FA 20:2	FA (20:2)	FA (20:2)	[M–H]–	2.91	307.26
Set-17	Fatty acid	UCD.Lipid.516	FA 22:2	FA (22:2)	FA (22:2)	[M–H]–	3.47	335.3
Set-18	Diacylglycerol	UCD.Lipid.115	DG 38:5	DG (38:5)	DG(18:1_20:4)	[M+NH4]+	6.246	660.5562
Set-18	Triacylglycerol	UCD.Lipid.222	TG 54:2	TG (54:2)	TG(18:0_18:1_18:1)	[M+NH4]+	11.22	904.8328
Set-18	Triacylglycerol	UCD.Lipid.230	TG 56:3	TG (56:3)	TG(18:0_18:1_20:2)	[M+NH4]+	11.22	930.8484
Set-18	Triacylglycerol	UCD.Lipid.231	TG 56:4	TG (56:4)	TG(18:0_18:1_20:3)	[M+NH4]+	10.9	928.8328
Set-18	Triacylglycerol	UCD.Lipid.232	TG 56:5	TG (56:5)	TG(18:0_18:1_20:4)	[M+NH4]+	10.78	926.8171
Set-18	Triacylglycerol	UCD.Lipid.233	TG 56:6	TG (56:6)	TG(18:1_18:1_20:4)	[M+NH4]+	10.34	924.8015
Set-18	Triacylglycerol	UCD.Lipid.234	TG 56:7	TG (56:7)	TG(18:1_18:2_20:4)	[M+NH4]+	9.968	922.7858
Set-18	Triacylglycerol	UCD.Lipid.243	TG 58:4	TG (58:4)	TG(18:1_18:2_22:1)	[M+NH4]+	11.25	956.8641
Set-18	Triacylglycerol	UCD.Lipid.244	TG 58:6	TG (58:6)	TG(18:0_18:1_22:5)	[M+NH4]+	10.67	952.8328
Set-19	Phosphatidylcholine	UCD.Lipid.147	PC 38:5 B	PC (38:5) B	PC(16:0_22:5)	[M+H]+	5.168	808.5848
Set-19	Phosphatidylcholine	UCD.Lipid.151	PC 40:5 B	PC (40:5) B	PC(18:0_22:5)	[M+H]+	5.806	836.6164
Set-19	Phosphatidylcholine	UCD.Lipid.152	PC 40:6 A	PC (40:6) A	PC(18:0_22:6)	[M+H]+	5.151	834.6007
Set-19	Phosphatidylcholine	UCD.Lipid.153	PC 42:5	PC (42:5)		[M+H]+	6.072	864.645
Set-19	Phosphatidylcholine	UCD.Lipid.154	PC 42:6	PC (42:6)		[M+H]+	5.773	862.6311
Set-19	Phosphatidylcholine	UCD.Lipid.405	PC 38:5 A	PC (38:5) A	PC(18:1_20:4)	[M+Ac–H]–	5.107	866.59
Set-19	Phosphatidylcholine	UCD.Lipid.408	PC 40:5 A	PC (40:5) A	PC(18:0_22:5)	[M+Ac–H]–	5.74	894.62
Set-19	Phosphatidylcholine	UCD.Lipid.411	PC 40:8	PC (40:8)	PC(18:2_22:6)	[M+Ac–H]–	4.533	888.58
Set-19	Phosphatidylinositol	UCD.Lipid.460	PI 38:4	PI (38:4)	PI(18:0_20:4)	[M–H]–	4.606	885.55
Set-20	Phosphatidylcholine	UCD.Lipid.158	PC p-38:2	PC (p-38:2) or PC (o-38:3)		[M+H]+	6.212	798.6359
Set-20	Phosphatidylcholine	UCD.Lipid.165	PC p-42:3	PC (p-42:3) or PC (o-42:4)		[M+H]+	7.224	852.6859
Set-20	Phosphatidylcholine	UCD.Lipid.421	PC p-38:3	PC (p-38:3) or PC (o-38:4)	PC(P-18:0_20:3)	[M+Ac–H]–	6.098	854.63
Set-20	Phosphatidylcholine	UCD.Lipid.424	PC p-40:3	PC (p-40:3) or PC (o-40:4)		[M+Ac–H]–	6.763	882.66
Set-20	Phosphatidylcholine	UCD.Lipid.425	PC p-40:4	PC (p-40:4) or PC (o-40:5)	PC(P-20:0_20:4)	[M+Ac–H]–	6.106	880.64
Set-20	Phosphatidylcholine	UCD.Lipid.426	PC p-42:4	PC (p-42:4) or PC (o-42:5)	PC(P-22:0_20:4)	[M+Ac–H]–	6.747	908.67
Set-20	Phosphatidylcholine	UCD.Lipid.427	PC p-42:5	PC (p-42:5) or PC (o-42:6)		[M+Ac–H]–	6.188	906.66
Set-20	Phosphatidylcholine	UCD.Lipid.428	PC p-44:4	PC (p-44:4) or PC (o-44:5)		[M+Ac–H]–	7.387	936.71
Set-21	Lysophosphatidylcholine	UCD.Lipid.125	LPC 20:4	LPC (20:4)	LysoPC 20:4	[M+H]+	1.205	544.3403
Set-21	Phosphatidylcholine	UCD.Lipid.146	PC 38:4 B	PC (38:4) B		[M+H]+	6.146	810.6004
Set-21	Lysophosphatidylethanolamine	UCD.Lipid.380	LPE 20:4	LPE (20:4)	LysoPE 20:4	[M–H]–	1.313	500.28

Set-21	Phosphatidylcholine	UCD.Lipid.394	PC 35:4	PC (35:4)	PC(15:0_20:4)	[M+Ac–H]–	4.765	826.56
Set-21	Phosphatidylcholine	UCD.Lipid.398	PC 36:4 B	PC (36:4) B	PC(16:0_20:4)	[M+Ac–H]–	5.074	840.58
Set-21	Phosphatidylcholine	UCD.Lipid.401	PC 37:4	PC (37:4)	PC(17:0_20:4)	[M+Ac–H]–	5.39	854.59
Set-21	Phosphatidylcholine	UCD.Lipid.404	PC 38:4 A	PC (38:4) A	PC(18:0_20:4)	[M+Ac–H]–	5.706	868.61
Set-21	Phosphatidylcholine	UCD.Lipid.407	PC 40:4	PC (40:4)	PC(18:0_22:4)	[M+Ac–H]–	6.172	896.64
Set-22	Phosphatidylethanolamine	UCD.Lipid.168	PE p-34:1	PE (p-34:1) or PE (o-34:2)	PE(16:0_18:1)	[M+H]+	6.16	702.5404
Set-22	Phosphatidylethanolamine	UCD.Lipid.438	PE p-34:2	PE (p-34:2) or PE (o-34:3)	PE(P-16:0_18:2)	[M–H]–	5.64	698.51
Set-22	Phosphatidylethanolamine	UCD.Lipid.440	PE p-36:2	PE (p-36:2) or PE (o-36:3)	PE(P-18:0_18:2)	[M–H]–	6.314	726.54
Set-22	Phosphatidylethanolamine	UCD.Lipid.441	PE p-36:4	PE (p-36:4) or PE (o-36:5)	PE(P-16:0_20:4)	[M–H]–	5.523	722.51
Set-22	Phosphatidylethanolamine	UCD.Lipid.444	PE p-38:3	PE (p-38:3) or PE (o-38:4)		[M–H]–	6.48	752.56
Set-22	Phosphatidylethanolamine	UCD.Lipid.445	PE p-38:4	PE (p-38:4) or PE (o-38:5)	PE(P-18:0_20:4)	[M–H]–	6.189	750.54
Set-22	Phosphatidylethanolamine	UCD.Lipid.446	PE p-38:5	PE (p-38:5) or PE (o-38:6)	PE(P-18:0_20:5)	[M–H]–	5.582	748.53
Set-22	Phosphatidylethanolamine	UCD.Lipid.449	PE p-40:5	PE (p-40:5) or PE (o-40:6)	PE(P-18:0_22:5)	[M–H]–	6.214	776.56
Set-23	Phosphatidylcholine	UCD.Lipid.161	PC p-40:5	PC (p-40:5) or PC (o-40:6)		[M+H]+	5.74	820.6215
Set-23	Phosphatidylcholine	UCD.Lipid.162	PC p-40:6	PC (p-40:6) or PC (o-40:7) A		[M+H]+	5.16	818.6058
Set-23	Phosphatidylcholine	UCD.Lipid.163	PC p-40:6	PC (p-40:6) or PC (o-40:7) B		[M+H]+	5.625	818.6058
Set-23	Phosphatidylcholine	UCD.Lipid.164	PC p-40:7	PC (p-40:7) or PC (o-40:8)		[M+H]+	5.085	816.5902
Set-23	Phosphatidylethanolamine	UCD.Lipid.447	PE p-38:6	PE (p-38:6) or PE (o-38:7)	PE(16:0_22:6)	[M–H]–	5.34	746.51
Set-23	Phosphatidylethanolamine	UCD.Lipid.450	PE p-40:6	PE (p-40:6) or PE (o-40:7)	PE(P-18:0_22:6)	[M–H]–	5.998	774.54
Set-23	Phosphatidylethanolamine	UCD.Lipid.451	PE p-40:7	PE (p-40:7) or PE (o-40:8)	PE(P-18:1_22:6)	[M–H]–	5.407	772.53
Set-24	Phosphatidylethanolamine	UCD.Lipid.166	PE 36:4	PE (36:4)	PE(16:0_20:4)	[M+H]+	5.25	740.5184
Set-24	Phosphatidylcholine	UCD.Lipid.429	PE 34:1	PE (34:1)	PE(16:0_18:1)	[M–H]–	5.831	716.52
Set-24	Phosphatidylethanolamine	UCD.Lipid.430	PE 34:2	PE (34:2)	PE(16:0_18:2)	[M–H]–	5.332	714.51
Set-24	Phosphatidylethanolamine	UCD.Lipid.432	PE 36:2	PE (36:2)	PE(18:0_18:2)	[M–H]–	5.989	742.54
Set-24	Phosphatidylethanolamine	UCD.Lipid.433	PE 36:3	PE (36:3)	PE(18:1_18:2)	[M–H]–	5.399	740.52
Set-24	Phosphatidylethanolamine	UCD.Lipid.435	PE 38:4 B	PE (38:4) B	PE(18:0_20:4)	[M–H]–	5.881	766.54
Set-24	Phosphatidylethanolamine	UCD.Lipid.436	PE 38:6	PE (38:6)	PE(16:0_22:6)	[M–H]–	5.074	762.51
Set-25	Phosphatidylcholine	UCD.Lipid.140	PC 35:3	PC (35:3)		[M+H]+	4.861	770.5694
Set-25	Phosphatidylcholine	UCD.Lipid.141	PC 36:3 B	PC (36:3) B	PC(18:1_18:2)	[M+H]+	5.184	784.5848
Set-25	Phosphatidylcholine	UCD.Lipid.144	PC 37:3	PC (37:3)		[M+H]+	5.425	798.6004
Set-25	Phosphatidylcholine	UCD.Lipid.397	PC 36:3 A	PC (36:3) A	PC(18:1_18:2)	[M+Ac–H]–	5.23	842.59
Set-25	Phosphatidylcholine	UCD.Lipid.402	PC 38:2	PC (38:2)	PC(20:0_18:2)	[M+Ac–H]–	6.389	872.64
Set-25	Phosphatidylcholine	UCD.Lipid.403	PC 38:3	PC (38:3)	PC(18:0_20:3)	[M+Ac–H]–	5.981	870.62
Set-25	Phosphatidylinositol	UCD.Lipid.459	PI 38:3	PI (38:3)	PI(18:0_20:3)	[M–H]–	4.9	887.57

Set -26	Triacylglycerol	UCD.Lipid.235	TG 56:8	TG (56:8)	TG(18:1_18:2_20:5)	[M+Na]+	9.769	925.7256
Set -26	Triacylglycerol	UCD.Lipid.236	TG 56:9	TG (56:9)	TG(18:2_18:2_20:5)	[M+Na]+	9.48	923.7057
Set -26	Triacylglycerol	UCD.Lipid.240	TG 58:10	TG (58:10)	TG(18:2_18:2_22:6)	[M+Na]+	9.396	949.7256
Set -26	Triacylglycerol	UCD.Lipid.245	TG 58:8	TG (58:8)	TG(18:1_18:2_22:5)	[M+NH4]+	10.17	948.8015
Set -26	Triacylglycerol	UCD.Lipid.246	TG 58:9	TG (58:9)	TG(18:2_18:2_22:5)	[M+Na]+	9.794	951.7412
Set -26	Triacylglycerol	UCD.Lipid.249	TG 60:11	TG (60:11)		[M+NH4]+	9.645	970.7859
Set -27	Sphingomyelin	UCD.Lipid.172	SM d42:2	SM (d42:2)	SM(d18:1_24:1)	[M+H]+	6.685	813.6873
Set -27	Sphingomyelin	UCD.Lipid.488	SM d42:2 A	SM (d42:2) A	SM(d18:1_24:1)	[M+Ac–H]–	6.863	871.69
Set -27	Sphingomyelin	UCD.Lipid.489	SM d42:3	SM (d42:3)	SM(d18:2_24:1)	[M+Ac–H]–	6.289	869.68
Set -27	Sphingomyelin	UCD.Lipid.492	SM d44:2	SM (d44:2)		[M+Ac–H]–	7.528	899.72
Set -28	Cholesteroyl ester	UCD.Lipid.11	CE 18:1	CE (18:1)	18:1 Cholesteryl ester	[M+Na]+	10.88	673.5894
Set -28	Cholesteroyl ester	UCD.Lipid.13	CE 18:3	CE (18:3)	18:3 Cholesteryl ester	[M+Na]+	9.985	669.5581
Set -28	Cholesteroyl ester	UCD.Lipid.14	CE 20:3	CE (20:3)	20:3 Cholesteryl ester	[M+NH4]+	10.49	692.634
Set -28	Cholesteroyl ester	UCD.Lipid.15	CE 20:4	CE (20:4)	20:4 Cholesteryl ester	[M+Na]+	10.16	695.5738

423