

**Getting “Under the Skin”: Human Social Genomics in the
Multi-Ethnic Study of Atherosclerosis**

by

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Dedication

I dedicate this dissertation to my grandmother, Gertrude Delores Hampton. Nanny, no one wanted to see me become “Dr. Brown” more than you. I know that you are standing over the bannister of heaven smiling and beaming with pride. I love you more than my words could ever fully express.

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Abstract

The field of human social genomics examines the role of gene expression as a biological mediator in the relationship between the social environment and health. As many studies in this field have been small, lacked replication, and a consensus on best methodological approach, a larger epidemiologic investigation of such associations was warranted. The overarching goal of this dissertation is to contribute to the human social genomics literature by investigating the association between a range of social environmental factors (i.e. adult and childhood socioeconomic status (SES), loneliness, major or lifetime discrimination, perceived stress, chronic burden, and social support) and monocyte gene expression using the Multi-Ethnic Study of Atherosclerosis (MESA) and three different methodological approaches (i.e. an omnibus test for the entire gene expression set using Global Analysis of Covariance, testing each pair of social environmental factors and gene expression using multiple linear regression, and a machine learning method that performs variable selection with a correlated set of predictors called elastic net).

Aim 1 provides evidence that some genes are “socially sensitive” (i.e. demonstrate differential expression across social environmental exposures). Of the 1,854 gene transcripts previously identified as part of the ‘conserved transcriptional response to adversity’ only a small percent - between 0 to 11% - were associated with one of the seven aforementioned exposures. In Aim 2, a focus specifically on expression of inflammation and immune response gene pathways based on Gene Ontology classifications found significant associations between loneliness ($p=0.003$), chronic burden ($p=0.002$), and major or lifetime discrimination ($p=0.045$)

in global analyses with expression of the small subset of 20 gene transcripts related to the chronic inflammation pathway. In Aim 3, the extent to which social environmental factors explain the relationship between race/ethnicity and gene expression was examined. Of the 1407 unique gene transcripts examined, there were Black/White differences in 32%, Black/Hispanic differences in 19%, and Hispanic/White differences in 24%. Accounting for a racially/ethnically patterned social environmental factor (i.e. discrimination, adult SES) in the association between race/ethnicity and gene expression changed the effect estimate by >10% for less than 5% of the inflammation and immune response genes investigated.

In investigating the associations between social environmental factors and monocyte gene expression, this dissertation provides an important contribution in the field of human social genomics by examining the reproducibility of associations, the utility of different methodological approaches, and its contribution to racial/ethnic differences in gene expression.

CHAPTER I: Introduction

Goal

The overarching goal of this dissertation is to investigate the association between the social environment and gene expression in peripheral blood cells (i.e. monocytes). The contribution of social determinants (e.g. socioeconomic status, psychosocial stress, neighborhood environment, socially constructed racial categories) to health has been well established¹⁻⁴. However, the mechanisms through which social exposures “get under the skin” to affect biological functioning are just starting to be elucidated. Gene expression is a likely biological mediator in the association between the social environment and health, yet more research is needed to interrogate this relationship.

As proper immune system functioning plays a role in risk for both acute and chronic diseases, we focus attention on expression of genes in this biological process with a particular focus on those involved in the inflammatory response. Epidemiologic evidence suggests that chronic inflammation varies by social environmental factors including race/ethnicity. Contemporaneously, studies also show that exposure to adverse social environments is associated with gene expression. A main objective of this dissertation is to bridge these literatures by conducting an epidemiological study investigating the association between social environmental factors and inflammatory gene expression.

Significance

Social environmental exposures such as socioeconomic status, psychosocial stress, and socially constructed racial/ethnic categorizations have been consistently found to associate with risk of chronic disease^{2,4,5}. The burden of disease is exacerbated by the pronounced disparities that exist in the U.S. across social environment measures. For example, it has been estimated that 100,000 additional Black people die each year that would not die without the existence of racial health disparities, costing approximately 34 billion dollars in direct medical excess cost^{6,7}. In order to develop the most efficient treatment and prevention approaches to address the burden of chronic diseases, it is imperative that research uncovers the ways in which the social environment mechanistically affects biological functioning.

The intended contribution of this dissertation is to examine altered gene expression as a biological mediator that in part explains how the social environment (including race/ethnicity) affects disease risk. This work contributes to the emerging body of human social genomics literature that identifies genes whose expression is affected by social environmental exposures. This inherently interdisciplinary area is explored in this dissertation from multiple intersecting angles including social determinants of health, genetics, and the utility of three different methodological approaches in an effort to move this field forward.

Background

Social Environment and Disease Risk

Substantial research in the epidemiological literature has found associations between the social environment and disease risk^{2,4,5}. Among the most consistent social environmental factors

found to associate with disease risk are socioeconomic status, psychosocial stress, and socially constructed race/ethnicity. Socioeconomic status (SES) is an important social environment exposure that is considered a fundamental cause of disease for four reasons: 1) it influences multiple disease outcomes 2) it affects disease outcomes through multiple risk factors 3) it involves access to resources that can be used to avoid or minimize consequences of disease once it occurs and 4) it is reproduced over time through the replacement of intervening mechanisms². SES is associated with health through a variety of environmental, behavioral, and physiological pathways^{2,8}. Factors such as access to health care, opportunities for physical activity, and access to healthy foods represent some of the mechanisms through which SES affect health⁹⁻¹¹. Despite use of varying measures of socioeconomic status (e.g. income, education, subjective social status) in the literature, SES has consistently been found to inversely associate with risk of many disease outcomes including heart disease, cancer, stroke, and diabetes¹²⁻¹⁵.

Interestingly, socioeconomic status in childhood has been found to be associated with adult health status even after controlling for adult SES¹⁶. Reviews by Galobardes and colleagues representing 40 studies found that childhood SES is inversely associated with risk of mortality from cardiovascular disease, respiratory disease, diabetes, cancers of the lung, liver, and stomach, diseases of the digestive system, and overall mortality^{16,17}. Like SES, psychosocial stress is an important social environmental exposure that has been shown to predict disease risk. Psychosocial stressors include factors such as perceived stress, discrimination, and chronic burden. The extant literature suggests that psychosocial stress exposure is associated with numerous disease outcomes including stroke, transient ischemic attack, atherosclerosis, and hypertension^{1,18,19}. Further, a meta-analysis of 10 prospective cohort studies (n=68,222) found

evidence of a dose response relationship between psychosocial distress with risk of mortality even after adjustment of behavioral and socioeconomic factors ²⁰.

The persistent racial/ethnic disparities in health can be partially explained by exposure to racially/ethnically patterned social environments²¹⁻²³. In the United States, Blacks and Hispanics tend to have lower socioeconomic statuses, as measured by income, education, and job status compared to non-White Hispanics ^{4,24}. Recent work suggests that socioeconomic and demographic variables together may explain up to 80% of Black-White racial disparity in life expectancy for males and 70% for females ²⁴. Further, Blacks and Hispanics consistently report higher levels of psychosocial stress in terms of frequency, longevity, and reactivity ^{25,26}. Interestingly, it has also been found that U.S. born Hispanics have similar stress levels as Blacks while foreign born Hispanics had levels similar to Whites suggesting that minority status contributes to stress levels²⁶.

Chronic Inflammation

To fully understand how social environmental factors affect disease risk, it is important to investigate the relationship between the social environment and biological risk factors. In this dissertation, we focus specifically on chronic inflammation, a risk factor for several chronic diseases that has been suggested as a mechanism through which the social environment (e.g. SES and psychosocial stress) associates with chronic disease risk²⁷⁻²⁹. Chronic inflammation is characterized by prolonged and persistent inflammation lasting weeks to years due to an inability to overcome the effects of an injuring agent or a hyperresponsiveness of the immune response ³⁰. In chronic inflammation, mononuclear cells in peripheral blood infiltrate tissues and produce proteinases and fibroblasts. While these processes are beneficial in the short term to rid the body

of injuring agents, in the long term, these processes lead to destruction of the body's healthy tissue.

The consequences of a chronic inflammatory profile extend across a number of high burden diseases. For example, chronic inflammation has been linked to cardiovascular disease, diabetes, and some types of cancer³¹⁻³⁵. A study by Zacho and colleagues found that people with high levels of the inflammatory biomarker C-reactive protein (CRP) had 1.6 times the risk of ischemic heart disease and 1.3 times the risk of cerebrovascular disease compared to those with low levels³¹. Levels of the inflammatory biomarkers interleukin-6 (IL-6), CRP, and tumor necrosis factor-alpha (TNF-a) were shown to be higher in colorectal cancer patients compared to normal controls³⁵. Among cancer patients, levels of these biomarkers were found to be associated with tumor size³⁵. Physiologically, chronic inflammation is believed to play a role in both atherosclerotic plaque formation and rupture which are characteristic of cardiovascular disease³⁰. In diabetes, inflammation is thought to lead to insulin resistance and beta cell dysfunction via oxidative stress and endoplasmic reticulum stress mechanisms^{36,37}. Inflammatory signaling also plays a role in processes associated with cancer development including cell proliferation, resistance to apoptosis, and genomic instability³⁴.

Based on its association with disease, investigations of the factors that affect chronic inflammation is warranted. The current literature suggests that chronic inflammation may be influenced by both environmental (social environment) and biological (e.g. gene expression) factors. A study using the Midlife in the United States (MIDUS) study found that socioeconomic status, as measured by income and education, was inversely associated with C-reactive protein (CRP), interleukin-6 (IL-6), and fibrinogen levels²⁹. This finding has held across additional measures of SES in other studies such as job status, which has been found to associate with CRP,

IL-6, sICAM1 and ET-1³¹⁻³⁴. These findings have also been consistent across geographic contexts. In an international review across 9 countries (US, UK, Finland, Greece, Germany, Canada, Italy, Turkey, and New Zealand) spanning 20 studies, SES was found to be inversely associated with CRP in 19/20 studies⁴¹. Similarly, socioeconomic status in childhood has also been found to associate with adult inflammatory profile. A study in the Atherosclerosis Risk in Communities (ARIC) found that childhood SES, as measured by social class and parental education, was inversely associated with levels of the inflammatory markers fibrinogen, von Willebrand factor, CRP, and WBCs in adulthood⁴². A 2011 study by Carroll and colleagues found childhood SES at 1-2 years of life to be associated with IL-6 levels in adulthood, even after controlling for adult SES⁴³. Further, it has been suggested that childhood SES can predict CRP levels of offspring pointing towards intergenerational effects of childhood SES on inflammation and disease risk⁴⁴.

Associations between psychosocial measures and inflammation have been previously investigated as well. Loneliness, the perception of the quality of one's personal relationships is a strong risk factor for both acute and chronic diseases⁴⁵⁻⁴⁷. Lonely individuals report higher levels of stress, even after accounting for objective measures of social isolation⁴⁸. Impaired immune system functioning is one the physiological consequences of loneliness^{28,46,48}. Research has shown that loneliness is associated with delayed wound healing and altered cytokine likes (e.g. interleukin-1B, tumor necrosis factor)²⁸.

Studies of the association between discrimination and protein level markers of inflammation have yielded mixed findings. For example, a study by Lewis and colleagues found a positive association between everyday discrimination and CRP using data from the Minority Aging Research Study of older African-Americans, yet a study by Kershaw and colleagues found

no relationship using the Multi-Ethnic Study of Atherosclerosis^{49,50}. The complex relationship between discrimination and inflammation may be modified by factors such as race/ethnicity, sex, and body mass index^{50,51}.

Stress, whether experienced acutely or chronically has been associated with levels of inflammatory biomarkers. A meta-analysis consisting of 30 studies that assessed the relationship between acute psychological stress found that stress exposure consistently led to altered levels of inflammatory biomarkers including CRP, IL-16, TNF-alpha, and IL-1 β ⁵² whereas chronic stress exposure such as job stress, childhood adversity, and caregiver stress, have been shown to have significant associations with levels of inflammatory biomarkers as well⁵³.

Social support has been found to positively influence health in part due to its ability to buffer the effects of exposure to social and psychosocial stressors⁵⁴. Epidemiologic evidence suggests that social support is associated with natural killer cell activity, an important leukocyte that is responsible for neutralizing tumor and virus infected cells⁵⁵. The relationship between social support and inflammation has been inconsistent with some studies showing an inverse relationship and others showing no relationship which may be in part due to lack of consideration of potential effect modifiers (e.g. race/ethnicity) in the literature⁵⁶.

Racial/ethnic differences in inflammation exist where minority groups, Blacks and Hispanics in particular, have adverse inflammatory profiles compared to non-Hispanic Whites. For example, Blacks had higher levels of CRP compared to non-Hispanic Whites in sex stratified analyses in a sample from the Dallas Heart Study⁵⁷. A study using NHANES assessing the association between acculturation and inflammation among Hispanics found that more acculturated Hispanics had higher levels of CRP compared to less acculturated Hispanics, suggesting that minority status may have an effect on inflammation⁵⁸. The association between

minority status and inflammation in U.S. samples is supported by a trans-country review of 15 studies where the minority group had a worse inflammatory profile compared to the majority group in all countries⁴¹. This review included studies from the United States, United Kingdom, and Canada. As expected, in the U.S. studies, Blacks and Hispanics tended to have higher levels of the inflammatory biomarker CRP compared to non-Hispanic Whites. Similarly, studies in the U.K. found that South Asians, the minority group, had higher levels compared to European Whites. South Asians and Aboriginals had higher CRP levels than Europeans and Chinese individuals in the Canadian study. This review suggests that membership in a racial/ethnic minority group is associated with adverse inflammatory profiles and highlights the effect of socially constructed race/ethnicity on inflammation.

While most work investigating the association between the social environment and inflammation has been conducted with protein markers, some studies have interrogated this relationship at the gene level. Investigators in the emerging field of human social genomics have begun to identify genes whose expression is sensitive to social environment stimuli^{59,60}. It has been proposed that genes involved in certain biological processes, including inflammation, are differentially expressed based on exposure to adverse social environments, a phenomenon that has been coined “conserved transcriptional response to adversity.” For example, genome wide analyses investigating the relationship between loneliness and socioeconomic status with gene expression implicated 144 and 387 genes respectively, and there was an enrichment of proinflammatory genes among the differentially expressed^{48,61}. Other studies have found a range of social environmental factors to associate with inflammatory gene expression including caregiver stress⁶², well-being⁶³, positive vs. negative affect⁶⁴, and grief⁶⁵. Such studies have

helped to shed additional light on gene expression as a biological mechanism whereby social environmental factors affect biological functioning and thereby overall health^{61,66}.

Although genome wide studies have found evidence for racial/ethnic differences in gene expression, previous human social genomics studies have had limited capacity to investigate social environmental exposures as contributors to race/ethnicity differences in gene expression due to the use of homogenous samples. Using samples from HapMap, Storey et. al and Zhang et al. found gene expression differences between Whites and Yoruba Nigerians in 17% and 34% of genes respectively^{67,68}. Storey found that among differentially expressed genes, there was an enrichment of inflammation associated genes⁶⁷. However, the extent to which the social environmental factors can explain racial/ethnic differences in gene expression remains largely uninvestigated.

In this dissertation, we extend our current understanding of human social genomics by investigating associations between the social environment and gene expression, with a particular focus on inflammation and immune response genes. This work helps to elucidate the mechanisms through which well-established associations between the social environment and health act such that more precise prevention and treatment approaches can be developed.

Research Aims

Aim 1: Conduct a large, epidemiologic study assessing the association between a range of social environmental factors (i.e. childhood SES, adult SES, perceived stress, social support, perceived discrimination, loneliness, and chronic burden) with expression of the set of genes previously implicated as part of the conserved transcriptional response to adversity

Hypothesis 1: We hypothesize that the social environmental factors will be significantly associated with the conserved transcriptional response to adversity genes in the MESA sample.

Aim 2: To examine the association between a range of social environmental factors (i.e. childhood SES, adult SES, perceived stress, social support, perceived discrimination, loneliness, and chronic burden) with the expression of inflammatory and immune system related genes identified using the Gene Ontology database including: chronic inflammatory response genes (GO: 0002544), inflammatory response genes (GO: 0006954), immune response (GO: GO:0006955), and the regulation of the inflammatory response (GO: 0002544).

Hypothesis 2: We hypothesize that there will be an association between the social environmental factors with the expression of genes in the selected gene sets.

Aim 3: Evaluate whether there exist racial/ethnic differences in gene expression for inflammatory and immune response genes and the extent to which the associations can be explained by racially/ethnically patterned social environmental factors.

Hypothesis 3: There will be racial/ethnic differences in gene expression which will be partially explained by social environmental factors.

CHAPTER II: Assessing the conserved transcriptional response to adversity in the Multi-Ethnic Study of Atherosclerosis: a gene set approach

Introduction

It has been well established that the social environment influences health. Social and psychosocial factors have been consistently associated with both acute and chronic diseases ranging from the common cold to cardiovascular disease and diabetes ^{2,22,69,70}. Research has now turned to understanding the ways in which these social exposures “get under the skin” and cause adverse biological effects that lead to these diseases. One of the mechanisms through which the social environment may increase disease risk is via impaired immune system functioning. A large literature has documented associations of low socioeconomic status (SES) in both childhood and adulthood with inflammation in later life ^{29,41,42,71}. Additionally, several psychosocial factors including discrimination, perceived stress, depression, and cynical distrust have also been associated with altered inflammatory processes ^{49,52,53,72,73}. Inflammatory processes have in turn been associated with chronic diseases such as cardiovascular disease and diabetes ^{34,74-76}. However, the precise biologic mechanisms linking low SES or psychosocial exposures to a heightened inflammatory response have yet to be determined.

Recent studies provide preliminary evidence that exposure to adverse social environments is associated with differential gene expression, particularly in immune system

related genes. For example, Cole and colleagues report differential expression of 144 genes (209 transcripts) between individuals with high levels of loneliness and low levels of loneliness where more lonely individuals overexpress proinflammatory genes and underexpress antiviral immune response genes⁴⁸. Subsequent studies by Cole and others have found similar associations between low socioeconomic status⁶¹, caregiver stress⁶², well-being⁶³, positive vs. negative affect⁶⁴, and grief⁶⁵ with expression of immune system related genes. Studies in animals have shown similar findings. Maternal versus surrogate rearing was associated with differential expression of immune system genes in rhesus macaques⁷⁷. In mice, differential expression of inflammatory genes was observed between mice exposed to repeated social defeat and control mice⁶¹. This response of altered gene expression in immune-system related genes in response to adverse social exposures has been coined the “conserved transcriptional response to adversity” (CTRA) and is categorized by an upregulation of proinflammatory genes and down regulation of Type 1 interferon and antibody synthesis genes^{60,78}.

The promising preliminary findings in previous studies of the conserved transcriptional response to adversity motivated us to raise the larger question of whether expression of previously implicated genes would vary with a range of social environmental factors in a large, epidemiologic cohort. To our knowledge, there has not been a systematic, large epidemiological study of the relationship between social environmental factors and gene expression.

In the present study, we use the Multi-Ethnic Study of Atherosclerosis to assess whether seven social and psychosocial factors (e.g. childhood socioeconomic status, adult socioeconomic status, loneliness, lifetime discrimination, chronic burden, perceived stress, and social support) are associated with expression of genes previously found to be related to social and psychosocial factors (Table 1). As the field of human social genomics is relatively new, there is not yet a

consensus on the best method for assessing the association between social exposures and gene expression. The correlated nature of gene expression across the genome and the high probability of false positives from multiple testing make it difficult to identify ideal statistical methods to assess associations with social environmental factors. In this paper, we examine three methods that provide different, but complementary insights into approaches to investigating the association between social environmental factors and genes expression by focusing on genes implicated as part of the conserved transcriptional response to adversity. In the first approach, we employ a self-contained gene set enrichment test, Global Analysis of Covariance (ANCOVA), to globally assess whether expressions of any of the CTRA genes are associated with the exposure. Secondly, we use multivariable linear regression to identify which specific genes are significantly associated with the exposure. In the third approach, we use elastic net penalized regression as a variable selection procedure to extricate the genes that are the most robustly associated with social environmental factors

Methods

Study sample

The Multi-Ethnic Study of Atherosclerosis (MESA) was designed to investigate risk factors for the development and progression of subclinical cardiovascular disease⁷⁹. The baseline cohort was comprised of 6,814 adults aged 45-84 who self-identified as African-American, Chinese-American, White, or Hispanic and were free from clinical cardiovascular disease. Participants were recruited from six field sites across the United States between 2000 and 2002 (Baltimore, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles,

California; New York, New York). Four follow-up examinations have been conducted with Exam 5, the fourth follow up, ending in December 2011. The response rate has been excellent with 78% participants returning for Exam 5. Each exam consisted of a clinic visit where questionnaires on demographic, psychosocial, and lifestyle factors were administered, and physical assessments including the blood draw needed for genetic analyses were conducted.

Gene expression was collected solely in Exam 5 on a random sample of 1,264 individuals⁸⁰. There were three racial/ethnic groups represented in this subsample where 272 participants were non-Hispanic Black, 402 were Hispanic, and 590 were non-Hispanic White. Participants were recruited from four of the six MESA study sites: Forsythe County, North Carolina (49 participants), New York, New York (424 participants) Baltimore, Maryland (317 participants) and St. Paul, Minnesota (474 participants). Non-Hispanic Blacks were recruited mostly from the New York and Baltimore sites. Hispanics were recruited from the New York and Rochester sites. Whites were recruited from all four sites. These individuals make up the study sample for the current study.

Social environmental factors

Adult Socioeconomic Status

Highest level of education was collected in Exam 1 and was dichotomized as a measure of adult socioeconomic status. Respondents were considered highly educated if they had obtained a college degree or higher.

Childhood Socioeconomic Status

Mother's and father's level of education were collected in Exam 2 to proxy childhood socioeconomic status. A parent was considered highly educated if he or she had achieved at least a high school degree. Childhood SES was dichotomized. The respondent was considered to have a high childhood SES if either parent had at least a high school degree.

Perceived Stress

Perceived stress was measured using Cohen's the 4-item Perceived Stress Scale (PSS) in Exam 5^{81,82}. The respondent was asked whether they felt: unable to control the important things in his or her life, confident about his or her ability to handle personal problems, things were going his or her way, or difficulties were piling up so high that he or she could not overcome them in the past month. Respondents answered on a five point Likert scale corresponding to the answer choices never, almost never, sometimes, fairly often, and very often. A summary measure was created by reverse coding the positive items (items 2 & 3) and summing the items such that a higher value indicated a higher level of perceived stress.

Major or Lifetime Discrimination

Major or lifetime discrimination was collected in Exam 1 and was adapted from the Detroit Area Study⁸³. Respondents were asked about whether they ever had been fired or denied a promotion, not hired for a job, treated unfairly by the police, discouraged by a teacher from continuing education, prevented from moving into a neighborhood, or neighbors have made their life difficult⁸³. A discrimination score was computed by summing the number of 'yes' responses. A higher score indicated higher exposure to major or lifetime discrimination.

Chronic Burden

Chronic burden was assessed using the Chronic Burden Scale in Exam 3⁸⁴. Participants were asked whether they had experienced ongoing problems in the following five domains: their own health, health of a loved one, job, relationship, financial problems. For affirmative responses, participants were subsequently asked whether this had been a problem for at least 6 months and whether this burden was not very stressful, moderately stressful, or very stressful. To estimate overall chronic burden, we summed the number of domains for which the respondent had experienced a chronic burden for at least 6 months and reported that it was either moderately or very stressful. A higher score indicated a higher level of chronic burden.

Social support

Social support was measured in Exam 4 using a 4-item scale adapted from the Midlife in the United States (MIDUS) study⁸⁵. The questions asked how much friends and family can be relied upon for help with a serious problem, how much friends and family can be opened up to talk about worries, how often friends and family make too many demands on the respondent, and how often friends and family let them down. Possible answer choices included: a lot, some, a little, not at all and were coded as 1, 2, 3, and 4 respectively. Positive items (items 1 & 2) were reverse coded and the sum of the 4 items was calculated to achieve an overall social support score where a higher score indicated greater social support.

Loneliness

Loneliness was measured in Exam 4 using a three item scale adapted from the UCLA Loneliness Scale⁸⁶. Participants were asked how often they lack companionship, feel left out, or

isolated from others. Possible answer choices included: hardly ever, some of the time, and often and were coded as 1, 2, and 3 respectively. A score was created by summing the three items. A higher score indicated a higher level of loneliness.

Covariates

Age, sex, and race/ethnicity were self-reported via questionnaire.

Gene expression

Gene expression data was collected from purified monocytes of 1,264 participants in MESA Exam 5. Detailed methods have been previously described⁸⁰. Briefly, peripheral blood mononuclear cells (PBMCs) were separated within two hours of blood draw using Vautainer CPT cell separation tubes. Monocytes were purified from the PBMCs using anti-CD14-coated magnetic beads. DNA and RNA were extracted using the AllPrep DNA/RNA Mini Kit. The resulting cRNA was hybridized to the Illumina HumanHT-12 v4 Expression BeadChip. This chip has probes for 47,231 transcripts (~31,000 genes), and is designed to assay 12 samples per chip. A stratified random sampling technique was used to assign samples to each chip avoid biases due to batch, chip, or position.

Preprocessing and quality controls steps were conducted to ensure accurate quantification of the gene expression data. Illumina's proprietary software Genome Studio was used to correct for local background. The remaining preprocessing steps were conducted using Bioconductor packages in R. Since the bead chip has multiple copies of each probe, a bead-type summarization (mean and variance) was produced for each transcripts using the *beadarray* package⁸⁷. The negative controls on the array were used to compute the detection p-value. The

limma package was used for background correction, quantile normalization, \log_2 transformation, and removal of control probes⁸⁸. Quality control criteria for elimination of a transcript included: ‘detected’ expression levels in <10% of MESA samples (detection p-value cut-off=0.01), probes that contain a SNP, probes with low variance across samples (<10th percentile), and probe overlap with a non-unique region.

Gene Set Definition

A systematic literature review was conducted in July 2015 to identify all primary studies that reported finding an association between a social or psychosocial factor and the “conserved transcriptional response to adversity.” Google Scholar and PubMed databases were used with the search term “conserved transcriptional response to adversity.” This search yielded a total of 63 unique studies. The following inclusion criteria were applied: primary study, gene expression as outcome, and a primate organism. These criteria resulted in 8 studies to be included in the analyses. Since the 2007 study by Cole and colleagues was foundational in describing the conserved transcriptional response to adversity research, we also included this study. Therefore, 9 studies were included (Table 1). Of these, 8 were conducted in humans and 1 in rhesus macaques. We combined all the genes that were considered differentially expressed to compose a single gene set. The HUGO Gene Nomenclature Committee (HGNC) Multisymbol Checker was used to identify all potential synonyms and previous names of genes. Genes that had been withdrawn or did not match to an approved HGNC gene name were excluded. We then used the BioMart ID Conversion tool to match gene names to the transcript identification number on the Illumina HumanHT-12 v4 gene chip. When a gene appeared in more than one study, we only included it once in the gene set (Appendix 1). There was some overlap in implicated genes

between studies (Appendix 2). For cases where a gene matched to more than one transcript, we included all transcripts in the analyses. *The final gene set consisted of 1,854 transcripts representing 1,305 unique genes* (Appendix 1).

Table 1. Primary Studies of the Conserved Transcriptional Response to Adversity in Primates			
Study	Exposure	# significant genes	Sample size
Cole, Steven W., et al. (2007)	Loneliness	144	14
Miller, Gregory E., et al. (2008)	Caregiving stress	542	21
Cole, Steven W., et al. (2012)	Maternal rearing vs. surrogate rearing	132	9 rhesus macaques
Antoni, Michael .H., et al (2012)	Affect Behavioral Therapy	201 90	199
Fredrickson, Barbara L., et al (2013):	Hedonic and eudaimonic well-being	53*	84
Powell, Nicole D., et al. (2013)	Socioeconomic status	387	60
O'Connor, M., et al (2014)	Non-complicated grief Complicated grief	285 83	63
Wingo, A. and Gibson, G (2015)	Anxiety disorder	631	336
Vedhara, K., et al (2015)	Neuroticism, Extraversion, Openness Agreeableness, Conscientiousness	53*	121
<i>Total number of unique genes (transcripts): 1,305 (1,854)</i>			
*In the Fredrickson and Vedhara studies, 53 genes representing the conserved transcriptional response to adversity were selected a priori to construct a contrast score used in the analysis.			

Statistical Analyses

Global Test

We assessed the association between each social environmental factor and the entire set of gene expression levels from all previously implicated genes (1,854 transcripts from 1,305

unique genes) using a global test (Global ANCOVA)⁸⁹. This test has the null hypothesis that no genes in the gene set are associated with exposure status^{89,90}.

Briefly, a linear model where the outcome is the expression of one gene transcript is the building block of the Global ANCOVA model. Expression for the i th gene is expressed as $\tilde{x}^{(i)}$, n is the number of individuals, and d is the number of covariates.

$$\tilde{x}^{(i)} = C \cdot \tilde{\beta}_i^t + \tilde{\xi}^{(i)} = \begin{pmatrix} 1 & c_{11} & \cdots & c_{1d} \\ \vdots & \vdots & & \vdots \\ 1 & c_{n1} & \cdots & c_{nd} \end{pmatrix} \cdot \begin{pmatrix} \beta_{i0} \\ \vdots \\ \beta_{id} \end{pmatrix} + \begin{pmatrix} \xi_1^i \\ \vdots \\ \xi_n^i \end{pmatrix} \quad (1)$$

C is a matrix of size $n \times (d+1)$ which represents the covariate values for each individual. The gene specific mean expression is quantified by β_{i0} . The regression coefficient vector, $\tilde{\beta}_i^t$, reflects the influence of each covariate on expression of the i^{th} gene. The residual for each individual is given by the $\tilde{\xi}^{(i)}$ vector.

In the Global ANCOVA model, expression of p genes is measured in n individuals. The linear model is extended to account for all genes under investigation in a gene set. This multivariate model is written as:

$$\tilde{X} = \begin{pmatrix} \tilde{x}^{(1)} \\ \vdots \\ \tilde{x}^{(p)} \end{pmatrix} = \tilde{C} \cdot \tilde{\beta} + \tilde{E} = \begin{pmatrix} C & 0 & 0 \\ 0 & \ddots & 0 \\ 0 & 0 & C \end{pmatrix} \cdot \begin{pmatrix} \tilde{\beta}_1^t \\ \vdots \\ \tilde{\beta}_p^t \end{pmatrix} + \begin{pmatrix} \tilde{\xi}^{(1)} \\ \vdots \\ \tilde{\xi}^{(p)} \end{pmatrix} \quad (2)$$

The gene expression values of the entire gene set (\tilde{X}), is expressed as an np column vector, representing gene expression of each gene for each individual. It is a function of the product \tilde{C} and $\tilde{\beta}$ plus \tilde{E} , where \tilde{C} is a $np \times (d+1)p$ sized diagonal block matrix representing the

covariate information for each individual, $\tilde{\beta}$ is a column vector of size $(d+1)p$ which quantifies the relationship of each exposure and covariate on the expression of each gene in the gene set, and \mathcal{E} is np sized column vector which accounts for the noise for each individual for each gene in the gene set. For each Global ANCOVA test in our study, there were 1,264 participants (n), one main social environmental factor of interest (each investigated in a separate model), and 4 additional covariates (age, sex, study site, and race/ethnicity).

To assess the null hypothesis, $H_0: \beta_1=\beta_2 \dots \beta_p=0$, an F test was conducted using the residual sum of squares from reduced (covariate matrix excludes the exposure of interest) and full (covariate matrix contains the exposure of interest) models to determine the significance for each social environmental factor of interest. The F statistic is given by:

$$F_{GA} = \frac{RSS_{RM} - RSS_{FM}}{RSS_{FM}} \cdot \frac{n-q}{f} \quad (3)$$

where RSS_{RM} is the residual sum of squares of the reduced model, RSS_{FM} is the residual sum of squares of the full model, n is the number of samples, q is the number of parameters in the full model, and f is the difference in number of parameters between the full and reduced models.

Since the assumption of independent homoscedastic gene expression is unlikely to hold and is necessary for the classical F-test, we used a permutation based approximation approach in order to assess significance of the Global ANCOVA tests using the strategy described in Hummel 2008⁸⁹. Briefly, we permuted the rows of covariate matrix of the reduced model 10,000 times and calculated the residuals of the full model by fitting the residuals of the reduced model to the permuted design matrix to develop the resampled value of the F statistic. An empirical p-value is given by the fraction of F statistics that are larger than the actual F_{GA} .

Gene level analyses

Multivariable linear regression

While the global test evaluates the compound hypothesis that at least one gene in a particular set is differentially expressed by social environmental exposure, it is not designed to identify which gene in the set leads to the rejection of the null hypothesis. We conducted subsequent analyses estimating the regression coefficient for each gene in a significant gene set using the multivariable linear regression model in Equation 1. A false discovery rate correction for correlated data of 10% was used to account for multiple testing⁹¹.

Elastic net regression

While examining single gene effects is common practice, it has low power to identify the key gene transcripts after adjustment for multiple testing. Moreover, a one at a time analysis can miss important correlation across gene transcripts. Ordinary least squares regression with all 1,854 gene transcripts simultaneously in the model cannot be operationalized when the number of gene transcripts (p) exceeds the number of individuals (n).

There are a number of alternative approaches that handle the curse of dimensionality (i.e. issues that arise in analyzing high dimensional data) by using penalization techniques⁹². Whereas OLS identifies the best fit line by minimizing the residual sum of squares (i.e. $\text{argmin}_{\beta} |y - X\beta|^2$), penalized regression methods subject this term to an additional penalty/penalties to improve prediction and interpretation^{93,94}. Ridge regression is one such alternative that minimizes

$$\min_{\beta_i} \left[\sum_{j=1}^n (y_j - \sum_{i=1}^p x_{ji} \beta_i)^2 + \lambda \sum_{i=1}^p \beta_i^2 \right],$$

where in our present analysis, the 1,854 gene transcript expressions that make up our CTRA gene set are the high dimensional predictor variables (x) and the social environmental factor is the outcome (y). For n individuals and p gene transcript expressions, let y_j be the value of the social environmental factor for person j , x_{ji} be the gene expression value for person j for gene i , and β_i be the beta estimate corresponding gene i . Ridge regression can be viewed as a way to handle collinearity among the predictors and stabilize OLS when $p > n$. The term $\sum_{i=1}^p \beta_i^2$ is often referred to as the L_2 norm. The tuning parameter λ is optimally chosen by using cross-validation techniques. Ridge regression shrinks the beta-coefficients towards zero and stabilizes the design matrix for the multivariable regression with correlated predictors. Ridge regression is particularly well-suited for prediction problems. It does not offer the option of variable selection or identifying important gene transcripts as it retains all the predictors in the model.

Modern variable selection methods, in particular LASSO (least absolute shrinkage and selection operator) conducts both shrinkage and variable selection and has the ability to shrink the beta coefficients exactly to zero, thus performing variable selection⁹³. In contrast to Ridge regression, LASSO minimizes the following penalized error:

$$\min_{\beta_i} \left[\sum_{j=1}^n \left(y_j - \sum_{i=1}^p x_{ji} \beta_i \right)^2 + \lambda \sum_{i=1}^p |\beta_i| \right]$$

Here the L_2 norm penalty in Ridge regression ($\sum_{i=1}^p \beta_i^2$) is replaced by L_1 norm penalty ($\sum_{i=1}^p |\beta_i|$). However, LASSO does not perform well when the predictors (x) are correlated.

LASSO indiscriminately selects just one gene if a group of genes are highly correlated. Since genes in a biological pathway are expected to be correlated, selection of just one gene would preclude detection of biological processes.

The elastic net is a hybrid of Ridge regression and LASSO and does a better job of conducting both variable selection and adjustment for correlation among predictors through use of both L_2 and L_1 penalties. For high-dimensional, correlated data, the elastic net has been demonstrated to be superior to LASSO for selecting predictors that create the most parsimonious model⁹⁴ with optimal prediction error. In this study, because of the large number of gene transcripts ($p=1,854$) that are potentially correlated and modest number of observations ($n=1,264$), to fit a multivariable regression model, we use elastic net to identify the transcripts most strongly related to the social/psychosocial factor.

The elastic net penalty is given by:

$$\min_{\beta_i} \left[\sum_{j=1}^n \left(y_j - \sum_{i=1}^p x_{ji} \beta_i \right)^2 + \lambda_2 \sum_{i=1}^p \beta_i^2 + \lambda_1 \sum_{i=1}^p |\beta_i| \right]$$

where in the present study $n=1264$ individuals and $p=1854$ gene transcripts.

The two tuning parameters λ_1 and λ_2 control the trade-off between bias and variance⁹⁵. To numerically search for the optimal set of tuning parameters, λ_1 is often fixed on a grid of values and the optimal λ_2 is determined using leave one out cross validation⁹⁶. Elastic net penalized regression⁹⁴ was employed using the ‘glmnet’ package in R⁹⁷. In actual implementation, an alternative parametrization of elastic net is used:

$$\min_{\beta_i} \left[\sum_{j=1}^n \left(y_j - \sum_{i=1}^p x_{ji} \beta_i \right)^2 + \lambda \left(\alpha \sum_{i=1}^p \beta_i^2 + (1 - \alpha) \sum_{i=1}^p |\beta_i| \right) \right]$$

Where $\lambda = \lambda_1 + \lambda_2$, and $w = \lambda_1/(\lambda_1 + \lambda_2)$. Note that $\alpha = 0$ corresponds to LASSO and $\alpha = 1$ corresponds to Ridge regression. Often the default choice of $\alpha = 0.5$ is used and λ is chosen by cross-validation.

Adjustments

To ensure comparability across the three statistical approaches, we adjusted the social environmental factors and gene expressions for age, sex, site, race/ethnicity, and chip prior to the main analyses.

Statistical software

SAS 9.3 and R were used to conduct statistical analyses.

Results

In the sample of 1,264 MESA participants with available gene expression data, the mean age was ~70 years and 51% of the sample was female. For the socioeconomic variables, 33% of the total sample of adults had finished a level of schooling equal to a college degree or higher. Slightly over half of the sample (56%) had either a mother or father achieve at least a high school degree. Whites (49%) were more likely to have obtained a college degree than Blacks (27%) or Hispanics (13%) ($p < 0.001$ and $p < 0.001$ respectively). Blacks were significantly more likely to have a college degree than Hispanics ($p < 0.0001$). The same pattern was observed for childhood SES where Whites (71%) were more likely to have at least one parent with a high school degree

compared to Blacks (55%) or Hispanics (31%) ($p < 0.001$ and $p < 0.001$ respectively), and Blacks were significantly more likely to have a parent with a high school degree than Hispanics ($p < 0.0001$). For most of the psychosocial exposures, the median scores were close to the minimum score indicating that most of the participants reported low levels of these exposures. There were not significant racial/ethnic differences in these scores except for major or lifetime discrimination ($p < 0.001$). Tukey's honest significance test indicated that Blacks reported higher levels than Whites ($p < 0.001$) or Hispanics ($p < 0.001$). There was not a significant difference between Whites and Hispanics ($p = 0.16$). The correlation among the psychosocial exposures is presented in Appendix 3. The most correlated exposures were loneliness and chronic burden ($r = 0.36$) and the least correlated exposures were major or life discrimination and perceived stress ($r = 0.05$). These relationships were similar across race/ethnicity.

Table 2. Characteristics of the MESA Study Sample with Gene Expression Data				
	Total sample (n=1264)	Non-Hispanic Black (n=272)	Hispanics (n=402)	Non-Hispanic White (n=590)
Demographics				
Age mean (SD)	69.6 (9.4)	69.6(9.0)	68.4(9.3)	70.2(9.5)
Sex (% female)	51	60	50	48%
Study Site (n,%)				
Forsythe County, North Carolina	49(4%)	1(0.3%)	0	48 (40%)
New York, New York	424(34%)	131(48%)	209 (52%)	84 (14%)
Baltimore, Maryland	317(25%)	140(51%)	0	177 (30%)
Rochester, Minnesota	474(38%)	0	193 (48%)	281 (48%)
Total	1264(100%)	272 (100%)	402 (100%)	590 (100%)
Socioeconomic Status				
High education- respondent*	33%	27%	13%	49%
High education- either parent**	56%	55%	31%	71%
Psychosocial factors Median, (Interquartile Range)				
Loneliness	3 (3-5)	3(3-4)	4(3-5)	3 (3-5)
Lifetime Discrimination	0 (0-1)	1(0-2)	0(0-1)	0 (0-1)
Chronic Burden	1 (0-2)	1(0-2)	0(0-2)	1 (0-2)
Perceived Stress	8 (5-10)	8(5-10)	8(5-10)	7 (6-10)
Social support	9 (8-10)	9(8-10)	9(8-10)	9 (8-10)
*High education for respondent indicates an educational level of a college or greater				
**High parental education indicates an educational level of high school or greater				

Gene set enrichment analyses

To assess the association between each psychosocial factor and the entire set of previously implicated genes (1,854 transcripts from 1,305 unique genes), we conducted Global ANCOVA gene set enrichment tests (Table 3). In global analyses, major or lifetime discrimination and chronic burden were significantly associated with the CTRA gene set ($p=0.019$ and $p=0.047$ respectively). Loneliness and adult socioeconomic status were marginally significant ($p=0.066$ and 0.093 respectively). Associations between perceived stress, social support, and child socioeconomic status were not statistically significant.

Single transcript analyses

In the second approach, we employed multivariable linear regression analyses to identify the specific transcripts that were associated with the exposure (Table 3). At $p<0.05$, we found major or lifetime discrimination to be associated with the greatest number of transcripts (196) of any of the other social environmental factors. The list of transcripts significant at $p<0.05$ is given in Appendix 4. Generally, the greatest number of significant linear regression results was found for the exposures that were significant or marginally significant in global analyses (i.e. loneliness, major or lifetime discrimination, chronic burden, and adult socioeconomic status). However, no transcripts were significant after false discovery rate multiple testing correction.

Shrinkage and Variable selection

Elastic net regression was used to identify the gene transcripts most strongly associated with each social environmental factor. The number of transcripts selected for each factor is presented in Table 3 and the list of transcripts and corresponding gene name is available in Appendix 5. The correlation among selected genes is presented in Appendices 6-10, and a

comparison to linear regression findings is given in Appendices 11-17. The greatest number of transcripts was selected for loneliness (74) and the least were selected for perceived stress (0). In total, elastic net regression identified relationships for 156 unique transcripts across the seven social environmental factors. There was very little overlap in implicated transcripts across social environmental factors.

Table 3. Results of the Association between Social Environmental Factors with the CTRA Gene Set as Assessed via Global ANCOVA, Linear Regression, and Elastic Net				
Exposure	Global ANCOVA p-value	Linear regression p<0.05	Elastic Net	Overlap between linear regression (p<0.05) and elastic net
Loneliness	0.066	192	74	55
Discrimination	0.019	196	27	27
Perceived Stress	0.611	93	0	0
Chronic Burden	0.047	175	12	2
Social Support	0.662	70	8	7
Adult SES	0.093	117	46	41
Child SES	0.435	63	1	1
# unique transcripts across all social factors		738	156	140
# unique transcripts where Global ANCOVA p<0.10		584	150	128

Discussion

In this study, we examined associations between seven social environmental factors with gene expression using a large, multi-ethnic cohort and three analytic approaches. We focused on genes whose expression varied with social environmental factors in previous conserved transcriptional response to adversity (CTRA) studies. The CTRA is characterized by differential expression in immune system related genes (i.e. proinflammatory, antiviral response, antibody

synthesis) in response to exposure to unfavorable social environmental factors. Since these studies have been limited to small sample sizes and lacked replication in a human cohort, a study designed to reproduce the findings in the CTRA literature was warranted. Further, previous studies have varied in statistical approaches, cell type used from which DNA was extracted, and model organism which has limited the ability to make cross study comparisons. To our knowledge, the present study is the first large, epidemiologic investigation of previously published CTRA findings.

As there is not yet a consensus on the best method for assessing the association between social environmental exposures and gene expression, we took three different analytical approaches to examine relationships between our seven exposures of interest with expression of previously implicated genes and make comparisons between results. Our statistical approaches were designed to account for correlation among gene expression, hypothesized confounders, and to minimize the number of statistical tests. In the first approach, a self-contained gene set enrichment approach⁹⁰ was employed to assess the relationship between social environmental factors with our CTRA gene set. This global test informs whether any gene in an a priori defined set of genes is associated with an exposure. The Global ANCOVA test was the best suited gene set enrichment test for the research question since the analysis included both binary and continuous phenotypes, the availability original expression data, and information on potential confounders^{89,98}. Simulation studies indicate that the Global ANCOVA is a highly powered self-contained gene set enrichment approach especially when using the permutation based p-value that helps to account for the unknown correlation structure and heteroscedasticity among gene expression^{89,98-101}. A gene set approach is useful above single gene approaches for two main reasons. As a single test, concern for type I error is reduced for a gene set test compared to

single gene analyses approaches. Secondly, since genes exist in complex biological networks where small changes in gene expression typically occur across many genes, a gene set approach that evaluates groups of genes at a time better reflects the relationships between social environmental exposures and gene expression compared to single gene analysis techniques¹⁰². In the Global ANCOVA analyses, major or lifetime discrimination and chronic burden were found to be statistically significant ($p=0.019$ and $p=0.047$, respectively) while loneliness and adult socioeconomic status were found to be marginally significant ($p=0.066$ and $p=0.093$, respectively). Perceived stress, social support, and child SES were not significant.

It is noteworthy that major or lifetime discrimination was statistically significant in global analyses ($p=0.019$). Discrimination is an important psychosocial stressor that has been associated with a range of mental and physical health measures^{3,103}. Studies of the association between discrimination and protein level markers of inflammation have elicited mixed findings and indicate a complicated relationship. For example, a study by Lewis et. al found a significant relationship between everyday discrimination and CRP in a sample of older African American adults, while a study by Kershaw et. al did not find a significant association in a multiethnic sample^{49,50}. Interestingly, in the same study Kershaw et al. did find significant associations with the inflammatory biomarker interleukin-6 (IL-6) highlighting the complex nature of the discrimination-inflammation relationship⁵⁰. The differences between the Lewis and Kershaw studies may be in part due to sample composition^{49,50}. Further evidence indicates that relationship between discrimination and inflammation may be modified by other factors such as sex and body mass index^{50,51}.

Chronic burden was also significant in global analyses ($p=0.047$). Like discrimination, chronic burden has been found to associate with several health measures such as metabolic

syndrome and cardiovascular disease^{104,105}. The relationship between chronic burden and altered immune system functioning has been well studied in the literature. Chronic stress can influence immune system functioning either directly through the hypothalamic-pituitary-adrenal axis (HPA) and sympathetic-adrenal-medullary (SAM) system or indirectly through risk behaviors^{5,72,73,106}. There is some evidence that chronic stress affects the three processes that compose the conserved transcriptional response to adversity (i.e. inflammation, antibody synthesis, antiviral response)^{73,107,108}. For example, chronic stress was found to be positively associated with the inflammatory biomarkers IL-6 and CRP in the MESA cohort⁷³, a decreased ability to synthesize antibodies in response to an influenza vaccine among individuals caregiving for a sick loved one¹⁰⁷, and stress of a parental caregiver trended toward association of interferon production in children¹⁰⁸.

We followed up with the global analyses by conducting multivariable linear regression analyses. To account for the number and correlation of the tests, the false discovery rate multiple testing correction for correlated data was used⁹¹. However, after employing this multiple testing correction no statistically significant relationships were identified and raise the possibility that it may be an insufficient approach for correlated, high-dimensional gene expression analyses.

In the third analytic approach, elastic net penalized regression was used to select and jointly estimate the best set of CTRA gene transcripts associated with the social factors. Penalized regression techniques have been developed to enhance prediction ability and select important variables through the addition of a penalty term⁹⁴. The range of transcripts selected via elastic net across the seven social and psychosocial exposures was 0 for perceived stress to 74 for loneliness. Across the seven exposures, there were a total of 150 unique transcripts selected via elastic net corresponding to 146 genes. Expectedly, the gene list was enriched for

immune system related processes. Ten transcripts were selected for exactly two exposures (3 between adult socioeconomic status and loneliness, 3 between chronic burden and loneliness, 2 between discrimination and loneliness and 2 between support and loneliness). These 10 transcripts corresponded to the following genes: SLC6A6, VNN1, SLC22A4, IFI6, IL12RB1, SART3, DPP7, VPS41, SEMA4C, and SHMT1¹⁰⁹. These genes have a myriad of functions, some with known involvement in the immune response and others that do not. For example, IFI6 is involved in interferon gamma signaling, IL12RB1 is a receptor for interleukin 12, and DPP7 prevents apoptosis in lymphocytes. On the other hand, SLC6A6 and SLC22A4 are transporters, SART3 is a tumor rejection antigen, VPS41 is involved in organelle development, SEMA4C is involved in axon guidance, and SHMT1 is an enzyme with involvement in glycine, serine, and threonine metabolism¹⁰⁹. One transcript was selected for three exposures (loneliness, adult socioeconomic status, and social support). This transcript corresponds to the gene for isocitrate dehydrogenase (IDH1), a metabolic enzyme that catalyzes oxidative decarboxylation of isocitrate to 2-oxoglutarate¹⁰⁹. Mutations in this gene have been associated with different types of cancers, potentially through epigenetic modifications of both oncogenes and tumor suppressor genes¹¹⁰⁻¹¹².

Comparison between results of the three approaches

The three methods that we used in this paper to investigate the conserved transcriptional response to adversity differ in the interpretation of the conclusions for the research question. As a self-contained gene set enrichment test, we conclude from the Global ANCOVA that at least one transcript among the 1,854 investigated would be expected to be significantly associated with the social environmental factor. We therefore expected that linear regression findings would

indicate the strongest significant associations for the social environmental factors where the Global ANCOVA test result was significant. While none of the transcripts were significant after adjusting for multiple testing, we did find that the social environmental factors that were at least marginally significant in the Global ANCOVA (major or lifetime discrimination, chronic burden, loneliness, and adult SES) did yield more significant linear regression results at $p < 0.05$ compared to exposures not significant in global analyses (Table 3). The linear regression findings did suggestively map on to the elastic net findings as well. Among the transcripts that were selected via elastic net most were found to have p-values less than 0.05 in linear regression analyses. Similarly, the exposures with the most transcripts selected in elastic net (loneliness, discrimination, chronic burden, and adult SES) also had the lower p-values in Global ANCOVA analyses.

Comparison to previous studies

Among the seven investigated exposures, loneliness and adult socioeconomic status were assessed in previous CTRA studies. In 2007, Cole found there to be at least a 30% difference in expression levels of 209 transcripts (144 genes) between high loneliness and low loneliness individuals⁴⁸. Comparatively, using the same measure of loneliness, the UCLA Loneliness Scale, we found marginal significance with the CTRA gene set in global analysis, no significant association after multiple testing adjustment in linear regression analyses, and 74 transcripts to be selected via elastic net. These 74 transcripts mapped onto 74 unique genes, only 6 of which were implicated in the Cole study of loneliness (CCR2, CD79B, IL10RA, LGALS8, RGS1, and VNN1).

Findings from a study of the association between adult socioeconomic status and gene expression were also included in our CTRA gene set⁶¹. In a sample of 60 individuals, 387 genes were found to be differentially expressed by 5-year occupational status. In the present study, we used highest level of education as a measure of SES. Education is considered to be a stable measure of SES as education is mostly acquired by young adulthood and is impervious to variations in job status and income¹¹³. In the present the study, the 387 genes found to associate with occupational status were included in our CTRA gene set. Our results indicate marginal significance in global analyses, no transcripts significant after multiple testing in linear regression analyses, and 46 transcripts selected via elastic net. These 46 transcripts map onto 44 unique genes. Only 4 genes were implicated in the Powell study (ETV3, GBGT1, MCEMP1, SLC31A2). Comparing the present results for both loneliness and adult socioeconomic status to that of previous studies highlights the difficulty in replicating single genes across studies.

Differences in methodological approach likely account for part of the discrepancy between our findings and that of previous studies. Among the nine previous CTRA studies, there were two analytical approaches employed to assess relationships between social environmental factors and gene expression. Seven of these studies use the Patient Rule Induction Method (PRIM) machine learning algorithm to establish an a priori definition of differential expression (e.g. 30 % difference in expression between low lonely and high lonely individuals)¹¹⁴. However, this approach of assessing single gene transcripts has garnered much criticism as findings typically fail to replicate in separate studies. Several explanations for this have been proposed. The signal to noise ratio inherent in microarray technology makes it difficult to detect true effects¹⁰². Secondly, gene expression, especially that of genes within the same biological pathway, tends to be correlated and statistical measures are needed to account for the unknown

correlation structure¹⁰². Thirdly, many of these studies have lacked replication in separate human cohort which subjects reported findings to type I error¹⁰².

The remaining two CTRA studies used a contrast score based on an a priori selected group of genes that had been implicated in prior studies⁶³. The development of the contrast score is described in Fredrickson 2013⁶³. Briefly, fifty-three genes were selected to be part of the contrast score representing three processes: proinflammation (19 genes), type I interferon (31 genes) and antibody synthesis (3 genes)⁶³. Generalized linear model analysis was conducted to produce a point estimate for the association between the exposure and transcript abundance (\log_2 transformed). This point estimate was multiplied by a signed contrast coefficient where proinflammatory genes were multiplied by +1 and type IFN and antibody synthesis genes were multiplied by -1. A two-tailed, one sample t-test determined whether the resulting average association measure significantly differed from 0. Use of this score has been subject to critique in the literature^{115,116}. First, the criterion for selection of the 53 genes is unclear¹¹⁵. Secondly, in the original use of the score by Fredrickson and colleagues, the point estimates developed as the first step of generating the contrast score were based on independent generalized linear model analysis for each of the 53 genes with 17 predictor variables (including the phenotype of interest) despite only having a sample size of 80. Lastly, simply equally weighting genes, the contrast score does not account for the correlation among genes or potential for differential effect sizes¹¹⁵.

Difference in sample composition and cell type may also be a reason for the lack of overlap between the present and previous studies. While previous studies tended to be mostly White and from the same geographic region, we had representation from 3 racial/ethnic groups (Hispanic, non-Hispanic Black, non-Hispanic White) from different regions of the country. Our

study also had a relatively balanced male to female ratio, whereas previous studies tended to be mostly female. With a mean age of ~70 years, our sample was older than most of the previous studies. While it is important to understand how these relationships may exist across race and sex, the heterogeneity of our sample likely contributed to the lack of overlap. We did, however, statistically account for these variables in all models. Additionally, differences in cell type may help to account for the different findings between the present and previous studies. Gene expression was collected in monocytes in our study, whereas most of the previous studies used peripheral blood mononuclear cells (PBMCs).

Significance

The CTRA studies were an important stimulus to the emergence of the new field of human social genomics. This area has received widespread attention as it offers a biological mechanism (i.e. gene expression) that has the potential to partly explain the well documented relationships between the social environment and health. As a new area of research, it is important to be particularly meticulous in ensuring that methodological approaches are appropriate in accounting for the complex nature of gene expression within biological pathways. Without this level of rigor, inaccurate findings can pervade the literature. To address these types of questions, multivariable and multivariate statistical tests that can account for complex and potentially unknown correlation structures are needed to comprehensive capture the social environment and the ways in which it affects biological functioning. This epidemiologic study suggests that the current approaches in small studies (i.e. differential expression, contrast score) are insufficient in uncovering the replicable relationships between the social environment and

gene expression in a separate, diverse human cohort. Studies such as present one are necessary to move the field of human social genomics forward.

Strengths and limitations

Use of the Multiethnic Study of Atherosclerosis dataset helps to overcome the limitations of previous studies in the literature in the following ways: 1) the large sample size (n=1,264) compared to previous studies allows increased power to detect differences in gene expression 2) the comprehensive collection of social and psychosocial risk factor and gene expression data makes MESA uniquely suited to address the research question and 3) gene expression was collected in monocytes, an important leukocyte of the innate immune system that prior research indicates is particularly sensitive to social environment exposures^{63,117}.

While there were notable strengths to this study, we acknowledge that there were some important limitations. In the interest of systematically identifying social sensitive genes, we limited our literature search to articles which referred to the “conserved transcriptional response to adversity.” In doing this, we may have missed other genes in the literature whose expression is modified by social environment exposures but did not use this term in the published article. A second limitation is the variation in time over which data was collected. Social and psychosocial exposures were collected at various exams in the MESA study while gene expression was only collected in exam 5. Therefore, there may be time between collection of the exposure and outcome. To minimize the bias this may introduce, we included data from the most recent exam available. Further, since we only had gene expression data available for one time point, we cannot comment on the dynamic nature of the tested relationships.

CHAPTER III: Social Regulation of the Inflammation Related Gene Expression in the Multi-Ethnic Study of Atherosclerosis

Introduction

Epidemiologic evidence suggests that low-grade chronic inflammation is a risk factor for many common diseases. Of the 10 leading causes of death, there is evidence that inflammation is a risk factor for eight (i.e. cardiovascular disease, cancer, chronic lower respiratory diseases, stroke, Alzheimer's disease, diabetes, nephritis, and influenza and pneumonia)¹¹⁸. Prior studies have found that people with high levels of the inflammatory biomarker C-reactive protein (CRP) had 1.6 times the risk of ischemic heart disease and 1.3 times the risk of cerebrovascular disease compared to those with low levels³¹. Along the same lines, an elevated CRP level has been found to increase risk of mortality^{119,120}. Understanding the factors that affect inflammation and the processes by which inflammation leads to disease will be important in reducing disease burden.

Social and psychosocial factors have been found to influence multiple inflammatory pathways. Low socioeconomic status (SES), a fundamental cause of disease, has been found to inversely associate with levels of several inflammatory biomarkers including CRP, IL-6, fibrinogen, sICAM1 and ET-1^{2,38-40}. These associations have been consistent across different measures of SES (e.g. education, income, job status) and for SES in both adulthood and childhood^{29,41-43,71}. Similarly, exposures to adverse psychosocial stressors such as

discrimination⁴⁹ and chronic stress⁷³ have been found to associate with inflammatory proteins. These studies suggest that the chronic inflammation may be a mechanism whereby adverse social and psychosocial exposures affect disease risk.

The current understanding of the biological mechanisms through which social and psychosocial exposures “get under the skin” to affect inflammatory processes has been limited. Altered gene expression patterns may be one part of the complex biological cascade whereby exposure to social and psychosocial stress affects inflammation. Results from prior studies have documented differential expression of genes by exposure to social and psychosocial stress with an overrepresentation of these differences occurring in inflammation related genes. In a 2007 study, Cole and colleagues report differential expression of 209 gene transcripts by loneliness status where more lonely individuals overexpressed proinflammatory genes. Subsequent studies have found associations between low socioeconomic status⁶¹ caregiver stress⁶², well-being⁶³, positive vs. negative affect⁶⁴, and grief⁶⁵ with altered expression of inflammation related genes. This evidence suggests that the effect of social experiences on the inflammatory processes may be occurring at the gene level.

While promising, previous studies have suffered from important methodological limitations that present the need for deeper research. Most notably, the sample sizes were small, which makes statistical tests of the genome wide studies difficult. We expand upon this prior research in the present study by investigating associations between a range of social and psychosocial exposures and inflammatory gene expression using a larger, multiethnic cohort (n=1,264). Further, we focus on inflammatory and immune response genes specifically using the Gene Ontology database.

Methods

Study sample

The Multi-Ethnic Study of Atherosclerosis (MESA) was designed to investigate risk factors for the development and progression of subclinical cardiovascular disease⁷⁹. The baseline cohort was comprised of 6,814 adults aged 45-84 who self-identified as African-American, Chinese-American, Caucasian, or Hispanic and were free from clinical cardiovascular disease. Participants were recruited from six field sites across the United States between 2000 and 2002 (Baltimore, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles, California; New York, New York). Four follow-up examinations have been conducted with Exam 5, the fourth follow up ending in December 2011. The response rate has been excellent with 78% participants returning for Exam 5. Each exam consisted of a clinic visit where questionnaires on demographic, psychosocial, and lifestyle factors were administered, and physical assessments including the blood draw needed for genetic analyses were conducted. Gene expression was collected solely in Exam 5 on a random sample of 1,264 individuals from four of the MESA study sites⁸⁰. These individuals make up the study sample for the current study.

Social Environmental Factors

Adult Socioeconomic Status

Highest level of education was collected in Exam 1 and was dichotomized as a measure of adult socioeconomic status. Respondents were considered highly educated if they had obtained a college degree or higher.

Childhood Socioeconomic Status

Mother's and father's level of education were collected in Exam 2 to proxy childhood socioeconomic status. A parent was considered highly educated if he or she had achieved at least a high school degree. Childhood SES was dichotomized. The respondent was considered to have a high childhood SES if either parent had at least a high school degree.

Perceived Stress

Perceived stress was measured using Cohen's the 4-item Perceived Stress Scale (PSS) in Exam 5^{81,82}. The respondent was asked whether they felt: unable to control the important things in his or her life, confident about his or her ability to handle personal problems, things were going his or her way, or difficulties were piling up so high that he or she could not overcome them in the past month. Respondents answered on a five point Likert scale corresponding to the answer choices never, almost never, sometimes, fairly often, and very often. A summary measure was created by reverse coding the positive items (items 2 & 3) and summing the items such that a higher value indicated a higher level of perceived stress.

Major or Lifetime Discrimination

Major or lifetime discrimination was collected in Exam 1 and was adapted from the Detroit Area Study⁸³. Respondents were asked about whether they ever had been fired or denied a promotion, not hired for a job, treated unfairly by the police, discouraged by a teacher from continuing education, prevented from moving into a neighborhood, or neighbors have made their life difficult⁸³. A discrimination score was computed by summing the number of 'yes' responses. A higher score indicated higher exposure to major or lifetime discrimination.

Chronic Burden

Chronic burden was assessed using the Chronic Burden Scale in Exam 3⁸⁴. Participants were asked whether they had experienced ongoing problems in the following five domains: their own health, health of a loved one, job, relationship, financial problems. For affirmative responses, participants were subsequently asked whether this had been a problem for at least 6 months and whether this burden was not very stressful, moderately stressful, or very stressful. To estimate overall chronic burden, we summed the number of domains for which the respondent had experienced a chronic burden for at least 6 months and reported that it was either moderately or very stressful. A higher score indicated a higher level of chronic burden.

Social Support

Social support was measured in Exam 4 using a 4-item scale adapted from the MIDUS study⁸⁵. The questions asked how much friends and family can be relied upon for help with a serious problem, how much friends and family can be opened up to to talk about worries, how often friends and family make too many demands on the respondent, and how often friends and family let them down. Possible answer choices included: a lot, some, a little, not at all and were coded as 1, 2, 3, and 4 respectively. Positive items (items 1 & 2) were reverse coded and the sum of the 4 items was calculated to achieve an overall social support score where a higher score indicated greater social support.

Loneliness

Loneliness was measured in Exam 4 using a three item scale adapted from the UCLA Loneliness Scale⁸⁶. Participants were asked how often they lack companionship, feel left out, or isolated from others. Possible answer choices included: hardly ever, some of the time, and often and were coded as 1,2, and 3 respectively. A score was created by summing the three items. A higher score indicated a higher level of loneliness.

Covariates

Age, sex, and race/ethnicity were self-reported via questionnaire.

Gene expression

Gene expression data was collected from purified monocytes of 1,264 participants in MESA Exam 5. Detailed methods have been previously described⁸⁰. Briefly, peripheral blood mononuclear cells (PBMCs) were separated within two hours of blood draw using Vautainer CPT cell separation tubes. Monocytes were purified from the PBMCs using anti-CD14-coated magnetic beads. DNA and RNA were extracted using the AllPrep DNA/RNA Mini Kit. The resulting cRNA was hybridized to the Illumina HumanHT-12 v4 Expression BeadChip. This chip has probes for 47,231 transcripts (~31,000 genes), and is designed to assay 12 samples per chip. A stratified random sampling technique was used to assign samples to each chip avoid biases due to batch, chip, or position.

Preprocessing and quality controls steps were conducted to ensure accurate quantification of the gene expression data. Illumina's proprietary software Genome Studio was used to correct for local background. The remaining preprocessing steps were conducted using Bioconductor

packages in R. Since the bead chip has multiple copies of each probe, a bead-type summarization (mean and variance) was produced for each transcripts using the *beadarray* package⁸⁷. The negative controls on the array were used to compute the detection p-value. The *limma* package was used for background correction, quantile normalization, log₂ transformation, and removal of control probes⁸⁸. Quality control criteria for elimination of a transcript included: ‘detected’ expression levels in <10% of MESA samples (detection p-value cutoff=0.01), probes that contain a SNP, probes with low variance across samples (<10th percentile), and probe overlap with a non-unique region.

Gene Set Generation

Gene Ontology is a bioinformatics database that uses findings from over 100,000 peer reviewed scientific papers to annotate the functions of gene products. Each gene is categorized by an associated biological process, cellular component, and/or molecular function. In the present analysis, we developed gene sets based on four inflammation related biological processes: chronic inflammatory response (GO: 0002544), inflammatory response (GO: 0006964), regulation of inflammatory response (GO:0050727), and the immune response (GO:0006955). The list of genes included in each investigated biological process is given in Appendix 17. Our genes sets were based on genes that were represented on the Illumina HumanHT-12 v4 Expression BeadChip used in the MESA study. For cases where a gene matched to more than one transcript, we included all transcripts in the analyses. Our final gene set consisted of 20 gene transcripts for chronic inflammation (corresponding to how many unique genes), 438 for the inflammatory response, 1251 for the immune response, and 192 for

regulation of the inflammatory response. There were some overlapping gene transcripts among the four categories (Appendix 18).

Table 4. Number of Overlapping Transcripts among Gene Ontology Biological Processes				
	Chronic Inflammation (k=20)	Inflammatory Response (k=438)	Immune Response (k=1251)	Regulation of Inflammatory Response (k=192)
Chronic Inflammation (k=20)	-	20	20	11
Inflammatory Response (k=438)		-	282	191
Immune Response (k=1251)			-	142
Regulation of Inflammatory Response (k=192)				-
k indicates the number of transcripts in the gene set				

Statistical Analyses:

To statistically test the association between the social environmental exposures and gene expression, we used a two part analytic approach. In the first part, we assessed the association between each of the seven social environmental factors with the each of the four gene sets using a global analysis (Global ANCOVA) for a total of independent 28 tests. The Global ANCOVA is self-contained gene set enrichment analysis that has the null hypothesis that no gene in a set of genes are associated with a given exposure. This method is described in detail in Hummel 2008⁸⁹.

Briefly, given expression of p genes in n individuals with d covariates, this multivariate model is written as:

$$\tilde{X} = \begin{pmatrix} \tilde{x}^{(1)} \\ \vdots \\ \tilde{x}^{(p)} \end{pmatrix} = \tilde{C} \cdot \tilde{\beta} + \mathcal{E} = \begin{pmatrix} C & 0 & 0 \\ 0 & \ddots & 0 \\ 0 & 0 & C \end{pmatrix} \cdot \begin{pmatrix} \tilde{\beta}_1^t \\ \vdots \\ \tilde{\beta}_p^t \end{pmatrix} + \begin{pmatrix} \tilde{\xi}^{(1)} \\ \vdots \\ \tilde{\xi}^{(p)} \end{pmatrix} \quad (2)$$

The gene expression values of the entire gene set (\tilde{X}), is expressed as an np column vector, representing gene expression of each gene for each individual. It is a function of the product \tilde{C} and $\tilde{\beta}$ plus \mathcal{E} , where \tilde{C} is a $np \times (d+1)p$ sized diagonal block matrix representing the covariate information for each individual, $\tilde{\beta}$ is a column vector of size $(d+1)p$ which quantifies the relationship of each exposure and covariate on the expression of each gene in the gene set, and \mathcal{E} is np sized column vector which accounts for the noise for each individual for each gene in the gene set. For each Global ANCOVA test in our study, there were 1,264 participants, one main psychosocial exposure of interest (each investigated in a separate model), and 4 additional covariates (age, sex, study site, and race/ethnicity). A permutation based approximation approach was used in order to assess significance of the Global ANCOVA tests using the strategy described in Hummel 2008⁸⁹.

In the second part of the approach we used multivariable linear regression and elastic net penalized regression techniques to identify which genes were driving significant Global ANCOVA findings. We limited these analyses to only instances where the social environmental factor was significantly associated with the gene set in Global ANCOVA analyses. This strategy reduces total number of tests and lessens the concern for Type 1 error.

The multivariable linear regression model is written as:

$$\tilde{x}^{(i)} = C \cdot \tilde{\beta}_i^t + \xi^{(i)} = \begin{pmatrix} 1 & c_{11} & \cdots & c_{1d} \\ \vdots & \vdots & & \vdots \\ 1 & c_{n1} & \cdots & c_{nd} \end{pmatrix} \cdot \begin{pmatrix} \beta_{i0} \\ \vdots \\ \beta_{id} \end{pmatrix} + \begin{pmatrix} \xi_1^i \\ \vdots \\ \xi_n^i \end{pmatrix}$$

where the outcome, the expression for the i th gene transcript, is expressed as $\tilde{x}^{(i)}$, n is the number of individuals, and d is the number of covariates. C is a matrix of size $n \times (d+1)$ which represents the covariate values for each individual. The gene specific mean expression is quantified by β_{i0} . The regression coefficient vector, $\tilde{\beta}_i^t$, reflects the influence of each covariate on expression of the i^{th} gene transcript. The residual for each individual is given by the $\xi^{(i)}$ vector.

Penalized regression methods, such as elastic net, subject the minimization the residual sum of squares (i.e. $\text{argmin}_{\beta} |y - X\beta|^2$) used in OLS regression to additional penalties to improve prediction and interpretation above multivariable linear regression^{93,94}. Detailed methods of the elastic net are given in Zou and Hastie 2005⁹⁴. As a hybrid of the LASSO (least absolute shrinkage and selection operator) and ridge regression penalties, elastic net exploits their ability to conduct variable selection and adjustment for correlation among predictors respectively.

For n individuals and p gene transcript expressions, let y_j be the value of the psychosocial factor for person j , x_{ji} be the gene expression value for person j for gene i , and β_i be the beta estimate corresponding gene i . The elastic net penalty is given by:

$$\min_{\beta_i} \left[\sum_{j=1}^n \left(y_j - \sum_{i=1}^p x_{ji} \beta_i \right)^2 + \lambda_2 \sum_{i=1}^p \beta_i^2 + \lambda_1 \sum_{i=1}^p |\beta_i| \right]$$

The two tuning parameters λ_1 and λ_2 control the trade-off between bias and variance⁹⁵ and were selected using cross validation techniques.

Results

In the sample of 1,264 MESA participants with available gene expression data, the mean age was ~70 years and 51% of the sample was female. There were three racial/ethnic groups represented where 272 participants were non-Hispanic Black, 402 were Hispanic, and 590 were non-Hispanic White. Participants were recruited from four of the six MESA study sites: Forsythe County, North Carolina (49 participants), New York, New York (424 participants) Baltimore, Maryland (317 participants) and St. Paul, Minnesota (474 participants). Non-Hispanic Blacks were recruited mostly from the New York and Baltimore sites. Hispanics were recruited from the New York and Rochester sites. Whites were recruited from all four sites. For the socioeconomic variables, 33% of the total sample of adults had finished a level of schooling equal to a college degree or higher. Slightly over half of the sample (56%) had either a mother or father achieve at least a high school degree. Whites (49%) were more likely to have obtained a college degree than Blacks (27%) or Hispanics (13%) ($p < 0.001$ and $p < 0.001$ respectively). Blacks were significantly more likely to have a college degree than Hispanics ($p < 0.0001$). The same pattern was observed for childhood SES where Whites (71%) were more likely to have at least one parent with a high school degree compared to Blacks (55%) or Hispanics (31%) ($p < 0.001$ and $p < 0.001$ respectively), and Blacks were significantly more likely to have a parent with a high school degree than Hispanics ($p < 0.0001$). For most of the social and psychosocial exposures, the median scores were close to the minimum score indicating that most of the sample reported low

levels of these exposures. There were not significant racial/ethnic differences in these scores except for major or lifetime discrimination ($p < 0.001$). Tukey's honest significance test indicated that Blacks reported higher levels than Whites ($p < 0.001$) or Hispanics ($p < 0.001$). There was not a significant difference between Whites and Hispanics ($p = 0.16$). The correlation among the psychosocial exposures is presented in Appendix 3. The most correlated exposures were loneliness and chronic burden ($r = 0.36$) and the least correlated exposures were major or life discrimination and perceived stress ($r = 0.05$). These relationships were mirrored across race/ethnicity.

Table 5. Characteristics of the MESA Study Sample with Gene Expression Data				
	Total sample (n=1264)	Non-Hispanic Black (n=272)	Hispanics (n=402)	Non-Hispanic White (n=590)
Demographics				
Age mean (SD)	69.6 (9.4)	69.6(9.0)	68.4(9.3)	70.2(9.5)
Sex (% female)	51	60	50	48%
Study Site (n,%)				
Forsythe County, North Carolina	49(4%)	1(0.3%)	0	48 (40%)
New York, New York	424(34%)	131(48%)	209 (52%)	84 (14%)
Baltimore, Maryland	317(25%)	140(51%)	0	177 (30%)
Rochester, Minnesota	474(38%)	0	193 (48%)	281 (48%)
Total	1264(100%)	272 (100%)	402 (100%)	590 (100%)
Socioeconomic Status				
High education- respondent*	33%	27%	13%	49%
High education- either parent**	56%	55%	31%	71%
Psychosocial factors Median, (Interquartile Range)				
Loneliness	3 (3-5)	3(3-4)	4(3-5)	3 (3-5)
Major or Lifetime Discrimination	0 (0-1)	1(0-2)	0(0-1)	0 (0-1)
Chronic Burden	1 (0-2)	1(0-2)	0(0-2)	1 (0-2)
Perceived Stress	8 (5-10)	8(5-10)	8(5-10)	7 (6-10)
Social support	9 (8-10)	9(8-10)	9(8-10)	9 (8-10)
*High education for respondent indicates an educational level of a college or greater				
**High parental education indicates an educational level of high school or greater				

Gene set enrichment analyses

For each of the seven social and psychosocial factors, we first conducted Global ANCOVA gene set enrichment tests for each of the four biological processes for a total of 28 tests. We found three (loneliness, discrimination, chronic burden) of the investigated exposures to be significantly associated with the chronic inflammation gene set at $p < 0.05$ and an additional two exposures (perceived stress and social support) to be marginally significant at $p < 0.10$ (Table 6). Major or lifetime discrimination was significantly associated with the other three gene sets as well (i.e. inflammatory response, immune response, regulation of inflammatory response). No other social environmental factors were associated with these gene sets.

	Chronic Inflammation (n=20)	Inflammatory Response (n=438)	Immune Response (n=1251)	Regulation of Inflammatory Response (n=192)
Loneliness	0.003	0.229	0.227	0.173
Discrimination	0.045	0.029	0.041	0.025
Perceived Stress	0.092	0.123	0.179	0.181
Chronic Burden	0.002	0.188	0.135	0.312
Social Support	0.053	0.571	0.599	0.344
Adult SES	0.110	0.300	0.179	0.076
Childhood SES	0.403	0.548	0.506	0.467

Multivariable linear regression

Multivariable linear regression was used to identify which gene transcripts were driving the significant associations from the Global ANCOVA analyses. Based on the Global ANCOVA

results, we conducted these analyses for each loneliness, major or lifetime discrimination, and chronic burden and each of the 20 transcripts of the chronic inflammation gene set. For loneliness and discrimination, we found significant associations at $p < 0.05$ for 5 and 4 transcripts respectively (Table 7). Three of these transcripts corresponding to two unique genes (CX3CR1 and VNN1) overlapped between exposures. Despite significant Global ANCOVA results for chronic burden with the chronic inflammation gene set, we surprisingly did not detect significant results with any of the 20 transcripts in the linear regression single gene analyses.

Since there were significant findings for major or lifetime discrimination and the other three gene sets in Global ANCOVA analyses, we also examined the regression relationship between major or lifetime discrimination and each of the 438 gene transcripts of the inflammatory response, 1251 gene transcripts of the immune response, and 192 gene transcripts of the regulation of the inflammatory response. For inflammatory response, immune response, and regulation of the inflammatory response, we found 12%, 11%, and 13.5% of investigated transcripts to be significant respectively, which exceeds the 5% that would be expected by chance alone (Appendix 19). However, none of the associations persisted after FDR multiple testing adjustment.

Elastic Net

Elastic net regression was also used to identify the gene transcripts most strongly associated with each social environmental factor. The number of transcripts selected via elastic net ranged from 3-42 dependent upon the exposure and gene set. The greatest number of transcripts selected was for discrimination and the immune response gene set, which also has the largest number of transcripts in the set (1251). The greatest proportion of transcripts was selected

for loneliness with the chronic inflammation gene set (12/20=0.60). The number of transcripts selected for each social environmental factor and gene set pair is presented in Table 7, the list of transcripts and corresponding gene name is available in Appendix 20, the correlation among selected genes in Appendices 21-26, and a comparison to linear regression findings in Appendices 27-32.

Overlap between linear regression and elastic net penalized regression findings

We used two complementary methods (i.e. multivariable linear regression and elastic net penalized regression) to identify the transcripts driving the significant association from the global analyses and assessed the overlap between these findings. The results indicate that transcripts that were selected via elastic net analyses were also likely to be significant at $p < 0.05$ in linear regression analyses across exposures and gene sets except for the chronic burden exposure with the chronic inflammation gene set where there were no significant associations in linear regression analyses (Table 7). In total, of the 53 unique gene transcripts that were selected in elastic net, 43 (81%) were also found to be significant in linear regression analyses at $p < 0.05$.

Table 7. Results from Linear Regression and Elastic Net Analyses				
Exposure	Gene Set	# of transcripts significant at p<0.05	# transcripts selected via elastic net	Overlap between linear regression and elastic net findings
Loneliness	Chronic Inflammation (k=20)	5	12	4
Chronic Burden	Chronic Inflammation (k=20)	0	8	0
Discrimination	Chronic Inflammation (k=20)	4	3	3
Discrimination	Inflammatory Response(k=438)	53	17	17
Discrimination	Immune Response (k=1251)	136	42	36
Discrimination	Regulation of the Inflammatory Response (k=192)	26	15	15
Overlap of total unique transcripts	Total: 1407	153	53	43
k indicates the number of transcripts in the gene set				

Discussion

While longstanding evidence indicates that exposure to stressful experiences is associated with an impaired immune response and susceptibility to disease, we have yet to fully understand how external social experiences are internalized to disrupt normal biological functioning. In this study, we investigated the association between a range of social environmental factors with expression of immune system genes, with a particular focus on genes involved in the inflammatory response. The results of the present study provide evidence that social environmental factors may be influencing chronic inflammation via gene level processes and

highlights a biological mechanism (i.e. gene expression) whereby the social environment gets “under the skin” to affect an important risk factor for several high burden diseases. Better understanding of the mechanisms through which these social environmental factors act biologically will enable the development of more precise prevention and treatment approaches.

We took a two part approach to investigate the association between social environmental factors with gene expression. We first conducted global analyses to assess the association for each social environmental factor with each of the biological gene sets. In global analyses, we found significant associations between three of the seven investigated social environmental factors with expression of chronic inflammation gene set. Significant associations were detected for loneliness, major or lifetime discrimination, and chronic burden ($p=0.003$, $p=0.045$, $p=0.002$, respectively). We also found marginally significant relationships for perceived stress and social support ($p=0.092$, $p=0.053$, respectively). We did not find significant associations for adult or childhood socioeconomic status. In the second part of the approach, we used multivariable linear regression and elastic net penalized regression to identify which transcripts were the drivers of the significant associations in global analyses. These results ranged dependent upon exposure and set of genes.

Our finding that loneliness was significantly associated with chronic inflammation is consistent with the literature. Loneliness is a psychosocial stressor characterized by a subjective stressful feeling due to unmet needs of social connections. It has been found to associate with several chronic diseases (e.g. coronary heart disease, stroke) for which chronic inflammation is a known risk factor^{46,47}. While there have been several studies investigating the relationship between loneliness and inflammation at a protein level^{28,121,122}, studies on loneliness and gene expression have been limited. The small, extant literature suggests that loneliness does associate

with biological functioning at the gene level, and the patterns may be altered by behavioral treatments^{48,123}. In the previously mentioned study by Cole and colleagues, 144 genes were found to be differentially expressed in a genome wide study between high lonely and low lonely individuals. The same measure of loneliness used in the Cole study, the UCLA loneliness scale, was also used in the present study. Of these 144 genes, 3 (IL1B, TNFAIP3, VNN1) were in the chronic inflammatory gene set. To identify which gene transcripts in the chronic inflammatory gene set were significantly associated with loneliness, we followed up the global analyses with linear regression and elastic net penalized regression. In linear regression analyses, we found 5 of the 20 transcripts to be significantly associated with loneliness at $p < 0.05$. However, none were statistically significant after false discovery rate multiple testing correction. Four of these 5 transcripts were among the 12 transcripts selected in elastic net, suggesting that a relationship may exist between loneliness and expression of these transcripts that the linear regression approach may have been ill powered to adequately detect. The 12 transcripts selected via elastic net correspond to 12 unique genes (IDO1, VNN1, THBS1, CAMP, IL1RN, CEBPB, ADORA2B, UNC13D, IL1B, LTA, CX3CR1, CCL5). These genes have a range of functions in the chronic inflammatory biological process. For example, CX3CR1 plays a role in leukocyte adhesion and migration, IL1B produces a protein product that is involved in cell proliferation, differentiation, and apoptosis, and THBS1 is a glycoprotein mediator of cell-to-cell and cell-to-matrix interactions¹⁰⁹. The present study builds upon the existing literature by providing evidence of associations between loneliness and gene expression in chronic inflammatory response genes while addressing some of the important limitations of previous work (e.g. small sample size, differing cell types).

Interestingly, the other two social environmental factors that were significant in global analyses- chronic burden and discrimination - were also implicated in the Aim 1 global analyses. As stated in Aim 1, chronic burden has been associated with altered immune functioning including inflammation. This relationship is hypothesized to act directly through the hypothalamic-pituitary-adrenal axis (HPA) and sympathetic-adrenal-medullary (SAM) system or indirectly through risk behaviors^{5,73,106}. In the MESA cohort, chronic burden has been found to significantly associate with inflammation as measured by IL-6 and CRP⁷³. In the present study, we followed up the global analyses to identify which genes specifically were associated with chronic burden. No gene transcripts were significant in linear regression analyses at $p < 0.05$ and 8 transcripts were selected in elastic net corresponding to 8 unique genes (VNN1, THBS1, IL1RN, ADORA2B, LTA, CX3CR1, CCL5, IDO1). Interestingly, all 8 of these genes were also selected in the elastic net analyses assessing the association between loneliness and the chronic inflammatory genes.

In global analyses, major or lifetime discrimination was the only measure that yielded significant findings across all four biological processes investigated (i.e. chronic inflammation, inflammatory response, immune response, and regulation of inflammatory processes). Discrimination, characterized by differential treatment of certain groups, has been associated with increased risk of morbidity and mortality¹⁰³. Further, while it has consistently been found to adversely affect mental health outcomes, the literature on physical health outcomes is less conclusive with some studies showing a relationship and others showing no relationship^{3,103,124}. In the MESA cohort, there has been mixed findings on the relationship between discrimination and inflammation at the protein level. A study by Kershaw et. al found discrimination to be significantly associated with the inflammatory biomarker IL-6, but interestingly no significant

association was detected for CRP⁵⁰. These findings were influenced by sex and body mass index⁵⁰. Although the genes encoding CRP and IL-6 were not part of the chronic inflammatory gene set, we were able to provide evidence that discrimination affects chronic inflammatory processes at a gene level in global analyses. In linear regression analyses, four transcripts were significant at $p < 0.05$, but like the relationships for loneliness and chronic burden there were no significant transcripts after multiple testing adjustment. Three transcripts were selected via elastic net (CX3CR1, VNN1, CHAMP1) all of which were significant at $p < 0.05$ in linear regression analyses. Two of these three genes (i.e. CX3CR1, VNN1) were also selected in elastic net analyses for loneliness and chronic burden.

Given that the Global ANCOVA test is designed to assess whether any gene transcript is associated with the exposure, the finding that loneliness and chronic burden were associated with the chronic inflammation gene set but not the other sets was unexpected since there was substantial overlap between the 20 transcripts that composed the chronic inflammatory gene set and the other gene sets (Table 4). It is likely that this is due to the common signal-to-noise ratio issue inherent to microarray studies¹⁰². With only 20 transcripts and a more focused biological process, we would expect the chronic inflammatory gene set to be less susceptible to this issue compared to the other three investigated biological processes.

This study has notable strengths which help to move the field of human social genomics forward. One strength of this current study is the sample size of 1,264 participants. Similar types of studies have been limited by very small sample sizes^{48,61–65,72,77,125}. The use of the MESA cohort makes the present study one of the largest human social genomics studies to date. Secondly, gene expression was measured in monocytes in MESA. Since gene expression is cell type specific, it is important to have it measured in a cell type that is known to play a role in

inflammation. Monocytes are phagocytic leukocytes of the innate immune response, and evidence suggests that even among the various types of white blood cells that are part of the inflammatory response, monocytes, along with dendritic cells, are most susceptible to changes in gene expression with exposure to an adverse social environment¹¹⁷. Third, our statistical approach was designed to account to the complexity of testing correlated variables. By using the permutation based approximation of the F distribution in the Global ANCOVA test, we accounted for the gene expressions being neither independent nor homoscedastic⁸⁹. In linear regression analyses, we employed false discovery rate multiple testing correction for correlated data to address issues related to the total number of statistical tests being conducted and the correlated nature of the gene expression outcomes. Elastic net, which was used as a variable selection procedure, is designed select groups of correlated genes.

This study is not without limitations. Most notably, we did not have a replication sample to verify the association between our implicated social environmental factors and gene expression. Replication reduces the concern for Type 1 error. Secondly, the variables -the social environmental factors, gene expression, and covariates-were collected at different times throughout the 5 MESA exams. Because gene expression values can be transient across time, these values collected in Exam 5 may be different from the prior exams when most of the social environmental factors were collected.

CHAPTER IV: Social environmental contributors to racial/ethnic differences in gene expression

Introduction

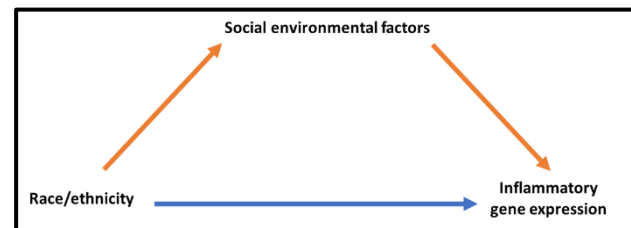
Chronic inflammation has emerged as an important risk factor for eight of the top ten leading causes of death in the United States^{126–128}. These diseases include cardiovascular disease, cancer, chronic lower respiratory disease, stroke, Alzheimer’s disease, diabetes, kidney disease, and influenza/pneumonia¹²⁹. Racial/ethnic disparities have been observed in seven of these diseases, where Blacks and Hispanics are disproportionately affected¹⁰³. This finding is consistent with studies that have demonstrated that Blacks and Hispanics are more likely to have adverse inflammatory profiles compared to non-Hispanic Whites^{41,57}. A wide range of evidence suggests that racially/ethnically patterned social and psychosocial stressors may be an important pathway through which race/ethnicity is associated with increased inflammation. Blacks and Hispanics have consistently reported higher levels of stress in terms of frequency, longevity, and reactivity²⁵. Social and psychosocial stress exposures have in turn been shown to have adverse effects on health including increased inflammation^{49,73,130}. Although a difference in exposure to social stressors has been hypothesized to influence the relationship between race/ethnicity and inflammation, the ways in which social environmental factors “get under the skin” to affect the biological process of inflammation could reflect many different types of mechanisms.

One mechanism through which race/ethnicity may be associated with inflammation is through the influence of genetic and environmental factors on gene expression. Racial

differences in gene expression have been observed in many studies^{67,68}. For example, using samples from HapMap, Storey et. al and Zhang et al. found gene expression differences between Whites and Yoruba Nigerians in 17% and 34% of genes respectively^{67,68}. Storey found that among differentially expressed genes, there was an enrichment of inflammation associated genes. Contemporaneously, exposure to adverse social exposures including loneliness, low socioeconomic status⁶¹ caregiver stress⁶², well-being⁶³, positive vs. negative affect⁶⁴, and grief⁶⁵ have also been found to be associated with variation in the expression of inflammatory genes. Given the expensive nature of gene expression testing, most studies of social environmental factor influences on gene expression have been small (n<250 participants).

In the present study, we use data from the Multi-Ethnic Study of Atherosclerosis to examine 1) racial/ethnic differences in a range of social environmental factors (e.g. childhood socioeconomic status, adult socioeconomic status, loneliness, major or lifetime discrimination, chronic burden, perceived stress, and social support 2) racial/ethnic differences in expression of inflammatory

Figure 1
Hypothesized Relationship between Race/Ethnicity and Inflammatory and Immune Response Genes



and immune response related genes and 3) the extent to which the social environmental exposures explain the association between race/ethnicity and gene expression. While it has been well documented that racial/ethnic differences in gene expression exist and an emerging literature in the field of human social genomics implicates associations between social environmental factors and gene expression as well, a dearth of research has investigated the influence of racially/ethnically patterned social environmental exposures on the relationship between race/ethnicity and gene expression. As racial/ethnic differences in gene expression are

due to both inherited DNA sequence and environmental exposures in unknown quantities, this study helps to fill an important gap by investigating the contribution of the social environmental factors on this relationship.

Methods

Study sample

The Multi-Ethnic Study of Atherosclerosis (MESA) was designed to investigate risk factors for the development and progression of subclinical cardiovascular disease⁷⁹. The baseline cohort was comprised of 6,814 adults aged 45-84 who self-identified as African-American, Chinese-American, White, or Hispanic and were free from clinical cardiovascular disease. Participants were recruited from six field sites across the United States between 2000 and 2002 (Baltimore, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles, California; New York, New York). Four follow-up examinations have been conducted with Exam 5, the fourth follow up ending in December 2011. The response rate has been excellent with 78% participants returning for Exam 5. Each exam consisted of a clinic visit where questionnaires on demographic, psychosocial, and lifestyle factors were administered, and physical assessments including the blood draw needed for genetic analyses were conducted. Gene expression was collected solely in Exam 5 on a random sample of 1,264 individuals from four of the MESA study sites⁸⁰. These individuals make up the study sample for the current study.

Social environmental factors

Adult Socioeconomic Status

Highest level of education was collected in Exam 1 and was dichotomized as a measure of adult socioeconomic status. Respondents were considered highly educated if they had obtained a college degree or higher.

Childhood Socioeconomic Status

Mother's and father's level of education were collected in Exam 2 to proxy childhood socioeconomic status. A parent was considered highly educated if he or she had achieved at least a high school degree. Childhood SES was dichotomized. The respondent was considered to have a high childhood SES if either parent had at least a high school degree.

Perceived Stress

Perceived stress was measured using Cohen's the 4-item Perceived Stress Scale (PSS) in Exam 5^{81,82}. The respondent was asked whether they felt: unable to control the important things in his or her life, confident about his or her ability to handle personal problems, things were going his or her way, or difficulties were piling up so high that he or she could not overcome them in the past month. Respondents answered on a five point Likert scale corresponding to the answer choices never, almost never, sometimes, fairly often, and very often. A summary measure was created by reverse coding the positive items (items 2 & 3) and summing the items such that a higher value indicated a higher level of perceived stress.

Major or Lifetime Discrimination

Major or lifetime discrimination was collected in Exam 1 and was adapted from the Detroit Area Study⁸³. Respondents were asked about whether they ever had been fired or denied a promotion, not hired for a job, treated unfairly by the police, discouraged by a teacher from continuing education, prevented from moving into a neighborhood, or neighbors have made their life difficult⁸³. A discrimination score was computed by summing the number of ‘yes’ responses. A higher score indicated higher exposure to major or lifetime discrimination.

Chronic Burden

Chronic burden was assessed using the Chronic Burden Scale in Exam 3⁸⁴. Participants were asked whether they had experienced ongoing problems in the following five domains: their own health, health of a loved one, job, relationship, financial problems. For affirmative responses, participants were subsequently asked whether this had been a problem for at least 6 months and whether this burden was not very stressful, moderately stressful, or very stressful. To estimate overall chronic burden, we summed the number of domains for which the respondent had experienced a chronic burden for at least 6 months and reported that it was either moderately or very stressful. A higher score indicated a higher level of chronic burden.

Social Support

Social support was measured in Exam 4 using a 4-item scale adapted from the Midlife in the United States (MIDUS) study⁸⁵. The questions asked how much friends and family can be relied upon for help with a serious problem, how much friends and family can be opened up to talk about worries, how often friends and family make too many demands on the respondent, and how often friends and family let them down. Possible answer choices included: a lot, some,

a little, not at all and were coded as 1,2,3, and 4 respectively. Positive items (items 1 & 2) were reverse coded and the sum of the 4 items was calculated to achieve an overall social support score where a higher score indicated greater social support.

Loneliness

Loneliness was measured in Exam 4 using a three item scale adapted from the UCLA Loneliness Scale⁸⁶. Participants were asked how often they lack companionship, feel left out, or isolated from others. Possible answer choices included: hardly ever, some of the time, and often and were coded as 1,2, and 3 respectively. A score was created by summing the three items. A higher score indicated a higher level of loneliness.

Covariates:

Age, sex, and race/ethnicity were self-reported via questionnaire.

Gene Expression

Gene expression data was collected from purified monocytes of 1,264 participants in MESA Exam 5. Detailed methods have been previously described⁸⁰. Briefly, peripheral blood mononuclear cells (PBMCs) were separated within two hours of blood draw using Vautainer CPT cell separation tubes. Monocytes were purified from the PBMCs using anti-CD14-coated magnetic beads. DNA and RNA were extracted using the AllPrep DNA/RNA Mini Kit. The resulting cRNA was hybridized to the Illumina HumanHT-12 v4 Expression BeadChip. This chip has probes for 47,231 transcripts (~31,000 genes), and is designed to assay 12 samples per

chip. A stratified random sampling technique was used to assign samples to each chip to avoid biases due to batch, chip, or position.

Preprocessing and quality controls steps were conducted to ensure accurate quantification of the gene expression data. Illumina's proprietary software Genome Studio was used to correct for local background. The remaining preprocessing steps were conducted using Bioconductor packages in R. Since the bead chip has multiple copies of each probe, a bead-type summarization (mean and variance) was produced for each transcripts using the *beadarray* package⁸⁷. The negative controls on the array were used to compute the detection p-value. The *limma* package was used for background correction, quantile normalization, log₂ transformation, and removal of control probes⁸⁸. Quality control criteria for elimination of a transcript included: 'detected' expression levels in <10% of MESA samples (detection p-value cut-off=0.01), probes that contain a SNP, probes with low variance across samples (<10th percentile), and probe overlap with a non-unique region.

Gene Set Definition

Four gene sets derived from the Gene Ontology database were investigated: chronic inflammatory response (GO: 0002544, 20 transcripts), inflammatory response (GO:0006964, 438 transcripts), regulation of inflammatory response (GO:0050727, 192 transcripts), and the immune response (GO:0006955, 1251 transcripts). The number of overlapping transcripts between the biological processes is presented below (Table 8) and the gene names are presented in Appendix 18. The cumulative total number of gene transcripts examined across these four biological processes was 1407 transcripts.

Table 8. Number of Overlapping Transcripts among Gene Ontology Biological Processes				
	Chronic Inflammation (k=20)	Inflammatory Response (k=438)	Immune Response (k=1251)	Regulation of Inflammatory Response (k=192)
Chronic Inflammation (k=20)	-	20	20	11
Inflammatory Response (k=438)		-	282	191
Immune Response (k=1251)			-	142
Regulation of Inflammatory Response (k=192)				-
k indicates the number of transcripts in the gene set				

Statistical Analyses:

Racial differences in social environmental factors

Bivariate tests were conducted to determine the social environmental factors for which racial differences existed. Chi-square was used for categorical variables (childhood and adult socioeconomic status), and analysis of variance (ANOVA) was used for continuous variables (loneliness, major or lifetime discrimination, chronic burden, perceived stress, and social support).

Race differences in transcript expression

Multivariable linear regression was employed to identify transcripts significantly associated with race/ethnicity. Bonferroni correction was employed to adjust for multiple comparisons. This adjustment tightly controls the family wise error rate (i.e. the probability of Type 1 error). It is employed by setting the p-value to the value of 0.05 divided by the number of tests. We did this individually for each of the five gene sets. For the chronic inflammatory response (20 transcripts), inflammatory response (438 transcripts), regulation of inflammatory (192 transcripts) response, and immune response (1251 transcripts) gene sets, p-values were adjusted to 2.5×10^{-3} , 1.1×10^{-4} , 2.6×10^{-4} , and 4.0×10^{-5} , respectively.

Race differences in transcript expression after accounting for social environmental factors

Three linear regression models were used to investigate the extent to which the social environmental factor explained the effect of race/ethnicity on gene expression.

Model 1: $geneexpression = \alpha_1 + \beta_1 race/ethnicity$

Model 2: $geneexpression = \alpha_2 + \beta_2 race/ethnicity + \beta_3 socialfactor$

Model 3: $geneexpression = \alpha_3 + \beta_4 socialfactor$

In all models, gene expression was adjusted for age, sex, and chip prior to analyses, and the residuals were used.

The effect of the social environmental factor on the association between race/ethnicity and gene expression was assessed in two ways. First, the absolute value of the percent change in

beta estimate from the race/ethnicity only model (Model 1) to the model with race/ethnicity adjusted for the social factor (Model 2) was examined using:

$$\left| \frac{\beta_1 - \beta_2}{\beta_1} \right| * 100 = \Delta \textit{percent in effect}.$$

Secondly, we compared the proportion in variability (R^2) in gene expression that can be attributed to race/ethnicity with and without accounting for racially/ethnically patterned social environmental factors. To minimize the total number of tests and reduce the concern for Type 1 error, the above models were run only where there were racial/ethnic differences in both the social environmental factor and gene expression.

Statistical software

All analyses were conducted in R.

Results

Among the social and psychosocial exposures, there were racial/ethnic differences for major or lifetime discrimination ($p < 0.001$) and adult socioeconomic status ($\chi^2 = 144.15$, $p < 0.001$) Tukey's honest significance test indicated that Blacks reported significantly higher levels of major or lifetime discrimination than Whites ($p < 0.001$) or Hispanics ($p < 0.001$). There was not a significant difference for major or lifetime discrimination between Whites and Hispanics ($p = 0.16$). With respect to education attainment, a significantly greater percentage of non-Hispanics Whites (49%) had obtained a bachelor's degree or higher compared to Blacks (27%) or Hispanics (13%).

Table 9. Characteristics of the MESA Study Sample with Gene Expression Data				
	Total sample (n=1264)	Non-Hispanic Black (n=272)	Hispanics (n=402)	Non-Hispanic White (n=590)
Demographics				
Age mean (SD)	69.6 (9.4)	69.6(9.0)	68.4(9.3)	70.2(9.5)
Sex (% female)	51	60	50	48%
Study Site (n,%)				
Forsythe County, North Carolina	49(4%)	1(0.3%)	0	48 (40%)
New York, New York	424(34%)	131(48%)	209 (52%)	84 (14%)
Baltimore, Maryland	317(25%)	140(51%)	0	177 (30%)
Rochester, Minnesota	474(38%)	0	193 (48%)	281 (48%)
Total	1264(100%)	272 (100%)	402 (100%)	590 (100%)
Socioeconomic Status				
High education- respondent*	33%	27%	13%	49%
High education- either parent**	56%	55%	31%	71%
Psychosocial factors <i>Median, (Interquartile Range)</i>				
Loneliness	3 (3-5)	3(3-4)	4(3-5)	3 (3-5)
Lifetime Discrimination	0 (0-1)	1(0-2)	0(0-1)	0 (0-1)
Chronic Burden	1 (0-2)	1(0-2)	0(0-2)	1 (0-2)
Perceived Stress	8 (5-10)	8(5-10)	8(5-10)	7 (6-10)
Social support	9 (8-10)	9(8-10)	9(8-10)	9 (8-10)
*High education for respondent indicates an educational level of a college or greater				
**High parental education indicates an educational level of high school or greater				

Racial/ethnic differences in gene expression

Using multivariable linear regression, we identified significant racial/ethnic differences in gene transcript expression in each of the four investigated gene sets (i.e. chronic inflammation, inflammatory response, immune response, and regulation of the inflammatory response) (Table 10). For each gene set, there were more transcripts differentially expressed between Blacks and Whites than Blacks and Hispanics or Hispanics and Whites. When combining all gene sets, there were Black/White differences in expression for 449 out of the 1407 (32%) genes, indicating that expression differences were most dissimilar between these two groups. Blacks and Hispanics were most similar yet still had 333 out of 1407 (19%) gene transcripts with significant mean differences. The corresponding gene names with racial/ethnic differences in gene expression are given in Appendix 33.

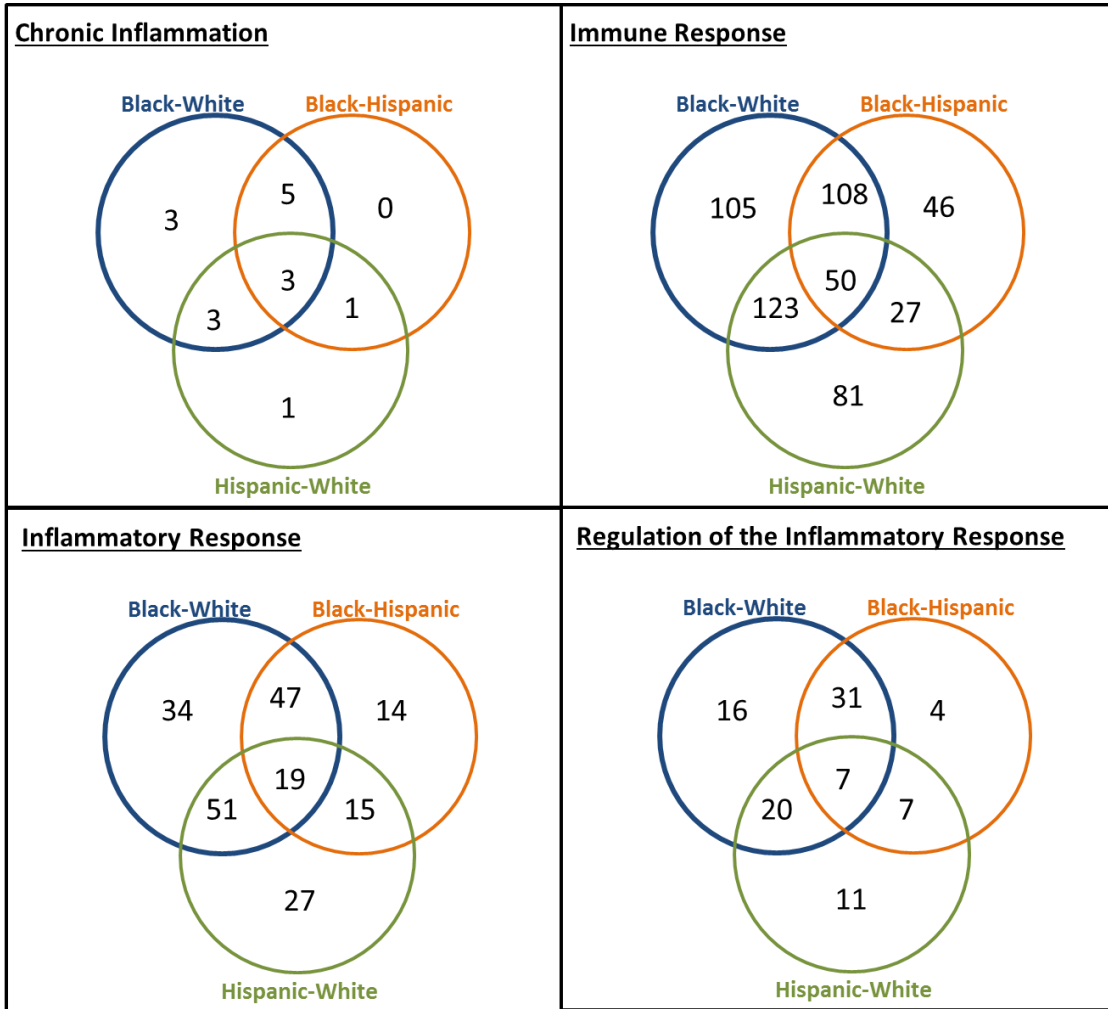
Table 10. Racial/Ethnic Differences in Gene Transcript Expression (Bonferroni Adjusted)			
Gene Set	Blacks/Whites m(%)	Blacks/Hispanic m(%)	Hispanic/White m(%)
Chronic Inflammation (k=20)	14(70%)	9 (45%)	8 (66%)
Inflammatory Response (k=438)	151(34%)	95 (22%)	112 (26%)
Immune Response (k=1251)	386(31%)	231 (18%)	281 (22%)
Regulation of the Inflammatory Response (k=192)	74(39%)	49(26%)	45(23%)
Overall unique transcripts (k=1407)	449(32%)	270(19%)	333(24%)
m(%)=number and percentage of transcripts with racial/ethnic differences within the gene set k = number of transcripts in the gene set Gene expression adjusted for age, sex, and chip Bonferroni adjusted within each gene set to family wise rate of 0.05			

Overlap

To investigate whether the gene transcripts that were differentially expressed between Blacks and Whites were also differentially expressed between Black and Hispanics or Hispanics and Whites, we plotted the findings from Table 10 using Venn diagrams for all inflammatory gene sets (Figure 2). Overall, we found 3 out of 20 transcripts in the chronic inflammatory pathway, 50 out of 1251 in the immune response pathway, 19 in inflammatory response pathway, and 7 out of 192 transcripts in the in the regulation of the inflammatory response pathway overlapped for all racial/ethnic comparisons. Interestingly, there tended to be more transcripts that were found to be differentially expressed across at least two race/ethnic comparisons than in just one racial/ethnic comparison alone. The gene names for each overlapping comparison are given in Appendix 34.

Figure 2

Overlap among Transcripts Significantly Associated with Race/Ethnicity by Biological Process (Bonferroni Adjusted)



Racial/ethnic differences in gene expression after accounting for social environmental factors.

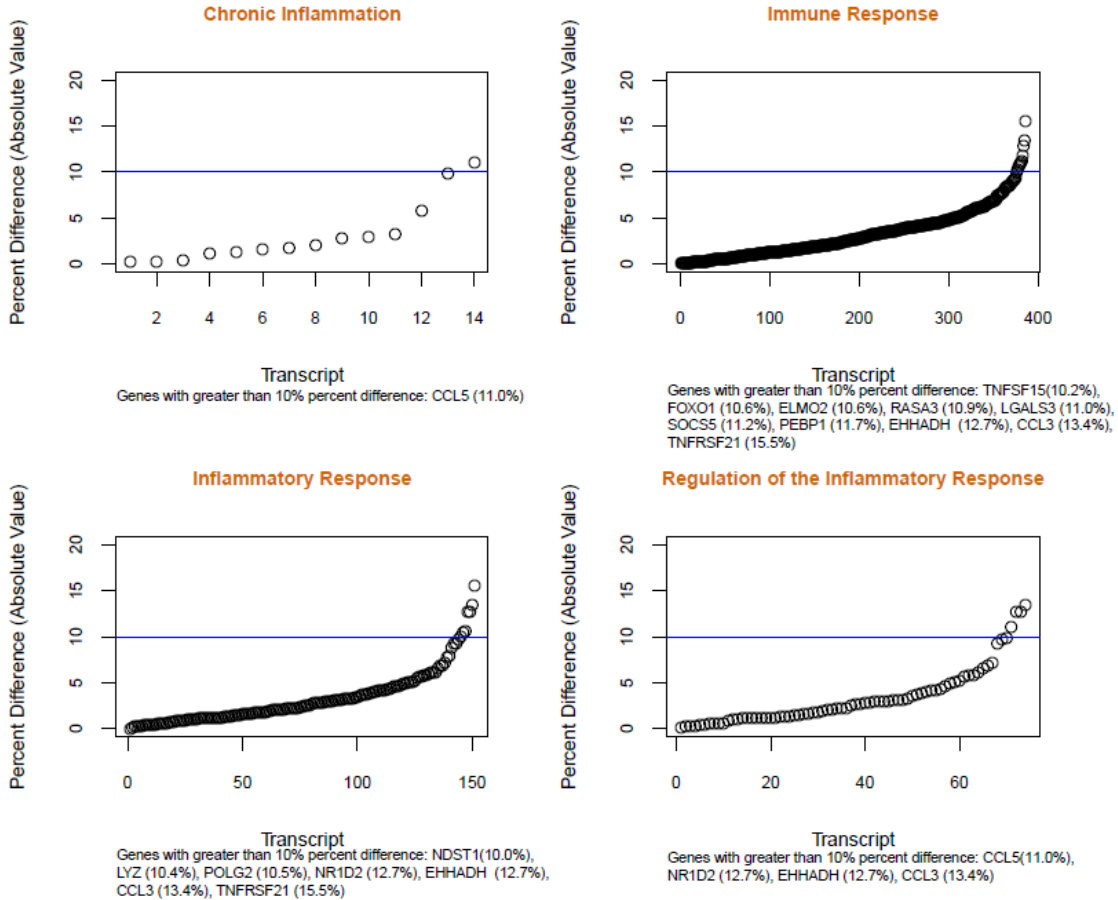
Accounting for major or lifetime discrimination (Model 2) led to at least a 10% change in the race/ethnicity beta estimate (from Model 1) for 15 of the total 449 gene transcripts with expression differences by race (~3%) in the Black/White comparison and 11 of the total 270 (~4%) in the Black/Hispanic comparison (Figures 3 and 4). Since there were significant racial/ethnic differences in adult socioeconomic status for all racial/ethnic comparisons, the

change in beta estimate was assessed for each the Black/White, Black/Hispanic, and Hispanic/White comparisons in gene expression when accounting for adult socioeconomic status. Across biological processes, accounting for adult socioeconomic status led to a change in the beta estimate for race/ethnicity for 6/449 (~1%) gene transcripts in the Black/White comparison, 6/270 (~2%) in the Black/Hispanic comparison, and 62/333 (19%) in the Hispanic/White comparison. Within all four biological processes (i.e. chronic inflammation, immune response, inflammatory response, regulation of the inflammatory response), there were a greater number of gene transcripts with at least a 10% difference in the Hispanic/White analyses compared to the Black/White or Black/Hispanic investigations (Figures 5-7). In general, across racial/ethnic comparisons, there was little overlap in the transcripts with greater than a 10% change in beta estimate (Figures 3-7).

In general, the proportion of variability that major or lifetime discrimination or adult socioeconomic status each explained was relatively small compared to that of race/ethnicity. Consequently, the proportion of variability (R^2) in gene expression explained by race/ethnicity was only modestly changed when accounting for the racially/ethnically patterned social environmental factors (R^2 Model 1 vs R^2 Model 2) (Appendix Figures 35-39).

Figure 3

**Distribution of the Absolute Value of the Percent Difference
in Beta Coefficients for Race/Ethnicity Between Models 1 and 2
Black-White: Major or Lifetime Discrimination**

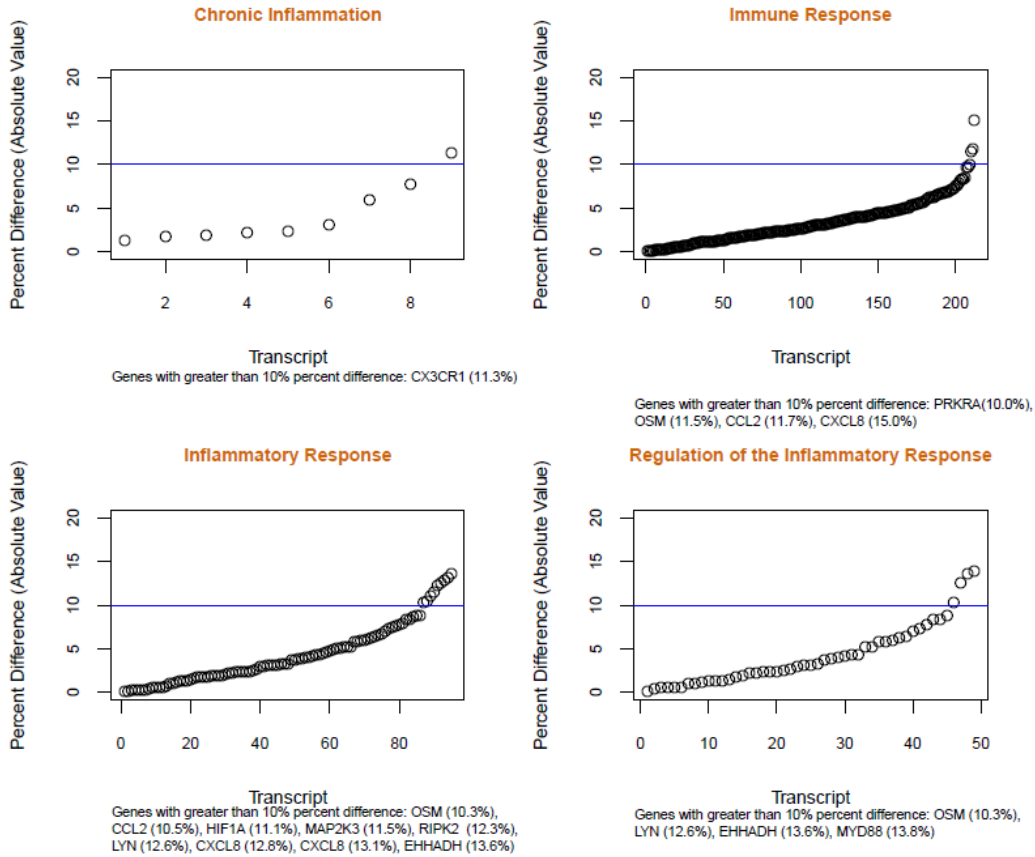


Model 1: gene expression = $\alpha_1 + \beta_1 \text{race/ethnicity}$

Model 2: gene expression = $\alpha_2 + \beta_2 \text{race/ethnicity} + \beta_3 \text{Discrimination}$

Figure 4

**Distribution of the Absolute Value of the Percent Difference in Beta Coefficients for Race/Ethnicity Between Models 1 and 2
Black-Hispanic: Major or Lifetime Discrimination**

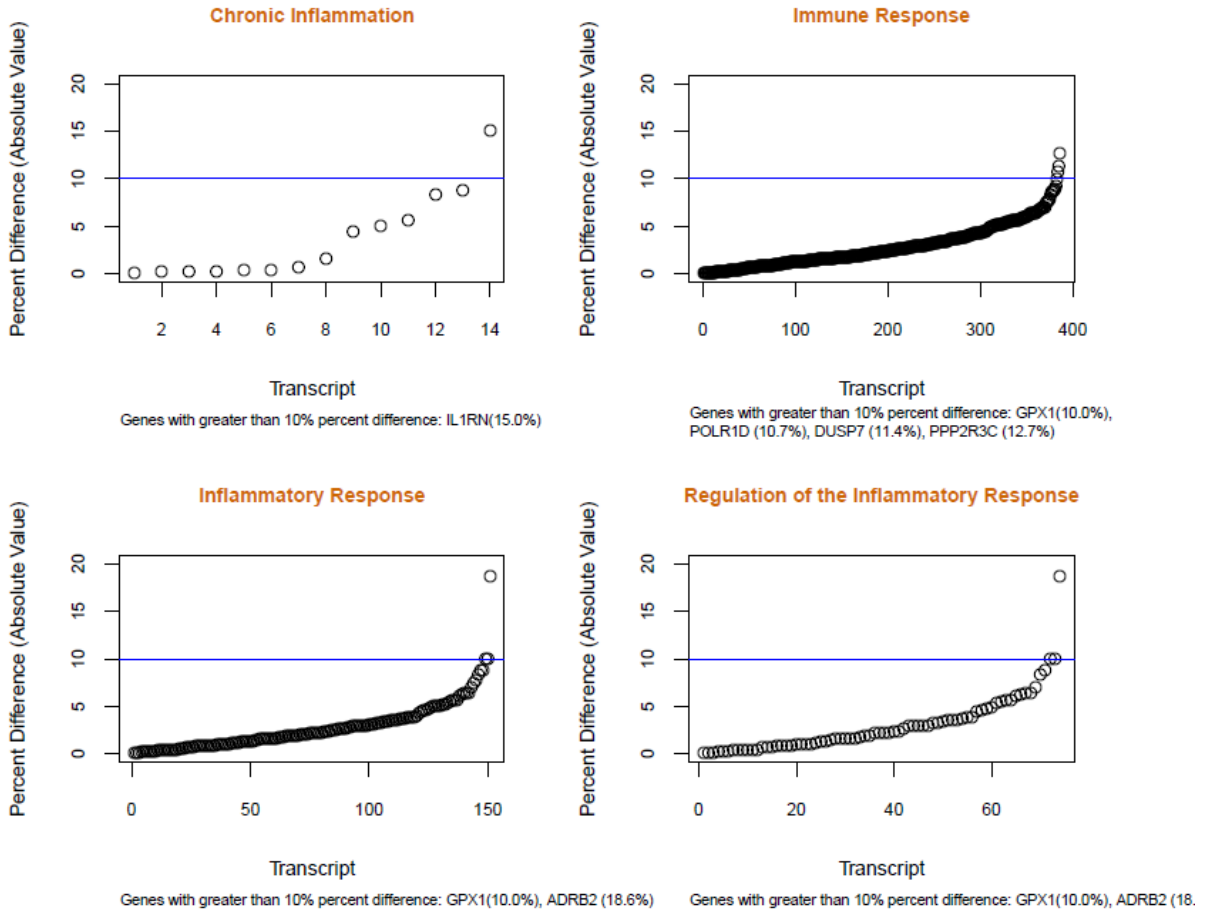


Model 1: gene expression= $\alpha_1 + \beta_1$ race/ethnicity

Model 2: gene expression= $\alpha_2 + \beta_2$ race/ethnicity+ β_3 Discrimination

Figure 5

**Distribution of the Absolute Value of the Percent Difference
in Beta Coefficients for Race/Ethnicity Between Models 1 and 2
Black-White: Socioeconomic Status**

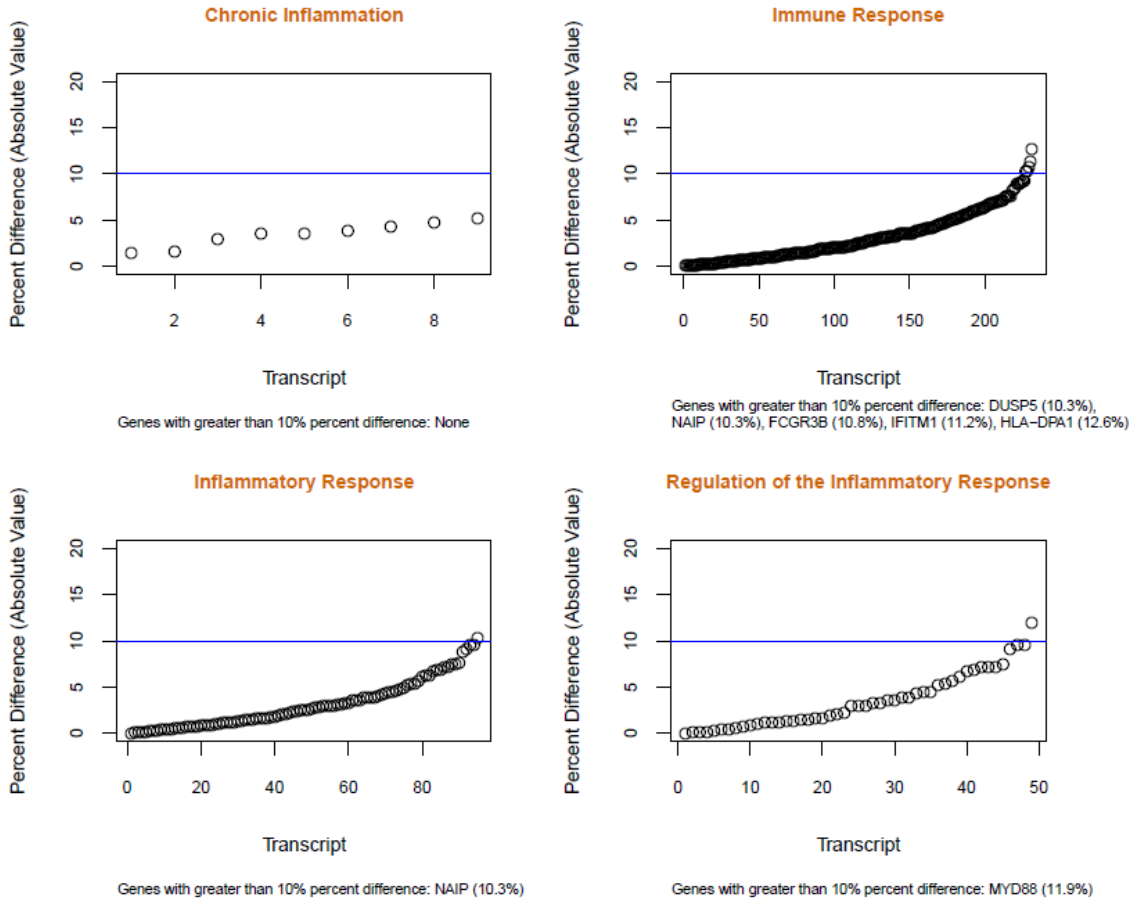


Model 1: gene expression = $\alpha_1 + \beta_1 \text{race/ethnicity}$

Model 2: gene expression = $\alpha_2 + \beta_2 \text{race/ethnicity} + \beta_3 \text{Adult SES}$

Figure 6

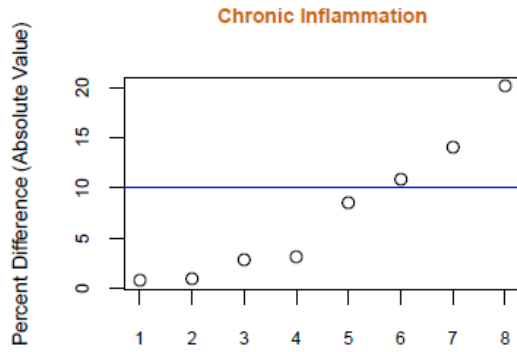
**Distribution of the Absolute Value of the Percent Difference
in Beta Coefficients for Race/Ethnicity Between Models 1 and 2
Black-Hispanic: Socioeconomic Status**



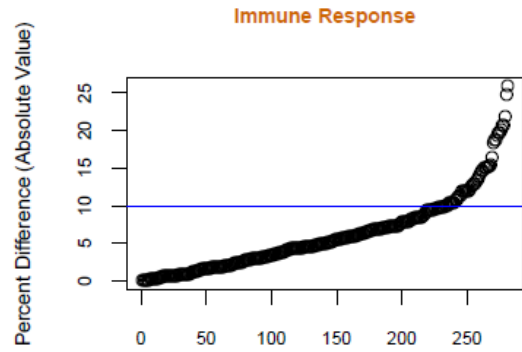
Model 1: gene expression = $\alpha_1 + \beta_1 \text{race/ethnicity}$
Model 2: gene expression = $\alpha_2 + \beta_2 \text{race/ethnicity} + \beta_3 \text{Adult SES}$

Figure 7

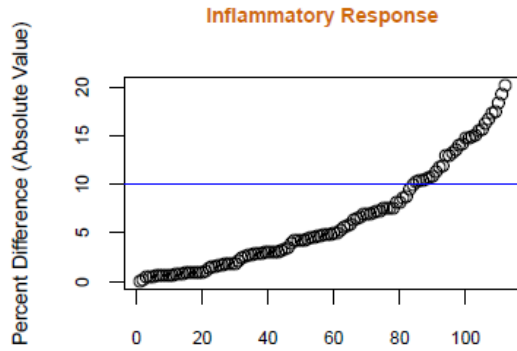
**Distribution of the Absolute Value of the Percent Difference
in Beta Coefficients for Race/Ethnicity Between Models 1 and 2
Hispanic-White: Socioeconomic Status**



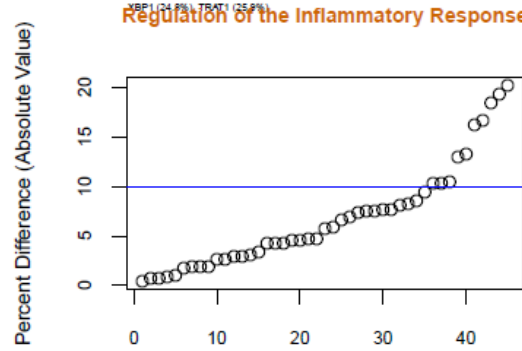
Chronic Inflammation
Transcript
Genes with greater than 10% percent difference: CEBPB (10.8%), TNF (13.9%), ADORA2B (20.1%)



Immune Response
Transcript
Genes with greater than 10% percent difference: CD35 (10.0%), CRK (10.1%), TNFRSF9 (10.3%), SOCS3 (10.3), XAF1 (10.4%), FOXF1 (10.4%), RASA2 (10.5%), FBXW11 (10.7%), CEBPB (10.8%), TRIM38 (11.1%), CXCL2 (11.4%), NFKB1B (11.9%), FLEKHA1 (11.9%), IFNGR2 (11.9%), SOCS5 (11.9%), POLR1C (11.9%), ITGB1 (12.2%), LAT2 (12.5%), ARPC4 (12.6%), WASL (12.8%), POLR3H (12.8%), BIRC3 (13.2%), IRAK2 (13.5%), ITGB1 (13.9%), PIN1 (14.2%), IRF5 (14.5%), TNFRSF21 (14.7%), NFKBIA (14.9%), CYFIP1 (15.0%), SOST (15.2%), NCAM1 (15.2%), KRAS (15.2%), NCF4 (15.5%), CAMK2D (16.4%), FFAR2 (18.4%), TNFRSF6A (18.7%), LEF1 (18.7%), CD3D (19.7%), ADORA2B (20.14%), CYFIP1 (20.6%), SWAP70 (20.7%), CD3D (21.8%), TRAF1 (25.8%)



Inflammatory Response
Transcript
Genes with greater than 10% percent difference: NFKB1 (10.2%), TNFRSF9 (10.3%), SOCS3 (10.3%), FOXF1 (10.4%), CAMK1D (10.7%), CEBPB (10.8%), CXCL2 (11.3%), CAMK1D (11.7%), NFE2L2 (11.9%), CEBPA (13.0%), STK39 (13.0%), BIRC3 (13.2%), IRAK2 (13.5%), GART (14.1%), NDST1 (14.2%), TUSC2 (14.7%), TNFRSF21 (14.7%), SIGIRR (14.8%), PABN (15.1%), CD14 (15.4%), LIAS (15.7%), NOV (16.2%), PLA2G7 (16.7%), ORM1 (17.3%), OLR1 (17.4%), FFAR2 (18.4%), CCR2 (19.2%), ADORA2B (20.1%)



Regulation of the Inflammatory Response
Transcript
Genes with greater than 10% percent difference: NFKB1 (10.2%), SOCS3 (10.3%), FOXF1 (10.4%), STK39 (13.0), BIRC3 (13.2%), NOV (16.2%), PLA2G7 (16.7%), FFAR2 (18.4%), CCR2 (19.2%), ADORA2B (20.1%)

Model 1: gene expression = $\alpha_1 + \beta_1 \text{race/ethnicity}$
Model 2: gene expression = $\alpha_2 + \beta_2 \text{race/ethnicity} + \beta_3 \text{Adult SES}$

Discussion

In this study we assessed racial and ethnic differences in gene expression and the extent to which these differences could be explained by racially/ethnically patterned social environmental factors. We specifically focused on genes involved in the inflammatory response since epidemiological evidence has pointed to chronic inflammation as a risk factor for several high burden, chronic diseases with known racial/ethnic disparities. Of the 1407 gene transcripts investigated, there were Black/White expression differences in 32%, Black/Hispanic differences in 19%, and Hispanic/White differences in 24% (Table 10). Further, there were racial/ethnic differences in two of the social environmental factors, major or lifetime discrimination and adult socioeconomic status. Although accounting for these social environmental factors led to at least a 10% change in the beta estimate in a total of 100 unique gene transcripts, the proportion in variability in gene expression explained by including the racially/ethnically patterned social environmental factors was modest. As race/ethnicity is a complex, multi-faceted constructs where differences in gene expression result from the combination of both biologic and racially/ethnically patterned environmental factors, these findings raise the question of the extent to which accounting for a single aspect of the social environment can substantially explain racial/ethnic differences in gene expression.

Racial/Ethnic Differences in Social Environmental Factors

Our findings indicate significant racial/ethnic differences in major or lifetime discrimination where Blacks reported higher levels compared to Whites and Hispanics. This finding is consistent with the literature^{131,132} and work in the MESA study specifically⁵⁰. This was expected as it is well known that Blacks in the U.S. have been disproportionately subject to

structural and individual experiences of discrimination¹³³. Interestingly, there were significant differences between Black and Hispanics but not Hispanics and Whites. This may be explained by heterogeneity in reports of discrimination among Hispanics by nativity^{134,135}. We also found significant differences for adult socioeconomic status across all racial/ethnic comparisons were Whites were most likely to have achieved a college degree (49%), followed by Blacks (27%), and then Hispanics (13%). This measure was collected in the baseline MESA sample in from 2000-2002. This is consistent with data from the 2000 Census which indicates that Whites were more likely to graduate college (27%) than Blacks (14%) or Hispanics (10%). However, the MESA sample is more educated than the national population. Despite studies that have found the contrary, we did not find racial/ethnic differences in childhood socioeconomic status, loneliness, perceived stress, chronic burden, or social support.

Racial/Ethnic Differences in Inflammatory Gene Expression

Even after employing the conservative Bonferroni multiple testing correction, we found that out of the 1407 of unique gene transcripts representing four different inflammatory processes, there were 449 (32%) with differential expression between Blacks and Whites, 270 (19%) between Blacks and Hispanics, and 333 (24%) between Hispanics and Whites. The observed racial/ethnic differences in gene expression result from differences in inherited DNA sequence and the regulation of the genome by environmental factors. Mechanistically, genetic expression is influenced by a number of factors including DNA sequence, epigenetic modifications, and mRNA degradation^{68,136,137}. Research investigating the contribution of genetic ancestry to racial/ethnic differences in gene expression is traditionally analyzed at the DNA sequence level by investigating differences in single nucleotide polymorphisms (SNPs)^{67,68}.

Over generations, allele frequency differences between races/ethnicities develop due to geographical, social, and political factors that produce assortative mating practices (i.e. sexual selection with similar individuals). Such allele frequency variations are a partial contributor to racial/ethnic differences in gene expression. Despite the contribution of population substructure, it has observed that within group variations are greater than between group variation which indicates that allelic frequencies differences between race and ethnicities are insufficient in explaining complex phenotypic differences in race/ethnicity¹³⁸. Environmental (e.g. physical, social) contributors play important roles as well. In a prototypic example, a study investigating comparing the effects of ethnicity and geographic residence (rural vs. urban environment) on gene expression found that more genes were differentially expressed by geographic residence than genetic ancestry¹³⁹. Other studies have found that exposure to adverse social environments is associated with an upregulation of proinflammatory genes across social environmental measures (e.g. loneliness, socioeconomic status, caregiver stress) and across species (e.g. human, rhesus macaques, mice), a phenomenon coined the “conserved transcriptional response to adversity (CTRA).” However, whether the social environmental factors contribute to racial/ethnic differences in gene expression was not examined in these prior CTRA studies.

Racial/Ethnic Differences in Inflammatory Gene Expression Accounting for Racially/Ethnically Patterned Social Environmental Factors

In our central analyses, we examined the effect of accounting for the racially/ethnically patterned social environmental factors (i.e. major or lifetime discrimination and adult socioeconomic status) on the association between race/ethnicity and gene expression by: 1) assessing the percent change in beta coefficient with and without the social environmental factor

and 2) comparing the proportion of variability in gene expression explained by race/ethnicity with and without the social environmental factor. Of the 449 unique genes with Black/White differences in expression, there was at least a 10% change in the beta coefficient in 15 (~3%) after accounting for major or lifetime discrimination. These genes had various functions such as leukocyte chemotaxis (CCL5, CCL3, LGALS3), mitogen-activated protein kinase signaling (NDST1, CCL5, CCL3, PEBP1, RASA3), and apoptosis (LGALS3, TNFRSF21). Similarly, there was at least a 10% change in the beta coefficient in 11 or the 270 gene transcripts (~4%) for the Black/Hispanic comparison after accounting for major or lifetime discrimination. These genes also had a variety of functions including regulating leukocyte migration (CCL2, MYD88, CXCL8, LYN) and positive regulation of chemokine production (RIPK2, HIF1A, MYD88).

Across race/ethnic comparisons, there was a range in the number of transcripts for which accounting for adult socioeconomic status led to at least a 10% change in the beta estimate (Figures 3-7). As in accounting for major or lifetime discrimination, these genes had a myriad of functions (e.g. regulating type 1 interferon production, leukocyte migration, and regulating cytokine production). Notably, compared to the other racial/ethnic comparisons accounting for adult socioeconomic status changed the beta estimate by at least 10% for the greatest number of gene transcripts in the Hispanics/Whites comparison. There are two main explanations for this finding. First, this can be attributed to the stark differences in adult SES between Hispanics and Whites. In the MESA sample, 49% of Whites had a college degree whereas only 13% of Hispanics achieved this level of education. This gap is wider than the other racial/ethnic comparisons (i.e. Black/White and Black/Hispanic). Further, previous studies indicate that education does not confer the same advantage for Hispanics and Whites which also contributes to the observed differences¹⁴⁰.

Next, the proportion of variability explained by race/ethnicity with and without the racially/ethnically patterned social environmental factors was examined. We found that major or lifetime discrimination and adult SES each only modestly increased the proportion of variability in gene expression explained by race/ethnicity (Appendix Figures 35-39). These findings were unexpected as we anticipated that racially/ethnically patterned social factors would explain a substantial part of the variability. However, there are a number of reasons that may explain this finding. One reason may be that the measures used to assess major or lifetime discrimination and socioeconomic status may not have long term effects on gene expression. Major or lifetime discrimination was measured by whether the event had ever happened in the participant's life. Therefore, the discriminatory event may have happened many years prior to the collection of the gene expression. A more recent measure of exposure to discriminatory experiences may be more likely to be associated with gene expression. Similarly, we used having a college education as a measure of socioeconomic status due to its stability of not changing over the life course. However, our participants were an average of ~70 years old and the effects of a college education, which would have been likely acquired approximately 50 years earlier may have diminished. Another explanation of the findings is that a single dimension of the social environment may be small compared to overall effect of race/ethnicity. Experiences of major or lifetime discrimination and socioeconomic status are each only one construct that can account for the experience of being in a certain racial/ethnic category, yet these factors of the social environment work in concert with other racially-patterned aspects of the social environment. Summary measures that capture more aspects of the social environment would help to better explain the contribution of the social environment to race/ethnic differences in gene expression.

Strengths and Limitations

There were strengths and limitations of the present study. One of the main strengths of the study was the use to the large, racially/ethnically diverse sample from the Multi-Ethnic Study of Atherosclerosis. Since gene expression is cell type specific, it is imperative that appropriate cell types are used for the biological process under investigation. In MESA, gene expression was collected in monocytes, an important cell type of the inflammatory response.

A second strength of the present study is the focus on inflammation related genes specifically. Most prior human social genomics studies have been conducted at a genome wide level and investigations of biological meaning of differentially expressed genes has been post hoc. However, by using the Gene Ontology database, we were able to use an a priori defined set inflammation and immune response related genes specifically. This study is not without limitations. Most notably we not account for genetic ancestry when accounting for race/ethnic effects as the data was not yet available. Secondly, we did not account for within race/ethnic heterogeneity. This is most notable in Hispanics where nativity has been shown be associated with social environmental and overall health measures.

Conclusion

In order to reduce the burden of racial/ethnic health disparities, it is important that research uncovers the mechanisms that allow these disparities to persist. This study contributes to the growing body of human social genomics and health disparities literatures by assessing racial/ethnic differences in gene expression and the extent to which social environmental factors explain these associations. Prior human social genomics studies were unable to conduct such investigations as they were limited by small, racially/ethnically homogeneous samples. Further,

little work in the health disparities literature has focused on gene expression as a potential biological mediator in the relationship between race/ethnicity and health. To develop effective prevention and treatment approaches, studies such as the present one that investigate how socially constructed race/ethnicity categorization affects biological functioning, are needed.

CHAPTER V: Conclusion

Review of Main Findings

In this dissertation, data from the large, racially/ethnically diverse Multi-Ethnic Study of Atherosclerosis (MESA) was used to investigate the association between a range of social environmental factors and gene expression. In aim 1, we hypothesized that expression of genes previously implicated as part of the conserved transcriptional response to adversity (CTRA) would be differentially expressed by seven a priori selected social environmental factors (i.e. adult socioeconomic status, child socioeconomic status, loneliness, major or lifetime discrimination, perceived stress, chronic burden, and social support) in MESA. Both global and gene level analyses were used to investigate this hypothesis. We found that major or lifetime discrimination and chronic burden were significant with a CTRA gene set in global analyses. In multivariable linear regression analyses, the number of transcripts associated with the social environmental factor at $p < 0.05$ ranged from 70-196 dependent upon social environmental factor. However, none were significant after adjustment for multiple testing. A varying number of transcripts (0-74) were selected via elastic net, the number of which varied dependent of social environmental exposure. These findings provide some evidence that there may be a concerted, conserved response to social environmental factors at the gene level. However, the characteristics (e.g. cumulative exposure, impact) of social environmental factors that associate

with gene expression has yet to be fully explored. This is further complicated by the difficulty in replicating individual gene associations across studies.

In aim 2, we built upon aim 1 by investigating whether the same social environmental factors were associated with the expression of a broader set of physiologically defined immune system related gene sets derived from the Gene Ontology database. Some genes investigated in this aim overlapped with genes investigated in aim 1 (Appendix 40). Due to its epidemiologic associations with several high burden diseases, in aim 2 we focused specifically on expression of genes involved in the inflammatory and immune response. In global analyses, we found major or lifetime discrimination to be significantly associated with each of the four investigated gene sets (i.e. chronic inflammation, inflammatory response, immune response, and regulation of the inflammatory response). Loneliness and chronic burden were also significantly associated with the chronic inflammatory gene set in global analyses. As in aim 1, there were not significant associations in the linear regression analyses after multiple testing and a varying number of transcripts selected in elastic net. Aim 2 analyses demonstrate that some social environmental factors are associated with expression of inflammation related genes, and exposure to major or lifetime discrimination may be especially influential in affecting inflammation and immune system related gene expression.

In aim 3, we investigated the association between race/ethnicity with a priori defined sets of inflammatory and immune system related genes and the extent to which this association could be explained by racially/ethnically patterned social environmental factors (i.e. major or lifetime discrimination and adult socioeconomic status). Accounting for a racially/ethnically patterned social environmental factor (i.e. major or lifetime discrimination, adult SES) in the association between race/ethnicity and gene expression changed the effect estimate by >10% for less than

5% of the inflammation and immune response genes investigated. Across gene sets and racial/ethnic comparisons, race/ethnicity on average explained more of the variability in gene expression than the racially/ethnically pattern social environmental factors. The findings in aim 3 indicate that accounting for a single dimension of the social environment may be small compared to the overall effect of race/ethnicity.

Significance

The conserved transcriptional response to adversity (CTRA) studies were an important stimulus to the emergence of the new field of human social genomics. The CTRA is categorized by and upregulation of proinflammatory genes and downregulation of Type 1 interferon and antibody synthesis genes in response to adverse social environmental exposures. This area has received widespread attention as it serves as a mechanism that has the potential to explain the well documented relationships between the social environment and health. One of the main shortcomings of these studies, however, is the lack of replication in a separate human cohort. Due to the infamous history of saturating the literature with false positive findings, replication is critical in genomic studies. Aim 1 of this dissertation served as a scientific evaluation of the reproducibility of the findings in the current CTRA literature. Our findings of aim 1 suggest that for some exposures (e.g. major or lifetime discrimination) but not others (e.g. perceived stress) there are differences in gene expression. This partially supports the idea of a conserved transcriptional response to adversity. At the same time, the aim 1 findings highlight the analytical challenges of such studies, particularly as we did not reproduce any of the gene level associations in linear regression analysis. Single gene analyses are difficult to replicate in part due to interindividual heterogeneity in genomes and environments. Further, the high number of

statistical tests makes these types of studies prone to Type 1 error. As a new area of research, scientists must be particularly meticulous in ensuring that our methodological approaches are appropriate, even if cutting-edge or yet to be developed statistical models are needed. We addressed the multiple testing issue by first assessing global significance using a single test, the Global ANCOVA. In the linear regression analyses, we employed the multiple testing correction designed to account for correlated data. In elastic net, we were able to identify transcripts most robustly associated with the social environmental exposures. In contrast to OLS regression, elastic net is able to handle $p > n$ investigations and account for multicollinearity. We were encouraged by discovering that many of the transcripts selected via elastic net tended to have lower p-values in the linear regression analysis, indicating suggestive associations between social environmental factors and gene expression that linear regression may have been ill powered to detect. The introduction of this the multi-faceted analytical approach in this dissertation to the human social genomics literature will help to move the field forward as we work towards identifying the best methods to address the complexity of the statistically assessing the relationship between social environment factors and the genome.

By focusing specifically on a priori biologically defined gene sets, our aim 2 uniquely contributes to the human social genomics literature by investigating chronic inflammation specifically. While other studies have focused on the entire genome or a subset of genes representing a few biological processes, we focus specifically on inflammation due to its known associations with chronic disease. This approach helps to reduce the dimensionality that is introduced by assessing the whole genome. Further, contrary to other previous studies that selected a small number of genes (i.e. range: 3-31) to represent a biological process in a contrast score, we assessed all of the genes available in the Gene Ontology database to give a more

comprehensive picture of the effect of social environmental factors on inflammatory gene expression.

Aim 3 further adds to the human social genomics literature by assessing the association between race/ethnicity and gene expression and the extent to which these associations are explained by racially/ethnically patterned social factors. As racism has been deemed a fundamental cause of health inequalities it is important to understand the mechanisms through which socially constructed racial and ethnic categorizations work to impact biological functioning and overall health. Consistent with previous literature, we found there to be racial/ethnicity differences in gene expression. We also found that the investigated social environmental factors individually only explained a small part of the effect. Work by Phelan and Link posit that racism is associated with health only in part through socioeconomic status, but that a range of other socially patterned factors (e.g. access and quality of health care, discrimination, residential segregation) also contribute to the relationship²³. Aim 3 highlighted the complexity of assessing the contribution of social environmental factors in explaining racial/ethnic differences in health. It is important to consider how such environmental factors act independently and together to affect gene expression.

Implications

The unveiling of the Precision Medicine Initiative has encouraged scientists to assess how differences in genes, environment, and lifestyles matter to best address the health needs of the individual¹⁴¹. With recent advancements in technology such as the completion of the Human Genome Project, decreasing costs of sequencing, and increased data storage ability, proponents of the initiative argue that an individualized approach to healthcare is now possible. Although

most of the attention has focused on genetic advancements, it is imperative that we understand the direct and indirect mechanisms through which environmental factors act on biological functioning and thereby overall health. By assessing the associations between social environmental factors and gene expression, this dissertation helps to lay the groundwork for future precision medicine efforts. It will be important to consider the whole person, both in terms of biological and non-biological factors, to tailor effective prevention and treatment strategies (e.g. pharmacogenetic, behavioral, and psychological). Studies that investigate how social environmental factors “get under the skin” will be critical in these efforts.

Strengths

Use of the Multi-Ethnic Study of Atherosclerosis study to investigate the research questions of this dissertation was a valuable strength. With 1,264 participants, the sample size nearly doubled that of any previous conserved transcriptional response to adversity studies, providing greater power to detect gene expression differences. Further compared to previous studies, the MESA study is more racial/ethnically heterogeneous. This enabled the investigation of racial/ethnic differences in gene expression and social environmental factors that may be influencing the relationship. As a study that has focused mostly on social determinants of health measures, the social environmental measures collected in MESA are particularly thorough compared to other studies. The collection of gene expression data in monocytes in MESA was also an asset to the current investigation. Monocytes are phagocytic cells that are an important leukocyte of the innate immune system. Previous human social genomics studies have primarily collected gene expression in peripheral blood mononuclear cells (PBMCs), which are an assortment of leukocytes including monocytes, T cells, B cells, natural killer (NK) cells. Since

gene expression is cell type specific, use of a variety of cell types in a single study is not ideal as associations that may be present with one cell type can be masked by non-significant associations in a different cell type. Prior research indicates that among the cell types of PBMCs, monocytes are particularly sensitive to social environment exposures^{63,117}. Use of solely monocytes in the MESA study enabled the greatest likelihood of detecting associations.

The analytical approach employed is strength of this dissertation. Human social genomic studies are inherently complex due to interrogation of a high number of variables with an unknown correlation structure. Our statistical approaches (i.e. Global ANCOVA, linear regression, and elastic net) were designed to account to the complexity of multivariate testing of correlated variables. By using the permutation based approximation of the F distribution in the Global ANCOVA test, we accounted for the gene expressions being neither independent nor homoscedastic⁸⁹. In linear regression analyses, we employed false discovery rate multiple testing correction for correlated data to address issues related to the total number of statistical tests being conducted and the correlated nature of the gene expression outcomes. Elastic net, which was used as a variable selection procedure, is designed to address multicollinearity.

Limitations

While there were notable strengths to this study, we acknowledge that there were some important limitations. In the interest of systematically identifying social sensitive genes, we limited our literature search to articles that specifically used the term “conserved transcriptional response to adversity”. In doing this, we may have missed other genes in the literature whose expression is modified by social environment exposures but did not use this term in the published article.

A second limitation is the variation in time over which data was collected. Social environmental factors were collected at various exams in the MESA study while gene expression was only collected in Exam 5. The baseline MESA exam was conducted in 2000 and Exam 5 ended in 2012. Therefore, there is a potential gap of 12 years from collection of the social environmental exposure and the blood draw used to assess gene expression. To minimize the bias this may introduce, we included data from the most recent exam available. Further, since we only had gene expression data available for one time point, we cannot comment on the dynamic nature of the tested relationships.

Future Work

There are several areas in which further research is needed as we seek to understand the complex interplay between genomics, the environment, and lifestyle. Our developing knowledge surrounding epigenetic responses to environmental and lifestyle cues hints at epigenetics modifications as a likely mediator between the social environment and gene expression. For example, a recent study by Needham et. al found significant relationships between low socioeconomic status and methylation of stress related genes¹⁴². Future research should investigate the mediating and causal roles of epigenetic modifications in the pathway between the social environment and gene expression.

Further, it will be increasingly important to understand how the environmental and lifestyles exposures work independently and in concert to affect the genome. Different social and psychosocial experiences may activate different genes and pathways. This is consistent with the little overlap between the nine identified CTRA studies (despite implication of many of the same biological processes) investigated in Aim 1. For example, similar types of exposures (e.g.

complicated and non-complicated grief, hedonic and eudemonic well-being) were associated with expression of different genes. Most extant studies, including this dissertation, have focused on a single social or psychosocial exposure at a time. However, the human social experience is composed of many interacting factors. Future research should investigate how such factors work together to affect the genome. This study helps demonstrate that the previously used approaches (i.e. differential expression, contrast score) have been unable to uncover replicable relationships between the social environment and gene expression. Latent variable models and sequence kernel analysis could be used to better represent the complexity of the human environment as we move the field of human social genomics forward.

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APPENDICES

Appendix 1

List of Conserved Transcriptional Response to Adversity (CTRA) Genes		
Illumina Transcript ID	Gene Name	Gene Symbol
ILMN_1651433	deoxycytidine kinase	DCK
ILMN_1651496	histone cluster 1 H2bd	HIST1H2B
ILMN_1651499	Unnamed Transcript	N/A
ILMN_1651735	trans-golgi network protein 2	TGOLN2
ILMN_1651826	brain abundant membrane attached signal protein 1	BASP1
ILMN_1651828	chaperonin containing TCP1 subunit 3 (gamma)	CCT3
ILMN_1652065	potassium large conductance calcium-activated channel subfamily M	KCNMB1
ILMN_1652085	M-phase phosphoprotein 10 (U3 small nucleolar ribonucleoprotein)	MPHOSP H1
ILMN_1652198	cerebral cavernous malformation 2	CCM2
ILMN_1652394	RAB2A member RAS oncogene family	RAB2A
ILMN_1652777	CDC42 effector protein (Rho GTPase binding) 2	CDC42EP 2
ILMN_1652790	CDC-like kinase 1	CLK1
ILMN_1652806	ATP synthase H ⁺ transporting mitochondrial Fo complex subunit F	ATP5J
ILMN_1652825	interleukin 10 receptor alpha	IL10RA
ILMN_1652906	globoside alpha-1 3-N-acetylgalactosaminyltransferase 1	GBGT1
ILMN_1653166	C-type lectin domain family 10 member A	CLEC10A
ILMN_1653266	DnaJ (Hsp40) homolog subfamily B member 14	DNAJB14
ILMN_1653283	amyloid beta (A4) precursor protein	APP
ILMN_1653443	cyclin-dependent kinase 2	CDK2
ILMN_1653466	hes family bHLH transcription factor 4	HES4
ILMN_1653480	coiled-coil domain containing 125	CCDC125
ILMN_1653708	coronin actin binding protein 1B	CORO1B
ILMN_1653711	frizzled class receptor 2	FZD2
ILMN_1653871	nicotinamide phosphoribosyltransferase	NAMPT
ILMN_1654262	zinc finger matrin-type 3	ZMAT3

ILMN_1654504	C-type lectin domain family 7 member A	CLEC7A
ILMN_1654545	cleavage and polyadenylation specific factor 1 160kDa	CPSF1
ILMN_1654560	Unnamed Transcript	
ILMN_1654583	chromodomain helicase DNA binding protein 1	CHD1
ILMN_1654685	multiple C2 domains transmembrane 1	MCTP1
ILMN_1654735	solute carrier organic anion transporter family member 3A1	SLCO3A1
ILMN_1654778	lymphocyte-specific protein 1	LSP1
ILMN_1654812	unc-93 homolog B1 (C. elegans)	UNC93B1
ILMN_1654851	family with sequence similarity 134 member A	FAM134A
ILMN_1655077	PR domain containing 1 with ZNF domain	PRDM1
ILMN_1655177	phosphatidylinositol 4-kinase catalytic alpha	PI4KA
ILMN_1655414	tumor necrosis factor (ligand) superfamily member 14	TNFSF14
ILMN_1655422	ribosomal protein L17	RPL17
ILMN_1655635	methyltransferase like 3	METTL3
ILMN_1655930	elongation factor RNA polymerase II 2	ELL2
ILMN_1655961	SND1 intronic transcript 1 (non-protein coding)	SND1-IT1
ILMN_1656011	regulator of G-protein signaling 1	RGS1
ILMN_1656184	Unnamed Transcript	
ILMN_1656254	Unnamed Transcript	
ILMN_1656310	indoleamine 2 3-dioxygenase 1	IDO1
ILMN_1656486	DnaJ (Hsp40) homolog subfamily C member 10	DNAJC10
ILMN_1656761	TGFB-induced factor homeobox 1	TGIF1
ILMN_1657619	DnaJ (Hsp40) homolog subfamily B member 14	DNAJB14
ILMN_1657790	serine/arginine-rich splicing factor 11	SRSF11
ILMN_1657797	fibroblast growth factor (acidic) intracellular binding protein	FIBP
ILMN_1657857	transmembrane protein 14C	TMEM14C
ILMN_1657862	adenosylhomocysteinase	AHCY
ILMN_1657871	radical S-adenosyl methionine domain containing 2	RSAD2
ILMN_1657977	methionine sulfoxide reductase B2	MSRB2
ILMN_1658015	muscleblind-like splicing regulator 2	MBNL2
ILMN_1658182	mex-3 RNA binding family member C	MEX3C
ILMN_1658247	2'-5'-oligoadenylate synthetase 1 40/46kDa	OAS1
ILMN_1658494	regulator of cell cycle	RGCC
ILMN_1658504	choline kinase alpha	CHKA
ILMN_1658798	multiple EGF-like-domains 9	MEGF9
ILMN_1658884	ATPase class VI type 11B	ATP11B
ILMN_1658920	multiple C2 domains transmembrane 1	MCTP1
ILMN_1659058	protein phosphatase 1 regulatory subunit 10	PPP1R10
ILMN_1659122	Kruppel-like factor 10	KLF10

ILMN_1659189	chromosome 9 open reading frame 89	C9orf89
ILMN_1659227	CD79a molecule immunoglobulin-associated alpha	CD79A
ILMN_1659257	tumor necrosis factor receptor superfamily member 8	TNFRSF8
ILMN_1659285	proteasome (prosome macropain) assembly chaperone 1	PSMG1
ILMN_1659463	apoptotic peptidase activating factor 1	APAF1
ILMN_1659524	NADH dehydrogenase (ubiquinone) complex I assembly factor 4	NDUFAF4
ILMN_1659620	Unnamed Transcript	
ILMN_1659688	lectin galactoside-binding soluble 3 binding protein	LGALS3BP
ILMN_1659913	interferon stimulated exonuclease gene 20kDa	ISG20
ILMN_1659923	guanine nucleotide binding protein (G protein) q polypeptide	GNAQ
ILMN_1659936	protein phosphatase 1 regulatory subunit 15A	PPP1R15A
ILMN_1659960	interleukin 4 induced 1	IL4I1
ILMN_1659990	hypoxia inducible lipid droplet-associated	HILPDA
ILMN_1660027	Fc fragment of IgG low affinity IIb receptor (CD32)	FCGR2B
ILMN_1660270	Unnamed Transcript	
ILMN_1660440	abhydrolase domain containing 17B	ABHD17B
ILMN_1660462	mucolipin 2	MCOLN2
ILMN_1660579	cytokine receptor-like factor 3	CRLF3
ILMN_1660582	ligase III DNA ATP-dependent	LIG3
ILMN_1660635	lactamase beta 2	LACTB2
ILMN_1660661	t-complex 1	TCP1
ILMN_1660847	6-phosphofructo-2-kinase/fructose-2,6-biphosphatase 3	PFKFB3
ILMN_1661197	cardiotrophin-like cytokine factor 1	CLCF1
ILMN_1661409	family with sequence similarity 208 member A	FAM208A
ILMN_1661432	nucleoporin 43kDa	NUP43
ILMN_1661439	flotillin 1	FLOT1
ILMN_1661589	CD151 molecule (Raph blood group)	CD151
ILMN_1661599	DNA-damage-inducible transcript 4	DDIT4
ILMN_1661646	B-cell scaffold protein with ankyrin repeats 1	BANK1
ILMN_1661695	interleukin-1 receptor-associated kinase 3	IRAK3
ILMN_1661833	ankyrin repeat domain 12	ANKRD12
ILMN_1662358	MX dynamin-like GTPase 1	MX1
ILMN_1662417	leucine-rich pentatricopeptide repeat containing	LRPPRC
ILMN_1662451	Fc fragment of IgE low affinity II receptor for (CD23)	FCER2
ILMN_1662488	mediator complex subunit 23	MED23
ILMN_1662617	protein phosphatase 2 regulatory subunit B" gamma	PPP2R3C
ILMN_1662795	carbonic anhydrase II	CA2
ILMN_1662865	calcineurin-like phosphoesterase domain containing 1	CPPED1
ILMN_1662896	WD repeat domain 11	WDR11

ILMN_1662973	CD82 molecule	CD82
ILMN_1663090	SON DNA binding protein	SON
ILMN_1663142	C-type lectin domain family 12 member A	CLEC12A
ILMN_1663149	signal recognition particle 54kDa	SRP54
ILMN_1663159	Unnamed Transcript	
ILMN_1663356	rabphilin 3A	RPH3A
ILMN_1663484	Unnamed Transcript	
ILMN_1663493	Unnamed Transcript	
ILMN_1663646	Dmx-like 1	DMXL1
ILMN_1663664	mitochondrial ribosomal protein S10	MRPS10
ILMN_1663866	transforming growth factor beta-induced 68kDa	TGFB1
ILMN_1664006	amylo-alpha-1 6-glucosidase 4-alpha-glucanotransferase	AGL
ILMN_1664016	Rho/Rac guanine nucleotide exchange factor (GEF) 18	ARHGEF18
ILMN_1664068	endoplasmic reticulum-golgi intermediate compartment (ERGIC) 1	ERGIC1
ILMN_1664094	purinergic receptor P2Y G-protein coupled 13	P2RY13
ILMN_1664098	Fas-activated serine/threonine kinase	FASTK
ILMN_1664371	Unnamed Transcript	
ILMN_1664449	ALG5 dolichyl-phosphate beta-glucosyltransferase	ALG5
ILMN_1664525	purinergic receptor P2Y G-protein coupled 13	P2RY13
ILMN_1664543	interferon-induced protein with tetratricopeptide repeats 3	IFIT3
ILMN_1664644	autophagy related 16-like 2 (<i>S. cerevisiae</i>)	ATG16L2
ILMN_1664691	dual adaptor of phosphotyrosine and 3-phosphoinositides	DAPP1
ILMN_1665049	spastic paraplegia 11 (autosomal recessive)	SPG11
ILMN_1665065	serine incorporator 3	SERINC3
ILMN_1665100	prostaglandin-endoperoxide synthase 1 (prostaglandin G/H synthase a)	PTGS1
ILMN_1665117	chromosome 6 open reading frame 89	C6orf89
ILMN_1665192	nudix (nucleoside diphosphate linked moiety X)-type motif 6	NUDT6
ILMN_1665217	centrosomal protein 19kDa	CEP19
ILMN_1665243	FK506 binding protein 14 22 kDa	FKBP14
ILMN_1665428	gasdermin D	GSDMD
ILMN_1665559	cyclin-dependent kinase 2	CDK2
ILMN_1665583	tubulin beta class I	TUBB
ILMN_1665859	RAB27A member RAS oncogene family	RAB27A
ILMN_1665964	GRB2-associated binding protein 2	GAB2
ILMN_1666007	trafficking protein particle complex 6B	TRAPPC6B
ILMN_1666122	heart development protein with EGF-like domains 1	HEG1
ILMN_1666269	cathepsin Z	CTSZ

ILMN_1666364	coenzyme Q10 homolog A (<i>S. cerevisiae</i>)	COQ10A
ILMN_1666552	zinc finger protein 75a	ZNF75A
ILMN_1666733	chemokine (C-X-C motif) ligand 8	CXCL8
ILMN_1666742	chromosome 9 open reading frame 72	C9orf72
ILMN_1666932	Fc fragment of IgG low affinity IIa receptor (CD32)	FCGR2A
ILMN_1667068	zinc finger CCCH-type antiviral 1	ZC3HAV1
ILMN_1667460	sulfatase 2	SULF2
ILMN_1667476	lymphotoxin beta receptor (TNFR superfamily member 3)	LTBR
ILMN_1667594	Kruppel-like factor 10	KLF10
ILMN_1667711	phospholipase A2 group XVI	PLA2G16
ILMN_1667796	Unnamed Transcript	
ILMN_1667883	THO complex 5	THOC5
ILMN_1667893	tensin 3	TNS3
ILMN_1668134	glutathione S-transferase mu 1	GSTM1
ILMN_1668345	OAF homolog (<i>Drosophila</i>)	OAF
ILMN_1668463	Unnamed Transcript	
ILMN_1668484	leucine rich repeat containing 47	LRRC47
ILMN_1668525	nuclear receptor subfamily 3 group C member 1 (glucocorticoid re	NR3C1
ILMN_1668526	GTPase very large interferon inducible pseudogene 1	GVINP1
ILMN_1668605	N-acylethanolamine acid amidase	NAAA
ILMN_1668721	cyclin D3	CCND3
ILMN_1668979	DnaJ (Hsp40) homolog subfamily B member 14	DNAJB14
ILMN_1669062	Unnamed Transcript	
ILMN_1669189	Ras association (RalGDS/AF-6) domain family member 5	RASSF5
ILMN_1669376	DNA-damage regulated autophagy modulator 1	DRAM1
ILMN_1669377	adaptor-related protein complex 4 beta 1 subunit	AP4B1
ILMN_1669523	FBJ murine osteosarcoma viral oncogene homolog	FOS
ILMN_1669572	ring finger protein 126	RNF126
ILMN_1669674	canopy FGF signaling regulator 3	CNPY3
ILMN_1669727	WW domain containing adaptor with coiled-coil	WAC
ILMN_1669888	cystatin A (stefin A)	CSTA
ILMN_1670037	polymerase (RNA) II (DNA directed) polypeptide L 7.6kDa	POLR2L
ILMN_1670134	fatty acid desaturase 1	FADS1
ILMN_1670145	deafness autosomal dominant 5	DFNA5
ILMN_1670305	serpin peptidase inhibitor clade G (C1 inhibitor) member 1	SERPING 1
ILMN_1670322	FCH domain only 2	FCHO2
ILMN_1670439	forty-two-three domain containing 1	FYTTD1
ILMN_1670576	interferon regulatory factor 5	IRF5
ILMN_1670875	protein phosphatase Mg ²⁺ /Mn ²⁺ dependent 1D	PPM1D

ILMN_1670899	fibrillin 2	FBN2
ILMN_1670925	cytochrome b5 domain containing 1	CYB5D1
ILMN_1670926	carbohydrate (N-acetylgalactosamine 4-sulfate 6-O) sulfotransferase	CHST15
ILMN_1670970	protein phosphatase 3 catalytic subunit alpha isozyme	PPP3CA
ILMN_1671048	zinc finger protein 644	ZNF644
ILMN_1671067	mediator complex subunit 13	MED13
ILMN_1671250	chloride intracellular channel 4	CLIC4
ILMN_1671404	supervillin	SVIL
ILMN_1671509	chemokine (C-C motif) ligand 3	CCL3
ILMN_1671703	actin alpha 2 smooth muscle aorta	ACTA2
ILMN_1671731	arginine vasopressin-induced 1	AVPI1
ILMN_1671766	coagulation factor XII (Hageman factor)	F12
ILMN_1671818	urotensin 2	UTS2
ILMN_1671891	phosphotyrosine interaction domain containing 1	PID1
ILMN_1671932	SAMM50 sorting and assembly machinery component	SAMM50
ILMN_1672004	transducer of ERBB2 1	TOB1
ILMN_1672122	prolyl 4-hydroxylase transmembrane (endoplasmic reticulum)	P4HTM
ILMN_1672124	family with sequence similarity 198 member B	FAM198B
ILMN_1672295	zinc finger CCCH-type containing 12A	ZC3H12A
ILMN_1672417	protein tyrosine phosphatase receptor type C-associated protein	PTPRCAP
ILMN_1672503	dihydropyrimidinase-like 2	DPYSL2
ILMN_1672606	2'-5'-oligoadenylate synthetase 1 40/46kDa	OAS1
ILMN_1672650	pyruvate kinase muscle	PKM
ILMN_1672660	myelin basic protein	MBP
ILMN_1672947	calpastatin	CAST
ILMN_1673023	E1A binding protein p400	EP400
ILMN_1673113	coagulation factor II (thrombin) receptor-like 1	F2RL1
ILMN_1673119	AF4/FMR2 family member 1	AFF1
ILMN_1673352	interferon induced transmembrane protein 2	IFITM2
ILMN_1673478	family with sequence similarity 13 member B	FAM13B
ILMN_1673586	solute carrier family 6 (neurotransmitter transporter) member 6	SLC6A6
ILMN_1673960	methionine adenosyltransferase II beta	MAT2B
ILMN_1673966	polymerase (RNA) III (DNA directed) polypeptide F 39 kDa	POLR3F
ILMN_1674063	2'-5'-oligoadenylate synthetase 2 69/71kDa	OAS2
ILMN_1674128	CWC22 spliceosome-associated protein	CWC22
ILMN_1674160	bridging integrator 1	BIN1
ILMN_1674297	host cell factor C2	HCFC2

ILMN_1674302	phosphoribosyl pyrophosphate amidotransferase	PPAT
ILMN_1674394	adipocyte plasma membrane associated protein	APMAP
ILMN_1674574	vanin 1	VNN1
ILMN_1674811	2'-5'-oligoadenylate synthetase-like	OASL
ILMN_1674985	transmembrane protein 51	TMEM51
ILMN_1675085	ubiquitin-like modifier activating enzyme 6	UBA6
ILMN_1675124	DEAD (Asp-Glu-Ala-Asp) box helicase 17	DDX17
ILMN_1675156	cell division cycle 42	CDC42
ILMN_1675190	resistin	RETN
ILMN_1675266	N-acetylglucosamine-1-phosphodiester alpha-N-acetylglucosaminidase	NAGPA
ILMN_1675365	La ribonucleoprotein domain family member 4	LARP4
ILMN_1675448	ZFP36 ring finger protein-like 1	ZFP36L1
ILMN_1675556	XK Kell blood group complex subunit-related family member 8	XKR8
ILMN_1675640	2'-5'-oligoadenylate synthetase 1 40/46kDa	OAS1
ILMN_1675669	inhibitor of Bruton agammaglobulinemia tyrosine kinase	IBTK
ILMN_1675693	protein phosphatase 2 catalytic subunit beta isozyme	PPP2CB
ILMN_1675695	PDS5 regulator of cohesion maintenance homolog B (S. cerevisiae)	PDS5B
ILMN_1675756	potassium inwardly-rectifying channel subfamily J member 15	KCNJ15
ILMN_1675844	WD repeat domain 1	WDR1
ILMN_1675956	lysosomal trafficking regulator	LYST
ILMN_1676254	myotubularin related protein 3	MTMR3
ILMN_1676448	WD repeat and FYVE domain containing 1	WDFY1
ILMN_1676718	Unnamed Transcript	
ILMN_1676984	DNA-damage-inducible transcript 3	DDIT3
ILMN_1677200	cytoplasmic FMR1 interacting protein 2	CYFIP2
ILMN_1677466	dual specificity phosphatase 6	DUSP6
ILMN_1677511	prostaglandin-endoperoxide synthase 2 (prostaglandin G/H synthase a	PTGS2
ILMN_1677574	polypeptide N-acetylgalactosaminyltransferase 6	GALNT6
ILMN_1677607	sterol-C5-desaturase	SC5D
ILMN_1677843	RAB24 member RAS oncogene family	RAB24
ILMN_1677920	lactotransferrin	LTF
ILMN_1678293	Unnamed Transcript	
ILMN_1678490	Rab interacting lysosomal protein-like 2	RILPL2
ILMN_1678517	acyl-CoA synthetase long-chain family member 5	ACSL5
ILMN_1678546	peroxisomal biogenesis factor 11 beta	PEX11B
ILMN_1678707	TAF15 RNA polymerase II TATA box binding protein (TBP)-associated	TAF15

ILMN_1678833	chemokine (C-C motif) receptor 1	CCR1
ILMN_1678974	mitochondrial ribosomal protein L43	MRPL43
ILMN_1679232	kinase D-interacting substrate 220kDa	KIDINS22
ILMN_1679268	pellino E3 ubiquitin protein ligase 1	PELI1
ILMN_1679655	WD repeat domain 82	WDR82
ILMN_1679666	secretoglobin family 3A member 1	SCGB3A1
ILMN_1679727	CDC-like kinase 1	CLK1
ILMN_1679929	Kruppel-like factor 13	KLF13
ILMN_1680192	apolipoprotein B mRNA editing enzyme catalytic polypeptide-like 3A	APOBEC3A
ILMN_1680246	methionine adenosyltransferase II beta	MAT2B
ILMN_1680397	chemokine (C-X-C motif) receptor 2	CXCR2
ILMN_1680424	cathepsin G	CTSG
ILMN_1680465	ADP-ribosylation factor-like 5B	ARL5B
ILMN_1680618	v-myc avian myelocytomatosis viral oncogene homolog	MYC
ILMN_1680624	cellular repressor of E1A-stimulated genes 1	CREG1
ILMN_1680687	N-ethylmaleimide-sensitive factor	NSF
ILMN_1681301	absent in melanoma 2	AIM2
ILMN_1681461	RAB27A member RAS oncogene family	RAB27A
ILMN_1681678	CTD (carboxy-terminal domain RNA polymerase II polypeptide A) sm	CTDSP1
ILMN_1681721	2'-5'-oligoadenylate synthetase-like	OASL
ILMN_1682081	ring finger protein 19B	RNF19B
ILMN_1682098	proteasome (prosome macropain) subunit alpha type 4	PSMA4
ILMN_1682147	hook microtubule-tethering protein 2	HOOK2
ILMN_1682180	valosin containing protein (p97)/p47 complex interacting protein 1	VCPIP1
ILMN_1682323	DEAD (Asp-Glu-Ala-Asp) box polypeptide 51	DDX51
ILMN_1682494	arginine/serine-rich coiled-coil 1	RSRC1
ILMN_1682501	CCR4-NOT transcription complex subunit 1	CNOT1
ILMN_1682636	chemokine (C-X-C motif) ligand 2	CXCL2
ILMN_1682717	immediate early response 3	IER3
ILMN_1682781	TEA domain family member 2	TEAD2
ILMN_1682928	carboxypeptidase vitellogenic-like	CPVL
ILMN_1682930	signal-induced proliferation-associated 1	SIPA1
ILMN_1682938	ADP-ribosylation factor 3	ARF3
ILMN_1682993	natural killer cell granule protein 7	NKG7
ILMN_1683023	platelet derived growth factor C	PDGFC
ILMN_1683026	proteasome (prosome macropain) subunit beta type 10	PSMB10
ILMN_1683127	zinc finger protein 281	ZNF281
ILMN_1683273	small nuclear RNA activating complex polypeptide 5 19kDa	SNAPC5

ILMN_1683313	ST3 beta-galactoside alpha-2 3-sialyltransferase 1	ST3GAL1
ILMN_1683595	methyl-CpG binding domain protein 1	MBD1
ILMN_1683786	Unnamed Transcript	
ILMN_1683792	leucine aminopeptidase 3	LAP3
ILMN_1684585	acyl-CoA synthetase long-chain family member 1	ACSL1
ILMN_1684887	SAM domain SH3 domain and nuclear localization signals 1	SAMSN1
ILMN_1684982	pyruvate dehydrogenase kinase isozyme 4	PDK4
ILMN_1685009	integrin alpha M (complement component 3 receptor 3 subunit)	ITGAM
ILMN_1685057	solute carrier family 22 (organic cation/zwitterion transporter) m	SLC22A4
ILMN_1685122	collagen type IX alpha 2	COL9A2
ILMN_1685289	chromosome 16 open reading frame 58	C16orf58
ILMN_1685327	Unnamed Transcript	
ILMN_1685445	notch 2 N-terminal like	NOTCH2 NL
ILMN_1685521	killer cell lectin-like receptor subfamily F member 1	KLRF1
ILMN_1685625	uncoupling protein 2 (mitochondrial proton carrier)	UCP2
ILMN_1685678	eukaryotic translation elongation factor 1 beta 2	EEF1B2
ILMN_1685810	nuclear factor of activated T-cells cytoplasmic calcineurin-depe	NFATC3
ILMN_1685824	UDP-Gal:betaGlcNAc beta 1 4- galactosyltransferase polypeptide 5	B4GALT5
ILMN_1686116	thrombospondin 1	THBS1
ILMN_1686135	centrosomal protein 95kDa	CEP95
ILMN_1686283	cat eye syndrome chromosome region candidate 1	CECR1
ILMN_1686573	defensin beta 1	DEFB1
ILMN_1686623	colony stimulating factor 1 receptor	CSF1R
ILMN_1686645	UTP14 U3 small nucleolar ribonucleoprotein homolog C (yeast)	UTP14C
ILMN_1686697	F-box protein 38	FBXO38
ILMN_1686862	H2.0-like homeobox	HLX
ILMN_1686884	interleukin 1 receptor accessory protein	IL1RAP
ILMN_1686981	sulfatase 2	SULF2
ILMN_1686989	insulin induced gene 1	INSIG1
ILMN_1687140	StAR-related lipid transfer (START) domain containing 7	STARD7
ILMN_1687247	spermatogenesis associated 20	SPATA20
ILMN_1687301	versican	VCAN
ILMN_1687306	lectin galactoside-binding soluble 2	LGALS2
ILMN_1687315	retinoid X receptor alpha	RXRA
ILMN_1687384	interferon alpha-inducible protein 6	IFI6
ILMN_1687410	oxysterol binding protein-like 11	OSBPL11

ILMN_1687519	synaptosomal-associated protein 23kDa	SNAP23
ILMN_1687533	sema domain immunoglobulin domain (Ig) transmembrane domain (TM)	SEMA4D
ILMN_1687757	Unnamed Transcript	
ILMN_1687785	pyrophosphatase (inorganic) 2	PPA2
ILMN_1687941	ring finger protein 4	RNF4
ILMN_1687998	lysophosphatidylglycerol acyltransferase 1	LPGAT1
ILMN_1688098	TBC1 domain family member 4	TBC1D4
ILMN_1688152	interleukin 27 receptor alpha	IL27RA
ILMN_1688231	triggering receptor expressed on myeloid cells 1	TREM1
ILMN_1688373	leukocyte specific transcript 1	LST1
ILMN_1688452	leucine carboxyl methyltransferase 1	LCMT1
ILMN_1688580	cathelicidin antimicrobial peptide	CAMP
ILMN_1688698	zinc finger E-box binding homeobox 2	ZEB2
ILMN_1688753	phosphatidylserine synthase 1	PTDSS1
ILMN_1688959	CD27 molecule	CD27
ILMN_1689029	WD repeat domain 7	WDR7
ILMN_1689160	dipeptidase 2	DPEP2
ILMN_1689274	non imprinted in Prader-Willi/Angelman syndrome 1	NIPA1
ILMN_1689400	CDC-like kinase 1	CLK1
ILMN_1689734	interleukin 1 receptor antagonist	IL1RN
ILMN_1689836	complement component 5a receptor 1	C5AR1
ILMN_1689953	CD81 molecule	CD81
ILMN_1690105	signal transducer and activator of transcription 1 91kDa	STAT1
ILMN_1690136	Nipped-B homolog (Drosophila)	NIPBL
ILMN_1690476	QKI KH domain containing RNA binding	QKI
ILMN_1690610	RALY heterogeneous nuclear ribonucleoprotein	RALY
ILMN_1690761	surfeit 4	SURF4
ILMN_1690783	triggering receptor expressed on myeloid cells-like 1	TREML1
ILMN_1690822	VAMP (vesicle-associated membrane protein)-associated protein A 33	VAPA
ILMN_1690921	signal transducer and activator of transcription 2 113kDa	STAT2
ILMN_1690999	mediator complex subunit 23	MED23
ILMN_1691071	Fc receptor-like A	FCRLA
ILMN_1691151	choline kinase alpha	CHKA
ILMN_1691276	CXXC finger protein 1	CXXC1
ILMN_1691364	signal transducer and activator of transcription 1 91kDa	STAT1
ILMN_1691508	plasminogen activator urokinase receptor	PLAUR
ILMN_1691693		
ILMN_1691717	rhomoid 5 homolog 2 (Drosophila)	RHBDF2
ILMN_1691747	KH domain containing RNA binding signal transduction associated	KHDRBS3

ILMN_1691846	G0/G1 switch 2	G0S2
ILMN_1691861	Fas-activated serine/threonine kinase	FASTK
ILMN_1691892	transgelin 2	TAGLN2
ILMN_1692145	zinc finger protein 14	ZNF14
ILMN_1692177	TSC22 domain family member 1	TSC22D1
ILMN_1692191	guanine nucleotide binding protein (G protein) alpha 12	GNA12
ILMN_1692223	lipocalin 2	LCN2
ILMN_1692260	v-maf avian musculoaponeurotic fibrosarcoma oncogene homolog G	MAFG
ILMN_1692271	cerebral cavernous malformation 2	CCM2
ILMN_1692705	RecQ helicase-like	RECQL
ILMN_1692742	DENN/MADD domain containing 3	DENND3
ILMN_1692962	CTD (carboxy-terminal domain RNA polymerase II polypeptide A) sm	CTDSP2
ILMN_1693045	transmembrane emp24 protein transport domain containing 1	TMED1
ILMN_1693136	vesicle transport through interaction with t-SNAREs 1B	VTI1B
ILMN_1693220	A kinase (PRKA) anchor protein 11	AKAP11
ILMN_1693242	zinc finger protein 296	ZNF296
ILMN_1693317	trafficking protein particle complex 12	TRAPPC12
ILMN_1693338	cytochrome P450 family 1 subfamily B polypeptide 1	CYP1B1
ILMN_1693352	mitochondrial ribosomal protein L20	MRPL20
ILMN_1693352	mitochondrial ribosomal protein L20	MRPL20
ILMN_1693552	CD300a molecule	CD300A
ILMN_1693650	FES proto-oncogene tyrosine kinase	FES
ILMN_1693771	aspartate beta-hydroxylase	ASPH
ILMN_1693905	histone acetyltransferase 1	HAT1
ILMN_1694058	interferon induced transmembrane protein 4 pseudogene	IFITM4P
ILMN_1694058	interferon induced transmembrane protein 4 pseudogene	IFITM4P
ILMN_1694268	hes family bHLH transcription factor 6	HES6
ILMN_1694274	NADH dehydrogenase (ubiquinone) 1 subcomplex unknown 2 14.5kDa	NDUFC2
ILMN_1694311	nudix (nucleoside diphosphate linked moiety X)-type motif 6	NUDT6
ILMN_1694587	eukaryotic translation elongation factor 1 beta 2	EEF1B2
ILMN_1694603	SWI/SNF related matrix associated actin dependent regulator of c	SMARCC1
ILMN_1694731	chloride channel voltage-sensitive 7	CLCN7
ILMN_1694757	cathepsin L	CTSL
ILMN_1694877	caspase 6 apoptosis-related cysteine peptidase	CASP6
ILMN_1694966	asialoglycoprotein receptor 2	ASGR2
ILMN_1695316	solute carrier family 39 (zinc transporter) member 8	SLC39A8

ILMN_1695404	lymphocyte antigen 6 complex locus E	LY6E
ILMN_1695423	CD9 molecule	CD9
ILMN_1695509	protein tyrosine phosphatase non-receptor type 12	PTPN12
ILMN_1695585	ribosomal protein S26	RPS26
ILMN_1695585	ribosomal protein S26	RPS26
ILMN_1695590	adrenoceptor beta 2 surface	ADRB2
ILMN_1695640	protein tyrosine phosphatase non-receptor type 22 (lymphoid)	PTPN22
ILMN_1695711	family with sequence similarity 105 member A	FAM105A
ILMN_1695744	leukocyte immunoglobulin-like receptor subfamily B (with TM and IT)	LILRB2
ILMN_1695827	protein phosphatase 1 catalytic subunit alpha isozyme	PPP1CA
ILMN_1696021	karyopherin alpha 6 (importin alpha 7)	KPNA6
ILMN_1696041	cell division cycle 42	CDC42
ILMN_1696318	uncharacterized LOC100130691	LOC10013
ILMN_1696360	cathepsin B	CTSB
ILMN_1696419	stomatin	STOM
ILMN_1696420	bromodomain containing 7	BRD7
ILMN_1696432	isocitrate dehydrogenase 1 (NADP+) soluble	IDH1
ILMN_1696463	Spi-1 proto-oncogene	SPI1
ILMN_1696512	alpha hemoglobin stabilizing protein	AHSP
ILMN_1696584	orosomuroid 1	ORM1
ILMN_1696654	interferon-induced protein with tetratricopeptide repeats 5	IFIT5
ILMN_1696749	lamin A/C	LMNA
ILMN_1696806	Unnamed Transcript	
ILMN_1696933	NLR family pyrin domain containing 3	NLRP3
ILMN_1696975	ubiquitin specific peptidase 1	USP1
ILMN_1697268	elastin microfibril interfacier 2	EMILIN2
ILMN_1697309	neutrophil cytosolic factor 1	NCF1
ILMN_1697469	serine/arginine-rich splicing factor 6	SRSF6
ILMN_1697957	KIAA0930	KIAA0930
ILMN_1698243	chromosome 1 open reading frame 85	C1orf85
ILMN_1698246	Unnamed Transcript	
ILMN_1698365	NHL repeat containing 3	NHLRC3
ILMN_1699071	MAP3K7 C-terminal like	MAP3K7C L
ILMN_1699358	guanine nucleotide binding protein (G protein) alpha 12	GNA12
ILMN_1699423	zinc finger protein 146	ZNF146
ILMN_1699570	tumor protein D52-like 2	TPD52L2
ILMN_1699695	tumor necrosis factor receptor superfamily member 21	TNFRSF2 1
ILMN_1699836	lymphocyte-specific protein 1	LSP1

ILMN_1699878	RAB27A member RAS oncogene family	RAB27A
ILMN_1699908	interleukin 12 receptor beta 1	IL12RB1
ILMN_1700047	aminolevulinate delta- synthase 1	ALAS1
ILMN_1700147	pre-B lymphocyte 3	VPREB3
ILMN_1700202	transmembrane protein 135	TMEM135
ILMN_1700306	OCIA domain containing 2	OCIAD2
ILMN_1700340	asialoglycoprotein receptor 2	ASGR2
ILMN_1700413	v-maf avian musculoaponeurotic fibrosarcoma oncogene homolog F	MAFF
ILMN_1700428	major histocompatibility complex class II DO beta	HLA-DOB
ILMN_1700549	ER lipid raft associated 2	ERLIN2
ILMN_1700584	immediate early response 2	IER2
ILMN_1700610	C-type lectin domain family 7 member A	CLEC7A
ILMN_1700628	DEAD (Asp-Glu-Ala-Asp) box helicase 24	DDX24
ILMN_1700695	solute carrier family 44 (choline transporter) member 1	SLC44A1
ILMN_1701006	hexamethylene bis-acetamide inducible 2	HEXIM2
ILMN_1701111	glycerophosphocholine phosphodiesterase GDE1 homolog (<i>S. cerevisiae</i>)	GPCPD1
ILMN_1701114	guanylate binding protein 1 interferon-inducible	GBP1
ILMN_1701114	guanylate binding protein 1 interferon-inducible	GBP1
ILMN_1701195	phospholipase A2 group VII (platelet-activating factor acetylhydro	PLA2G7
ILMN_1701237	SH2 domain containing 1B	SH2D1B
ILMN_1701466	peroxisomal biogenesis factor 16	PEX16
ILMN_1701711	bromodomain containing 8	BRD8
ILMN_1701789	interferon-induced protein with tetratricopeptide repeats 3	IFIT3
ILMN_1701906	CD300c molecule	CD300C
ILMN_1701930	eukaryotic translation elongation factor 1 beta 2	EEF1B2
ILMN_1701933	synuclein alpha (non A4 component of amyloid precursor)	SNCA
ILMN_1702114	Unnamed Transcript	
ILMN_1702301	dedicator of cytokinesis 10	DOCK10
ILMN_1702592	WD repeat domain 73	WDR73
ILMN_1702635	kelch domain containing 10	KLHDC10
ILMN_1702691	tumor necrosis factor alpha-induced protein 3	TNFAIP3
ILMN_1702759	thioredoxin-related transmembrane protein 4	TMX4
ILMN_1702763	zinc finger MYM-type 1	ZMYM1
ILMN_1702787	sema domain immunoglobulin domain (Ig) transmembrane domain (TM)	SEMA4A
ILMN_1703108	ubiquitin-conjugating enzyme E2L 6	UBE2L6
ILMN_1703123	cysteine-serine-rich nuclear protein 1	CSRNP1
ILMN_1703180	ets variant 3	ETV3
ILMN_1703229	Unnamed Transcript	

ILMN_1703305	twinfilin actin-binding protein 2	TWF2
ILMN_1703330	fem-1 homolog c (C. elegans)	FEM1C
ILMN_1703427	SON DNA binding protein	SON
ILMN_1703617	AHA1 activator of heat shock 90kDa protein ATPase homolog 1 (yeast)	AHSA1
ILMN_1703622	peptidylprolyl isomerase B (cyclophilin B)	PPIB
ILMN_1703650	TNFAIP3 interacting protein 1	TNIP1
ILMN_1703697	LanC lantibiotic synthetase component C-like 1 (bacterial)	LANCL1
ILMN_1703891	TBC1 domain family member 9 (with GRAM domain)	TBC1D9
ILMN_1703946	adenosine A2b receptor	ADORA2B
ILMN_1703949	karyopherin (importin) beta 1	KPNB1
ILMN_1704236	MYC associated factor X	MAX
ILMN_1704404	proteasome (prosome macropain) 26S subunit non-ATPase 13	PSMD13
ILMN_1704672	nucleic acid binding protein 2	NABP2
ILMN_1704713	casein kinase 1 gamma 1	CSNK1G1
ILMN_1704730	CD93 molecule	CD93
ILMN_1704797	C-type lectin domain family 10 member A	CLEC10A
ILMN_1704980	ERI1 exoribonuclease family member 3	ERI3
ILMN_1704985	cytochrome P450 family 27 subfamily A polypeptide 1	CYP27A1
ILMN_1705111	fibronectin type III domain containing 3A	FNDC3A
ILMN_1705141	calcyclin binding protein	CACYBP
ILMN_1705266	v-rel avian reticuloendotheliosis viral oncogene homolog A	RELA
ILMN_1705663	Dmx-like 2	DMXL2
ILMN_1705686	neurogranin (protein kinase C substrate RC3)	NRGN
ILMN_1705783	nuclear RNA export factor 1	NXF1
ILMN_1705928	small nuclear ribonucleoprotein 200kDa (U5)	SNRNP200
ILMN_1706015	family with sequence similarity 43 member A	FAM43A
ILMN_1706217	toll-like receptor 4	TLR4
ILMN_1706261	solute carrier organic anion transporter family member 3A1	SLCO3A1
ILMN_1706413	ribosomal RNA adenine dimethylase domain containing 1	RRNAD1
ILMN_1706502	eukaryotic translation initiation factor 2-alpha kinase 2	EIF2AK2
ILMN_1706523	Fc fragment of IgG low affinity IIa receptor (CD32)	FCGR2A
ILMN_1706546	MYC associated factor X	MAX
ILMN_1706553	SMG7 nonsense mediated mRNA decay factor	SMG7
ILMN_1706635	elastase neutrophil expressed	ELANE
ILMN_1706784	H2A histone family member V	H2AFV
ILMN_1707062	REV1 polymerase (DNA directed)	REV1
ILMN_1707077	sortilin 1	SORT1

ILMN_1707137	chromosome 17 open reading frame 97	C17orf97
ILMN_1707312	nuclear factor interleukin 3 regulated	NFIL3
ILMN_1707336	actin related protein 2/3 complex subunit 4 20kDa	ARPC4
ILMN_1707339	BTG family member 3	BTG3
ILMN_1707481	zinc finger and BTB domain containing 44	ZBTB44
ILMN_1707551	arylformamidase	AFMID
ILMN_1707695	interferon-induced protein with tetratricopeptide repeats 1	IFIT1
ILMN_1707815	Unnamed Transcript	
ILMN_1708041	pleckstrin homology domain containing family F (with FYVE domain)	PLEKHF1
ILMN_1708164	eukaryotic translation initiation factor 3 subunit A	EIF3A
ILMN_1708340	death-associated protein kinase 1	DAPK1
ILMN_1708348	ADAM metallopeptidase domain 8	ADAM8
ILMN_1708416	ADP-ribosylation factor-like 6 interacting protein 1	ARL6IP1
ILMN_1708721	RAB GTPase activating protein 1-like	RABGAP1 L
ILMN_1708728	H2A histone family member J	H2AFJ
ILMN_1708779	granulysin	GNLY
ILMN_1708881	RAB20 member RAS oncogene family	RAB20
ILMN_1708934	adrenomedullin	ADM
ILMN_1708946	vacuolar protein sorting 4 homolog A (S. cerevisiae)	VPS4A
ILMN_1709032	FYVE and coiled-coil domain containing 1	FYCO1
ILMN_1709233	coagulation factor V (proaccelerin labile factor)	F5
ILMN_1709333	2'-5'-oligoadenylate synthetase 2 69/71kDa	OAS2
ILMN_1709334	transmembrane 9 superfamily member 1	TM9SF1
ILMN_1709439	charged multivesicular body protein 1A	CHMP1A
ILMN_1709683	Ras association (RalGDS/AF-6) domain family member 2	RASSF2
ILMN_1709817	solute carrier family 35 member A5	SLC35A5
ILMN_1710017	CD79b molecule immunoglobulin-associated beta	CD79B
ILMN_1710150	embryonic ectoderm development	EED
ILMN_1710280	tubulin beta 1 class VI	TUBB1
ILMN_1710514	B-cell CLL/lymphoma 3	BCL3
ILMN_1710734	granzyme K (granzyme 3; tryptase II)	GZMK
ILMN_1710937	interferon gamma-inducible protein 16	IFI16
ILMN_1711048	zinc finger and BTB domain containing 17	ZBTB17
ILMN_1711189	exosome component 10	EXOSC10
ILMN_1711199	zinc finger protein 331	ZNF331
ILMN_1711414	mitochondrial ribosomal protein S27	MRPS27
ILMN_1711453	Unnamed Transcript	
ILMN_1711490	Rho GTPase activating protein 26	ARHGAP2 6
ILMN_1711493	PML-RARA regulated adaptor molecule 1	PRAM1

ILMN_1711786	nuclear factor erythroid 2	NFE2
ILMN_1711799	chromosome 9 open reading frame 40	C9orf40
ILMN_1711828	ankyrin repeat domain 10	ANKRD10
ILMN_1712019	ankyrin repeat domain 17	ANKRD17
ILMN_1712026	NLR family pyrin domain containing 3	NLRP3
ILMN_1712400	serpin peptidase inhibitor clade B (ovalbumin) member 6	SERPINB6
ILMN_1712532	caspase recruitment domain family member 9	CARD9
ILMN_1712577	family with sequence similarity 174 member A	FAM174A
ILMN_1712673	SAM and SH3 domain containing 1	SASH1
ILMN_1712773	sperm associated antigen 1	SPAG1
ILMN_1712918	NAD(P)H dehydrogenase quinone 2	NQO2
ILMN_1712944	amino-terminal enhancer of split	AES
ILMN_1712959	dual specificity phosphatase 2	DUSP2
ILMN_1713124	aldo-keto reductase family 1 member C3	AKR1C3
ILMN_1713162	glutathione S-transferase mu 2 (muscle)	GSTM2
ILMN_1713163	SWI/SNF related matrix associated actin dependent regulator of c	SMARCA5
ILMN_1713266	family with sequence similarity 46 member C	FAM46C
ILMN_1713505	Niemann-Pick disease type C1	NPC1
ILMN_1713603	protein kinase C beta	PRKCB
ILMN_1713668	translin-associated factor X	TSNAX
ILMN_1713749	coronin actin binding protein 1A	CORO1A
ILMN_1713752	serine incorporator 3	SERINC3
ILMN_1713803	chromosome 17 open reading frame 97	C17orf97
ILMN_1714093	required for meiotic nuclear division 5 homolog A (S. cerevisiae)	RMND5A
ILMN_1714159	leucine zipper protein 1	LUZP1
ILMN_1714393	RAB24 member RAS oncogene family	RAB24
ILMN_1714418	family with sequence similarity 101 member B	FAM101B
ILMN_1714650	RAS guanyl releasing protein 4	RASGRP4
ILMN_1714896	squamous cell carcinoma antigen recognized by T cells 3	SART3
ILMN_1714965	nuclear factor of kappa light polypeptide gene enhancer in B-cells	NFKB1
ILMN_1715068	aquaporin 9	AQP9
ILMN_1715169	major histocompatibility complex class II DR beta 1	HLA-DRB1
ILMN_1715416	nucleoporin 188kDa	NUP188
ILMN_1715636	eukaryotic translation initiation factor 3 subunit B	EIF3B
ILMN_1715969	solute carrier family 25 (mitochondrial iron transporter) member 3	SLC25A37
ILMN_1715991	serum deprivation response	SDPR
ILMN_1716105	NLR family pyrin domain containing 12	NLRP12

ILMN_1716276	Unnamed Transcript	
ILMN_1716446	B-cell CLL/lymphoma 10	BCL10
ILMN_1716563	protein kinase C beta	PRKCB
ILMN_1717063	F-box protein 9	FBXO9
ILMN_1717163	coagulation factor XIII A1 polypeptide	F13A1
ILMN_1717180	myotubularin related protein 6	MTMR6
ILMN_1717261	major histocompatibility complex class II DR beta 3	HLA-DRB3
ILMN_1717313	nuclear factor of kappa light polypeptide gene enhancer in B-cells	NFKBIE
ILMN_1717639	salt-inducible kinase 1	SIK1
ILMN_1717809	ring finger protein 24	RNF24
ILMN_1717973	Unnamed Transcript	
ILMN_1718023	acylaminoacyl-peptide hydrolase	APEH
ILMN_1718042	zinc finger protein 549	ZNF549
ILMN_1718063	lipase A lysosomal acid cholesterol esterase	LIPA
ILMN_1718558	poly (ADP-ribose) polymerase family member 12	PARP12
ILMN_1718565	cyclin-dependent kinase inhibitor 1C (p57 Kip2)	CDKN1C
ILMN_1718633	low density lipoprotein receptor-related protein 5-like	LRP5L
ILMN_1718718	McKusick-Kaufman syndrome	MKKS
ILMN_1718807	structural maintenance of chromosomes 3	SMC3
ILMN_1718932	5-methyltetrahydrofolate-homocysteine methyltransferase reductase	MTRR
ILMN_1718936	leukocyte specific transcript 1	LST1
ILMN_1718960	serpin peptidase inhibitor clade B (ovalbumin) member 8	SERPINB8
ILMN_1718977	growth arrest and DNA-damage-inducible beta	GADD45B
ILMN_1719163	zinc finger protein 557	ZNF557
ILMN_1719185	Unnamed Transcript	
ILMN_1719232	DiGeorge syndrome critical region gene 14	DGCR14
ILMN_1719316	transmembrane emp24 protein transport domain containing 3	TMED3
ILMN_1719392	fumarate hydratase	FH
ILMN_1719433	CD1d molecule	CD1D
ILMN_1719611		
ILMN_1719627	solute carrier family 27 (fatty acid transporter) member 3	SLC27A3
ILMN_1719695	nuclear factor of kappa light polypeptide gene enhancer in B-cells	NFKBIZ
ILMN_1719905	toll-like receptor 10	TLR10
ILMN_1720048	chemokine (C-C motif) ligand 2	CCL2
ILMN_1720088	splicing regulatory glutamine/lysine-rich protein 1	SREK1
ILMN_1720158	v-ets avian erythroblastosis virus E26 oncogene homolog 2	ETS2
ILMN_1720303	osteopetrosis associated transmembrane protein 1	OSTM1

ILMN_1720373	solute carrier family 7 (amino acid transporter light chain L syst	SLC7A5
ILMN_1720513	SET binding protein 1	SETBP1
ILMN_1720623	synaptotagmin-like 3	SYTL3
ILMN_1720771	syntaxin 11	STX11
ILMN_1720996	solute carrier family 12 (sodium/potassium/chloride transporter) m	SLC12A2
ILMN_1721008	deoxyuridine triphosphatase	DUT
ILMN_1721026	SAM domain and HD domain 1	SAMHD1
ILMN_1721081	Sp4 transcription factor	SP4
ILMN_1721138	GrpE-like 2 mitochondrial (E. coli)	GRPEL2
ILMN_1721868	karyopherin alpha 2 (RAG cohort 1 importin alpha 1)	KPNA2
ILMN_1721876	TIMP metalloproteinase inhibitor 2	TIMP2
ILMN_1721978	caspase recruitment domain family member 11	CARD11
ILMN_1722059	scaffold attachment factor B	SAFB
ILMN_1722276	platelet-activating factor acetylhydrolase 1b regulatory subunit 1	PAFAH1B1
ILMN_1722294	copine VIII	CPNE8
ILMN_1722502	chaperonin containing TCP1 subunit 6A (zeta 1)	CCT6A
ILMN_1722532	lysine (K)-specific demethylase 3A	KDM3A
ILMN_1722622	CD163 molecule	CD163
ILMN_1722698	ring finger and CHY zinc finger domain containing 1 E3 ubiquitin p	RCHY1
ILMN_1722811	cyclin-dependent kinase inhibitor 1B (p27 Kip1)	CDKN1B
ILMN_1722838	mitochondrial ribosomal protein L46	MRPL46
ILMN_1722981	toll-like receptor 5	TLR5
ILMN_1723035	oxidized low density lipoprotein (lectin-like) receptor 1	OLR1
ILMN_1723043	napsin B aspartic peptidase pseudogene	NAPSB
ILMN_1723115	C-type lectin domain family 4 member F	CLEC4F
ILMN_1723480	bone marrow stromal cell antigen 2	BST2
ILMN_1723486	hexokinase 2	HK2
ILMN_1723912	interferon-induced protein 44-like	IFI44L
ILMN_1723969	phospholipase C beta 1 (phosphoinositide-specific)	PLCB1
ILMN_1724250	granulin	GRN
ILMN_1724495	SEC14 and spectrin domains 1	SESTD1
ILMN_1724658	BCL2/adenovirus E1B 19kDa interacting protein 3	BNIP3
ILMN_1724837	zinc finger CCCH-type antiviral 1	ZC3HAV1
ILMN_1724984	eukaryotic translation initiation factor 2-alpha kinase 3	EIF2AK3
ILMN_1725175	FOS-like antigen 2	FOSL2
ILMN_1725244	histone acetyltransferase 1	HAT1
ILMN_1726189	membrane-spanning 4-domains subfamily A member 14	MS4A14

ILMN_1726288	transmembrane protein 106B	TMEM106B
ILMN_1726289	KIAA1551	KIAA1551
ILMN_1726545	leukocyte immunoglobulin-like receptor subfamily A (with TM domain)	LILRA5
ILMN_1726574	calcyclin binding protein	CACYBP
ILMN_1726597	family with sequence similarity 65 member B	FAM65B
ILMN_1726693	general transcription factor IIIH polypeptide 1 62kDa	GTF2H1
ILMN_1726769	CNDP dipeptidase 2 (metallopeptidase M20 family)	CNDP2
ILMN_1727051	DEAD (Asp-Glu-Ala-Asp) box polypeptide 19A	DDX19A
ILMN_1727098	protein phosphatase 1 regulatory subunit 16B	PPP1R16B
ILMN_1727150	dehydrogenase/reductase (SDR family) member 9	DHRS9
ILMN_1727271	tryptophanyl-tRNA synthetase	WARS
ILMN_1727761	glucocorticoid modulatory element binding protein 1	GMEB1
ILMN_1727762	caspase 1 apoptosis-related cysteine peptidase	CASP1
ILMN_1727965	aldehyde dehydrogenase 3 family member B1	ALDH3B1
ILMN_1728047	aldo-keto reductase family 1 member A1 (aldehyde reductase)	AKR1A1
ILMN_1728071	Kirsten rat sarcoma viral oncogene homolog	KRAS
ILMN_1728106	tumor necrosis factor	TNF
ILMN_1728163	CTD (carboxy-terminal domain RNA polymerase II polypeptide A) sm	CTDSP1
ILMN_1728298	SH3 domain binding kinase 1	SBK1
ILMN_1728471	ADP-ribosylation factor guanine nucleotide-exchange factor 1 (brefe	ARFGEF1
ILMN_1728478	chemokine (C-X-C motif) ligand 16	CXCL16
ILMN_1728498	poly(rC) binding protein 4	PCBP4
ILMN_1728662	aldehyde dehydrogenase 3 family member B1	ALDH3B1
ILMN_1728677	cAMP responsive element binding protein 5	CREB5
ILMN_1728698	glycerophosphodiester phosphodiesterase 1	GDE1
ILMN_1728984	proliferation-associated 2G4 38kDa	PA2G4
ILMN_1729175	F-box protein 3	FBXO3
ILMN_1729234	tripeptidyl peptidase I	TPP1
ILMN_1729417	glucosamine (UDP-N-acetyl)-2-epimerase/N-acetylmannosamine kinase	GNE
ILMN_1729453	tetraspanin 9	TSPAN9
ILMN_1729563	UDP-glucose 6-dehydrogenase	UGDH
ILMN_1729691	solute carrier family 16 member 6	SLC16A6
ILMN_1729775	optic atrophy 1 (autosomal dominant)	OPA1
ILMN_1729915	paired immunoglobulin-like type 2 receptor alpha	PILRA
ILMN_1729973	zinc finger CCCH-type antiviral 1	ZC3HAV1
ILMN_1729976	chromosome alignment maintaining phosphoprotein 1	CHAMP1

ILMN_1730084	catechol-O-methyltransferase	COMT
ILMN_1730118	zinc finger protein 644	ZNF644
ILMN_1730307	mediator complex subunit 16	MED16
ILMN_1730416	cytochrome c somatic	CYCS
ILMN_1730454	folate receptor 3 (gamma)	FOLR3
ILMN_1730628	ribonuclease RNase A family 2 (liver eosinophil-derived neuroto	RNASE2
ILMN_1730631	chromosome 2 open reading frame 44	C2orf44
ILMN_1730639	solute carrier family 22 member 15	SLC22A15
ILMN_1730660	lectin galactoside-binding soluble 3	LGALS3
ILMN_1730816	G protein-coupled receptor 162	GPR162
ILMN_1730986	mucosa associated lymphoid tissue lymphoma translocation gene 1	MALT1
ILMN_1731048	toll-like receptor 1	TLR1
ILMN_1731107	coiled-coil domain containing 92	CCDC92
ILMN_1731113	zinc finger and BTB domain containing 43	ZBTB43
ILMN_1731123	ring finger protein 7	RNF7
ILMN_1731233	granzyme H (cathepsin G-like 2 protein h-CCPX)	GZMH
ILMN_1731714	cAMP responsive element binding protein 5	CREB5
ILMN_1732198	urotensin 2	UTS2
ILMN_1732216	asparaginyl-tRNA synthetase	NARS
ILMN_1732296	inhibitor of DNA binding 3 dominant negative helix-loop-helix prot	ID3
ILMN_1732452	mitogen-activated protein kinase-activated protein kinase 3	MAPKAP K3
ILMN_1732475	serine/threonine/tyrosine interacting protein	STYX
ILMN_1732575	SEC14-like 1 (<i>S. cerevisiae</i>)	SEC14L1
ILMN_1732688	deoxyuridine triphosphatase	DUT
ILMN_1732810	sorting nexin 17	SNX17
ILMN_1733221	sema domain immunoglobulin domain (Ig) transmembrane domain (TM)	SEMA4D
ILMN_1733248	nuclear receptor binding protein 2	NRBP2
ILMN_1733270	CD163 molecule	CD163
ILMN_1733956	isoleucyl-tRNA synthetase	IARS
ILMN_1733997		
ILMN_1733998	dehydrogenase/reductase (SDR family) member 9	DHRS9
ILMN_1734748	leukocyte immunoglobulin-like receptor subfamily A (with TM domain	LILRA1
ILMN_1734855	selectin P ligand	SELPLG
ILMN_1734878	CD79a molecule immunoglobulin-associated alpha	CD79A
ILMN_1735151	eukaryotic translation initiation factor 5A2	EIF5A2
ILMN_1735333	neighbor of BRCA1 gene 1	NBR1

ILMN_1735453	family with sequence similarity 98 member A	FAM98A
ILMN_1735792	rhomoid 5 homolog 2 (Drosophila)	RHBDF2
ILMN_1735908	UTP15 U3 small nucleolar ribonucleoprotein homolog (S. cerevisia	UTP15
ILMN_1736068	CCR4-NOT transcription complex subunit 8	CNOT8
ILMN_1736180	frequently rearranged in advanced T-cell lymphomas 1	FRAT1
ILMN_1736568	caspase 2 apoptosis-related cysteine peptidase	CASP2
ILMN_1736585	transmembrane emp24-like trafficking protein 10 (yeast)	TMED10
ILMN_1736700	aldolase A fructose-bisphosphate	ALDOA
ILMN_1736729	2'-5'-oligoadenylate synthetase 2 69/71kDa	OAS2
ILMN_1736863	transmembrane protein 140	TMEM140
ILMN_1736982	phosphatase and actin regulator 1	PHACTR1
ILMN_1737003	Unnamed Transcript	
ILMN_1737025	phospholipase C-like 2	PLCL2
ILMN_1737084	taxilin alpha	TXLNA
ILMN_1737164	transmembrane 9 superfamily member 1	TM9SF1
ILMN_1737252	neuregulin 1	NRG1
ILMN_1737254	ubiquitin specific peptidase 1	USP1
ILMN_1737314	B-cell CLL/lymphoma 6	BCL6
ILMN_1737394	lamin A/C	LMNA
ILMN_1737396	proteasome (prosome macropain) 26S subunit non-ATPase 14	PSMD14
ILMN_1737514	kynureninase	KYNU
ILMN_1737627	mitogen-activated protein kinase 14	MAPK14
ILMN_1737705	UHRF1 binding protein 1-like	UHRF1BP1
ILMN_1737813	ERI1 exoribonuclease family member 3	ERI3
ILMN_1737947	LSM5 homolog U6 small nuclear RNA associated (S. cerevisiae)	LSM5
ILMN_1738207	cytokine inducible SH2-containing protein	CISH
ILMN_1738424	cell division cycle 42	CDC42
ILMN_1738632	protein kinase cAMP-dependent regulatory type I alpha	PRKAR1A
ILMN_1738725	leukemia inhibitory factor	LIF
ILMN_1738909	TROVE domain family member 2	TROVE2
ILMN_1738921	acetyl-CoA acyltransferase 1	ACAA1
ILMN_1738992	myeloid cell nuclear differentiation antigen	MNDA
ILMN_1739001	tumor-associated calcium signal transducer 2	TACSTD2
ILMN_1739032	transmembrane protein 70	TMEM70
ILMN_1739151	mannosyl (alpha-1 6-)-glycoprotein beta-1 2-N-acetylglucosaminyltr	MGAT2
ILMN_1739586	fasciculation and elongation protein zeta 2 (zygin II)	FEZ2
ILMN_1739605	LY6/PLAUR domain containing 3	LYPD3

ILMN_1739641	myotubularin related protein 3	MTMR3
ILMN_1739674	Unnamed Transcript	
ILMN_1739815	NLR family pyrin domain containing 12	NLRP12
ILMN_1740015	CD14 molecule	CD14
ILMN_1740171	dual specificity phosphatase 11 (RNA/RNP complex 1-interacting)	DUSP11
ILMN_1740319	interferon alpha-inducible protein 27-like 2	IFI27L2
ILMN_1740426	RAS dexamethasone-induced 1	RASD1
ILMN_1740572	transcobalamin II	TCN2
ILMN_1740597	cyclin-dependent kinase inhibitor 2D (p19 inhibits CDK4)	CDKN2D
ILMN_1740604	RAB11 family interacting protein 5 (class I)	RAB11FIP
ILMN_1740633	perforin 1 (pore forming protein)	PRF1
ILMN_1740737	decapping enzyme scavenger	DCPS
ILMN_1740742	uroporphyrinogen decarboxylase	UROD
ILMN_1740819	StAR-related lipid transfer (START) domain containing 7	STARD7
ILMN_1740875	formyl peptide receptor 2	FPR2
ILMN_1741003	annexin A5	ANXA5
ILMN_1741148	aldolase A fructose-bisphosphate	ALDOA
ILMN_1741159	mitogen-activated protein kinase kinase kinase 8	MAP3K8
ILMN_1741165	solute carrier family 11 (proton-coupled divalent metal ion transpo	SLC11A1
ILMN_1741464	hook microtubule-tethering protein 3	HOOK3
ILMN_1741881	chromosome 9 open reading frame 72	C9orf72
ILMN_1741917	osteoclast associated immunoglobulin-like receptor	OSCAR
ILMN_1741942	syntaxin 16	STX16
ILMN_1742052	serpin peptidase inhibitor clade B (ovalbumin) member 9	SERPINB9
ILMN_1742324	SUN domain containing ossification factor	SUCO
ILMN_1742332	potassium channel tetramerization domain containing 12	KCTD12
ILMN_1742400	centrosomal protein 350kDa	CEP350
ILMN_1742544	myocyte enhancer factor 2C	MEF2C
ILMN_1742618	XIAP associated factor 1	XAF1
ILMN_1742981	tubulin alpha 1a	TUBA1A
ILMN_1743145	endoplasmic reticulum aminopeptidase 2	ERAP2
ILMN_1743187	chromosome 6 open reading frame 120	C6orf120
ILMN_1743241	ADP-ribosylation factor-like 4A	ARL4A
ILMN_1743290	glycoprotein IX (platelet)	GP9
ILMN_1743619	neural precursor cell expressed developmentally down-regulated 9	NEDD9
ILMN_1743966	B-cell CLL/lymphoma 9-like	BCL9L
ILMN_1744113	tumor necrosis factor alpha-induced protein 8-like 2	TNFAIP8L
ILMN_1744517	glucosamine (N-acetyl)-6-sulfatase	GNS
ILMN_1744649	proteasome (prosome macropain) subunit beta type 5	PSMB5

ILMN_1744963	ERO1-like (<i>S. cerevisiae</i>)	ERO1L
ILMN_1745075	ribosomal protein large P0	RPLP0
ILMN_1745103	C-type lectin domain family 1 member B	CLEC1B
ILMN_1745242	phospholipid scramblase 1	PLSCR1
ILMN_1745356	chemokine (C-X-C motif) ligand 9	CXCL9
ILMN_1745374	interferon-induced protein 35	IFI35
ILMN_1745397	2'-5'-oligoadenylate synthetase 3 100kDa	OAS3
ILMN_1745423	UTP3 small subunit (SSU) processome component homolog (<i>S. cerevi</i>	UTP3
ILMN_1745573	tetratricopeptide repeat domain 13	TTC13
ILMN_1745772	activating signal cointegrator 1 complex subunit 3	ASCC3
ILMN_1745788	chemokine (C-X3-C motif) receptor 1	CX3CR1
ILMN_1745964	interleukin-1 receptor-associated kinase 2	IRAK2
ILMN_1745994	growth arrest-specific 7	GAS7
ILMN_1746243	testis derived transcript (3 LIM domains)	TES
ILMN_1746317	nucleoporin like 1	NUPL1
ILMN_1746426	translocase of outer mitochondrial membrane 70 homolog A (<i>S. cerevi</i>	TOMM70A
ILMN_1746517	kynureninase	KYNU
ILMN_1746664	WD repeat and SOCS box containing 2	WSB2
ILMN_1746696	PDS5 regulator of cohesion maintenance homolog B (<i>S. cerevisiae</i>)	PDS5B
ILMN_1746836	platelet-activating factor receptor	PTAFR
ILMN_1746846	tubulin tyrosine ligase-like family member 4	TTLL4
ILMN_1747052	integrin alpha 4 (antigen CD49D alpha 4 subunit of VLA-4 recepto	ITGA4
ILMN_1747058	trafficking protein particle complex 2-like	TRAPPC2L
ILMN_1747195	proteasome (prosome macropain) subunit beta type 8	PSMB8
ILMN_1747355	Unnamed Transcript	
ILMN_1747504	AT hook containing transcription factor 1	AHCTF1
ILMN_1747598	protein phosphatase 1 regulatory (inhibitor) subunit 11	PPP1R11
ILMN_1747622	CD33 molecule	CD33
ILMN_1747630	DEK proto-oncogene	DEK
ILMN_1747744	lipoma HMGIC fusion partner-like 2	LHFPL2
ILMN_1747870	CD3e molecule epsilon associated protein	CD3EAP
ILMN_1748077	DEAD (Asp-Glu-Ala-Asp) box polypeptide 59	DDX59
ILMN_1748147	mitochondrial tRNA translation optimization 1	MTO1
ILMN_1748473	GTPase IMAP family member 4	GIMAP4
ILMN_1748883	cyclin-dependent kinase inhibitor 2D (p19 inhibits CDK4)	CDKN2D
ILMN_1748911	synaptosomal-associated protein 23kDa	SNAP23
ILMN_1748915	S100 calcium binding protein A12	S100A12

ILMN_1749011	NECAP endocytosis associated 2	NECAP2
ILMN_1749078	TIMP metallopeptidase inhibitor 2	TIMP2
ILMN_1749253	tubulin delta 1	TUBD1
ILMN_1749317	cilia and flagella associated protein 74	CFAP74
ILMN_1749368	histone cluster 1 H3h	HIST1H3H
ILMN_1749419	protein kinase AMP-activated gamma 2 non-catalytic subunit	PRKAG2
ILMN_1749641	F-box protein 3	FBXO3
ILMN_1750075	cyclin D binding myb-like transcription factor 1	DMTF1
ILMN_1750273	ribosomal protein L23a pseudogene 7	RPL23AP7
ILMN_1750395	methyl-CpG binding domain protein 2	MBD2
ILMN_1750400	chromosome 19 open reading frame 66	C19orf66
ILMN_1750401	chromosome 17 open reading frame 62	C17orf62
ILMN_1750636	ribosomal protein S26	RPS26
ILMN_1750805	Rho GTPase activating protein 30	ARHGAP30
ILMN_1751072	signal recognition particle receptor B subunit	SRPRB
ILMN_1751079	transporter 1 ATP-binding cassette sub-family B (MDR/TAP)	TAP1
ILMN_1751164	Rho GTPase activating protein 30	ARHGAP30
ILMN_1751400	src kinase associated phosphoprotein 1	SKAP1
ILMN_1751589	NudC domain containing 2	NUDCD2
ILMN_1751607	FBJ murine osteosarcoma viral oncogene homolog B	FOSB
ILMN_1752249	piezo-type mechanosensitive ion channel component 1	PIEZO1
ILMN_1752478	dehydrogenase/reductase (SDR family) member 3	DHRS3
ILMN_1752526	ring finger protein 144B	RNF144B
ILMN_1752591	leptin receptor overlapping transcript-like 1	LEPROTL1
ILMN_1752606	Unnamed Transcript	
ILMN_1752932	myelin protein zero-like 2	MPZL2
ILMN_1753064	tetratricopeptide repeat domain 13	TTC13
ILMN_1753111	nicotinamide phosphoribosyltransferase	NAMPT
ILMN_1753498	CoA synthase	COASY
ILMN_1753613	homeobox A5	HOXA5
ILMN_1753663	ADP-ribosylation factor-like 4A	ARL4A
ILMN_1753805	protein kinase D2	PRKD2
ILMN_1754421	NADH dehydrogenase (ubiquinone) complex I assembly factor 1	NDUFAF1
ILMN_1754788	guanosine monophosphate reductase 2	GMPR2
ILMN_1755115	ribosomal protein L23	RPL23

ILMN_1755983	nuclear factor of activated T-cells cytoplasmic calcineurin-depe	NFATC3
ILMN_1756086	integrator complex subunit 3	INTS3
ILMN_1756417	ankyrin repeat domain 37	ANKRD37
ILMN_1756723	dipeptidyl-peptidase 7	DPP7
ILMN_1756779	clathrin heavy chain (Hc)	CLTC
ILMN_1756806	myeloid cell leukemia 1	MCL1
ILMN_1756928	reticulon 1	RTN1
ILMN_1756942	Sp3 transcription factor	SP3
ILMN_1757165	limb development membrane protein 1-like	LMBR1L
ILMN_1757186	GTPase IMAP family member 1	GIMAP1
ILMN_1757347	KIAA0930	KIAA0930
ILMN_1757361	neutrophil cytosolic factor 4 40kDa	NCF4
ILMN_1757406	histone cluster 1 H1c	HIST1H1C
ILMN_1757467	H1 histone family member 0	H1F0
ILMN_1757627	zinc finger MYND-type containing 19	ZMYND1 9
ILMN_1758146	signal-regulatory protein alpha	SIRPA
ILMN_1758323	acid phosphatase prostate	ACPP
ILMN_1758418	tumor necrosis factor (ligand) superfamily member 13b	TNFSF13 B
ILMN_1758619	S-antigen; retina and pineal gland (arrestin)	SAG
ILMN_1758623	histone cluster 1 H2bd	HIST1H2B
ILMN_1758673	solute carrier family 44 (choline transporter) member 1	SLC44A1
ILMN_1758719	neural precursor cell expressed developmentally down-regulated 9	NEDD9
ILMN_1758735	NLR family pyrin domain containing 12	NLRP12
ILMN_1758778	centriolin	CNTRL
ILMN_1758895	cathepsin K	CTSK
ILMN_1758906	guanine nucleotide binding protein (G protein) alpha 13	GNA13
ILMN_1758918	bromodomain containing 2	BRD2
ILMN_1758928	Unnamed Transcript	
ILMN_1758938	solute carrier family 31 (copper transporter) member 2	SLC31A2
ILMN_1758939	receptor-interacting serine-threonine kinase 2	RIPK2
ILMN_1758963	NAD kinase	NADK
ILMN_1759008	zinc finger protein 689	ZNF689
ILMN_1759084	integrator complex subunit 8	INTS8
ILMN_1759787	thrombomodulin	THBD
ILMN_1759801	dipeptidyl-peptidase 8	DPP8
ILMN_1759818	sortilin-related receptor L(DLR class) A repeats containing	SORL1
ILMN_1759952	proteasome (prosome macropain) subunit alpha type 5	PSMA5

ILMN_1759983	down-regulator of transcription 1 TBP-binding (negative cofactor 2	DR1
ILMN_1760062	interferon-induced protein 44	IFI44
ILMN_1760189	NLR family apoptosis inhibitory protein	NAIP
ILMN_1760280	nuclear transport factor 2-like export factor 1	NXT1
ILMN_1760849	neuropilin (NRP) and tolloid (TLL)-like 2	NETO2
ILMN_1761260	cordon-bleu WH2 repeat protein-like 1	COBLL1
ILMN_1761411	minichromosome maintenance complex binding protein	MCMBP
ILMN_1761778	tumor necrosis factor (ligand) superfamily member 8	TNFSF8
ILMN_1761833	solute carrier family 40 (iron-regulated transporter) member 1	SLC40A1
ILMN_1761941	family with sequence similarity 198 member B	FAM198B
ILMN_1761945	fibroblast growth factor binding protein 2	FGFBP2
ILMN_1761968	protein phosphatase 1 regulatory (inhibitor) subunit 14A	PPP1R14A
ILMN_1762255	glutathione S-transferase mu 1	GSTM1
ILMN_1762594	nucleotide-binding oligomerization domain containing 2	NOD2
ILMN_1762674	nucleoporin 43kDa	NUP43
ILMN_1762678	N-myristoyltransferase 1	NMT1
ILMN_1762713	mast cell-expressed membrane protein 1	MCEMP1
ILMN_1762888	methyltransferase like 21A	METTL21A
ILMN_1762899	early growth response 1	EGR1
ILMN_1762972	chromodomain helicase DNA binding protein 9	CHD9
ILMN_1763080	glutaminyl-tRNA synthetase	QARS
ILMN_1763144	sialidase 1 (lysosomal sialidase)	NEU1
ILMN_1763436	senataxin	SETX
ILMN_1763627	transportin 1	TNPO1
ILMN_1763730	adaptor protein phosphotyrosine interaction PH domain and leucin	APPL1
ILMN_1763837	alanyl (membrane) aminopeptidase	ANPEP
ILMN_1764177	jumonji AT rich interactive domain 2	JARID2
ILMN_1764414	son of sevenless homolog 2 (Drosophila)	SOS2
ILMN_1764709	v-maf avian musculoaponeurotic fibrosarcoma oncogene homolog B	MAFB
ILMN_1764891	zinc finger protein 384	ZNF384
ILMN_1764945	adaptor-related protein complex 3 delta 1 subunit	AP3D1
ILMN_1765019	SAC1 suppressor of actin mutations 1-like (yeast)	SACM1L
ILMN_1765165	vacuolar protein sorting 13 homolog B (yeast)	VPS13B
ILMN_1765547	interferon regulatory factor 2	IRF2
ILMN_1765578	TCDD-inducible poly(ADP-ribose) polymerase	TIPARP
ILMN_1765801	glucosidase alpha; acid	GAA
ILMN_1765825	DDB1 and CUL4 associated factor 11	DCAF11

ILMN_1766054	ATP-binding cassette sub-family A (ABC1) member 1	ABCA1
ILMN_1766085	v-rel avian reticuloendotheliosis viral oncogene homolog	REL
ILMN_1766123	fragile histidine triad	FHIT
ILMN_1766125	lon peptidase 1 mitochondrial	LONP1
ILMN_1766165	synuclein alpha (non A4 component of amyloid precursor)	SNCA
ILMN_1766169	branched chain amino-acid transaminase 1 cytosolic	BCAT1
ILMN_1766269	histocompatibility (minor) 13	HM13
ILMN_1766275	phosphatidylinositol-4 5-bisphosphate 3-kinase catalytic subunit	PIK3CD
ILMN_1766487	leucine rich repeat containing 25	LRRC25
ILMN_1766657	stomatin	STOM
ILMN_1766718	LysM putative peptidoglycan-binding domain containing 3	LYSMD3
ILMN_1766736	bactericidal/permeability-increasing protein	BPI
ILMN_1766762	dynein light chain roadblock-type 1	DYNLRB1
ILMN_1766814	thymidine kinase 2 mitochondrial	TK2
ILMN_1767006	proteasome (prosome macropain) subunit beta type 8	PSMB8
ILMN_1767139	NADH dehydrogenase (ubiquinone) 1 alpha subcomplex 13	NDUFA13
ILMN_1767193	complement component (3b/4b) receptor 1 (Knops blood group)	CR1
ILMN_1767281	pro-platelet basic protein (chemokine (C-X-C motif) ligand 7)	PPBP
ILMN_1768020	HAUS augmin-like complex subunit 8	HAUS8
ILMN_1768357	reticulon 1	RTN1
ILMN_1768391	ADP-ribosylation factor-like 4C	ARL4C
ILMN_1768393	small nuclear ribonucleoprotein D1 polypeptide 16kDa	SNRPD1
ILMN_1768470	eukaryotic translation initiation factor 4 gamma 1	EIF4G1
ILMN_1768510	mannosidase alpha class 2B member 2	MAN2B2
ILMN_1768534	basic helix-loop-helix family member e40	BHLHE40
ILMN_1768582	protein phosphatase 2 catalytic subunit beta isozyme	PPP2CB
ILMN_1768754	Unnamed Transcript	
ILMN_1768773	Unnamed Transcript	
ILMN_1768913	translin	TSN
ILMN_1768958	RAS guanyl releasing protein 1 (calcium and DAG-regulated)	RASGRP1
ILMN_1768969	lamin B receptor	LBR
ILMN_1768973	histone cluster 2 H2ac	HIST2H2A
ILMN_1769135	dipeptidyl-peptidase 7	DPP7
ILMN_1769299	myotubularin related protein 11	MTMR11
ILMN_1769383	Unnamed Transcript	
ILMN_1769520	ubiquitin-conjugating enzyme E2L 6	UBE2L6

ILMN_1769546	Ras and Rab interactor 2	RIN2
ILMN_1769665	RAB5C member RAS oncogene family	RAB5C
ILMN_1769931	splicing factor proline/glutamine-rich	SFPQ
ILMN_1770071	interferon induced transmembrane protein 4 pseudogene	IFITM4P
ILMN_1770071	interferon induced transmembrane protein 4 pseudogene	IFITM4P
ILMN_1770085	BTG family member 2	BTG2
ILMN_1770260	nuclear factor of kappa light polypeptide gene enhancer in B-cells	NFKBIZ
ILMN_1770425	CDP-diacylglycerol--inositol 3-phosphatidyltransferase	CDIPT
ILMN_1770977	transmembrane protein 134	TMEM134
ILMN_1771333	CD47 molecule	CD47
ILMN_1771627	zinc finger MIZ-type containing 1	ZMIZ1
ILMN_1771664	C-type lectin domain family 4 member E	CLEC4E
ILMN_1771695	regulator of chromosome condensation (RCC1) and BTB (POZ) domain co	RCBTB2
ILMN_1772387	toll-like receptor 2	TLR2
ILMN_1772540	ATM interactor	ATMIN
ILMN_1772692	dicer 1 ribonuclease type III	DICER1
ILMN_1772814	nemo-like kinase	NLK
ILMN_1772876	zinc finger protein 395	ZNF395
ILMN_1772929	ATP synthase H ⁺ transporting mitochondrial Fo complex subunit F	ATP5J
ILMN_1772964	chemokine (C-C motif) ligand 8	CCL8
ILMN_1773245	chemokine (C-C motif) ligand 3-like 1	CCL3L1
ILMN_1773313	Unnamed Transcript	
ILMN_1773352	chemokine (C-C motif) ligand 5	CCL5
ILMN_1773485	QKI KH domain containing RNA binding	QKI
ILMN_1773620	SWI/SNF related matrix associated actin dependent regulator of c	SMARCC2
ILMN_1773780	family with sequence similarity 173 member A	FAM173A
ILMN_1773935	transmembrane protein 165	TMEM165
ILMN_1773963	guanine nucleotide binding protein (G protein) alpha 15 (Gq class)	GNA15
ILMN_1774077	guanylate binding protein 2 interferon-inducible	GBP2
ILMN_1774083	TP53 regulated inhibitor of apoptosis 1	TRIAP1
ILMN_1774513	Unnamed Transcript	
ILMN_1774661	small nuclear ribonucleoprotein polypeptides B and B1	SNRPB
ILMN_1774761	chemokine (C-C motif) receptor 2	CCR2
ILMN_1774874	interleukin 1 receptor antagonist	IL1RN
ILMN_1774938	aldo-keto reductase family 1 member A1 (aldehyde reductase)	AKR1A1
ILMN_1775016	myelin protein zero-like 2	MPZL2

ILMN_1775085	zinc finger protein 232	ZNF232
ILMN_1775304	DnaJ (Hsp40) homolog subfamily B member 1	DNAJB1
ILMN_1775327	pyruvate kinase muscle	PKM
ILMN_1775501	interleukin 1 beta	IL1B
ILMN_1775508	cylindromatosis (turban tumor syndrome)	CYLD
ILMN_1775780	urotensin 2	UTS2
ILMN_1776021	eukaryotic translation initiation factor 4H	EIF4H
ILMN_1776073	chaperonin containing TCP1 subunit 4 (delta)	CCT4
ILMN_1776119	ATP-binding cassette sub-family C (CFTR/MRP) member 10	ABCC10
ILMN_1776347	t-complex 1	TCP1
ILMN_1776487	transcriptional adaptor 1	TADA1
ILMN_1776649	leucine-rich repeat kinase 2	LRRK2
ILMN_1776678	GTPase IMAP family member 7	GIMAP7
ILMN_1776777	adenosine deaminase RNA-specific	ADAR
ILMN_1777058	Unnamed Transcript	
ILMN_1777139	MAK16 homolog (S. cerevisiae)	MAK16
ILMN_1777190	complement factor D (adipsin)	CFD
ILMN_1777325	signal transducer and activator of transcription 1 91kDa	STAT1
ILMN_1777461	chemokine (C-C motif) receptor 2	CCR2
ILMN_1777663	topoisomerase (DNA) II beta 180kDa	TOP2B
ILMN_1777881	tetraspanin 17	TSPAN17
ILMN_1777906	MAP7 domain containing 1	MAP7D1
ILMN_1778136	zinc finger MYND-type containing 15	ZMYND15
ILMN_1778236	protein tyrosine phosphatase non-receptor type 11	PTPN11
ILMN_1778321	solute carrier family 2 (facilitated glucose transporter) member 6	SLC2A6
ILMN_1778444	FK506 binding protein 5	FKBP5
ILMN_1778464	trafficking protein particle complex 10	TRAPPC10
ILMN_1778488	WD repeat domain 41	WDR41
ILMN_1778756	sulfotransferase family cytosolic 1A phenol-preferring member	SULT1A1
ILMN_1778764	BUB3 mitotic checkpoint protein	BUB3
ILMN_1779015	zinc finger protein 467	ZNF467
ILMN_1779252	tripartite motif containing 22	TRIM22
ILMN_1779264	proteasome (prosome macropain) assembly chaperone 1	PSMG1
ILMN_1779410	VAMP (vesicle-associated membrane protein)-associated protein A 33	VAPA
ILMN_1779677	zinc finger CCHC domain containing 6	ZCCHC6
ILMN_1779751	chromosome 7 open reading frame 55	C7orf55

ILMN_1779857	Kruppel-like factor 4 (gut)	KLF4
ILMN_1780132	pellino E3 ubiquitin protein ligase family member 2	PELI2
ILMN_1780302	dynein cytoplasmic 1 heavy chain 1	DYNC1H1
ILMN_1780334	potassium inwardly-rectifying channel subfamily J member 2	KCNJ2
ILMN_1780368	G protein-coupled receptor 18	GPR18
ILMN_1780546	oncostatin M	OSM
ILMN_1780582	CD83 molecule	CD83
ILMN_1780601	egf-like module containing mucin-like hormone receptor-like 1	EMR1
ILMN_1780659	nudix (nucleoside diphosphate linked moiety X)-type motif 6	NUDT6
ILMN_1780913	protein phosphatase 2 regulatory subunit B' gamma	PPP2R5C
ILMN_1781373	interferon induced with helicase C domain 1	IFIH1
ILMN_1781416	frequently rearranged in advanced T-cell lymphomas 1	FRAT1
ILMN_1782015	Fc receptor-like B	FCRLB
ILMN_1782069	trafficking protein kinesin binding 1	TRAK1
ILMN_1782085	diphthamine biosynthesis 6	DPH6
ILMN_1782305	nuclear receptor subfamily 4 group A member 2	NR4A2
ILMN_1782331	Unnamed Transcript	
ILMN_1782352	VENT homeobox	VENTX
ILMN_1782419	guanine nucleotide binding protein (G protein) gamma 11	GNG11
ILMN_1782488	ribonuclease H2 subunit B	RNASEH2B
ILMN_1782551	E2F transcription factor 5 p130-binding	E2F5
ILMN_1782633	Unnamed Transcript	
ILMN_1782704	CD19 molecule	CD19
ILMN_1782729	C-type lectin-like 1	CLECL1
ILMN_1782741	CD300 molecule-like family member b	CD300LB
ILMN_1783627	calpastatin	CAST
ILMN_1783675	ankyrin repeat and SOCS box containing 8	ASB8
ILMN_1783695	glutamyl-prolyl-tRNA synthetase	EPRS
ILMN_1783852	CD164 molecule sialomucin	CD164
ILMN_1783956	ATPase class I type 8B member 4	ATP8B4
ILMN_1784203	acrosin binding protein	ACRBP
ILMN_1784218	DEAD (Asp-Glu-Ala-Asp) box polypeptide 23	DDX23
ILMN_1784287	transforming growth factor beta receptor III	TGFBR3
ILMN_1784352	cerebral cavernous malformation 2	CCM2
ILMN_1784602	cyclin-dependent kinase inhibitor 1A (p21 Cip1)	CDKN1A
ILMN_1784651	N-acetylgalactosaminidase alpha-	NAGA
ILMN_1784709	glucosamine-6-phosphate deaminase 1	GNPDA1
ILMN_1784737	sphingosine-1-phosphate receptor 4	S1PR4

ILMN_1784766	minichromosome maintenance complex component 3 associated protein	MCM3AP
ILMN_1784863	CD36 molecule (thrombospondin receptor)	CD36
ILMN_1784871	fatty acid synthase	FASN
ILMN_1785005	neutrophil cytosolic factor 4 40kDa	NCF4
ILMN_1785113	methylmalonyl CoA mutase	MUT
ILMN_1785141	microtubule associated monooxygenase calponin and LIM domain conta	MICAL2
ILMN_1785175	SWAP switching B-cell complex 70kDa subunit	SWAP70
ILMN_1785345	G protein-coupled receptor 84	GPR84
ILMN_1785439	CD79b molecule immunoglobulin-associated beta	CD79B
ILMN_1785660	signal recognition particle receptor (docking protein)	SRPR
ILMN_1785661	abhydrolase domain containing 17B	ABHD17B
ILMN_1785703	leiomodin 3 (fetal)	LMOD3
ILMN_1785711	Unnamed Transcript	
ILMN_1785732	tumor necrosis factor alpha-induced protein 6	TNFAIP6
ILMN_1785852	nucleic acid binding protein 1	NABP1
ILMN_1786015	CCCTC-binding factor (zinc finger protein)	CTCF
ILMN_1786065	ubiquitin-like with PHD and ring finger domains 1	UHRF1
ILMN_1786275	poly(A) polymerase gamma	PAPOLG
ILMN_1786347	transportin 1	TNPO1
ILMN_1786658	bolA family member 3	BOLA3
ILMN_1786722	zinc finger protein 385A	ZNF385A
ILMN_1786823	intercellular adhesion molecule 2	ICAM2
ILMN_1787064	ankyrin repeat domain 17	ANKRD17
ILMN_1787127	solute carrier family 43 (amino acid system L transporter) member	SLC43A2
ILMN_1787212	cyclin-dependent kinase inhibitor 1A (p21 Cip1)	CDKN1A
ILMN_1787410	eukaryotic translation initiation factor 6	EIF6
ILMN_1787461	runt-related transcription factor 3	RUNX3
ILMN_1787509	helicase with zinc finger 2 transcriptional coactivator	HELZ2
ILMN_1787529	complement component 3a receptor 1	C3AR1
ILMN_1787705	ATPase H ⁺ transporting lysosomal 56/58kDa V1 subunit B2	ATP6V1B2
ILMN_1787813	solute carrier family 5 (sodium/myo-inositol cotransporter) member	SLC5A3
ILMN_1787879	ADP-ribosylation factor-like 2	ARL2
ILMN_1787897	chemokine (C-X-C motif) ligand 1 (melanoma growth stimulating activ	CXCL1
ILMN_1788002	mitogen-activated protein kinase 14	MAPK14
ILMN_1788213	frequently rearranged in advanced T-cell lymphomas 2	FRAT2
ILMN_1788416	abhydrolase domain containing 17C	ABHD17C

ILMN_1788531	signaling threshold regulating transmembrane adaptor 1	SIT1
ILMN_1788547	glutamate-cysteine ligase modifier subunit	GCLM
ILMN_1788689	pleckstrin homology domain interacting protein	PHIP
ILMN_1788841	T-cell leukemia/lymphoma 1A	TCL1A
ILMN_1789136	Unnamed Transcript	
ILMN_1789244	SRY (sex determining region Y)-box 8	SOX8
ILMN_1789283	protein phosphatase 2 regulatory subunit B' gamma	PPP2R5C
ILMN_1789342	NADH dehydrogenase (ubiquinone) Fe-S protein 2 49kDa (NADH-coenzym	NDUFS2
ILMN_1789436	DENN/MADD domain containing 1B	DENND1 B
ILMN_1789596	ets variant 6	ETV6
ILMN_1789627	Unnamed Transcript	
ILMN_1789781	Pim-3 proto-oncogene serine/threonine kinase	PIM3
ILMN_1789955	proline-rich nuclear receptor coactivator 1	PNRC1
ILMN_1789990	ADP-ribosylation factor guanine nucleotide-exchange factor 2 (brefe	ARFGEF2
ILMN_1790228	furin (paired basic amino acid cleaving enzyme)	FURIN
ILMN_1790230	zinc finger protein 181	ZNF181
ILMN_1790555	coiled-coil domain containing 146	CCDC146
ILMN_1790689	cysteine-rich secretory protein LCCL domain containing 2	CRISPLD2
ILMN_1790692	granulysin	GNLY
ILMN_1790741	ring finger protein 126	RNF126
ILMN_1790757	adenylosuccinate lyase	ADSL
ILMN_1790782	mediator complex subunit 16	MED16
ILMN_1790909	nuclear factor erythroid 2-like 2	NFE2L2
ILMN_1791329	Fc receptor-like 2	FCRL2
ILMN_1791396	Unnamed Transcript	
ILMN_1791483	phosphodiesterase 4D cAMP-specific	PDE4D
ILMN_1791511	transmembrane protein 176A	TMEM176 A
ILMN_1791759	chemokine (C-X-C motif) ligand 10	CXCL10
ILMN_1791770	SWI/SNF related matrix associated actin dependent regulator of c	SMARCC 2
ILMN_1792072	fucosyltransferase 4 (alpha (1 3) fucosyltransferase myeloid-spec	FUT4
ILMN_1792518	syntaxin 7	STX7
ILMN_1792922	superoxide dismutase 2 mitochondrial	SOD2
ILMN_1793474	insulin induced gene 1	INSIG1
ILMN_1793563	dynactin 1	DCTN1
ILMN_1793743	disrupted in renal carcinoma 2	DIRC2
ILMN_1793859	aldehyde dehydrogenase 2 family (mitochondrial)	ALDH2

ILMN_1793990	inhibitor of DNA binding 2 dominant negative helix-loop-helix prot	ID2
ILMN_1794038	family with sequence similarity 49 member A	FAM49A
ILMN_1794165	phosphogluconate dehydrogenase	PGD
ILMN_1794740	CD151 molecule (Raph blood group)	CD151
ILMN_1794781	vav 2 guanine nucleotide exchange factor	VAV2
ILMN_1794782	ATP-binding cassette sub-family G (WHITE) member 1	ABCG1
ILMN_1794875	1-acylglycerol-3-phosphate O-acyltransferase 9	AGPAT9
ILMN_1795715	dihydropyrimidine dehydrogenase	DPYD
ILMN_1795719	replication protein A1 70kDa	RPA1
ILMN_1795846	protein phosphatase 2 regulatory subunit B' gamma	PPP2R5C
ILMN_1796075	vacuolar protein sorting 41 homolog (S. cerevisiae)	VPS41
ILMN_1796094	CD36 molecule (thrombospondin receptor)	CD36
ILMN_1796210	peroxisome proliferator-activated receptor gamma coactivator-relat	PPRC1
ILMN_1796316	matrix metalloproteinase 9 (gelatinase B 92kDa gelatinase 92kDa t	MMP9
ILMN_1796409	complement component 1 q subcomponent B chain	C1QB
ILMN_1796642	neutrophil cytosolic factor 2	NCF2
ILMN_1796669	presenilin 1	PSEN1
ILMN_1796813	embryonic ectoderm development	EED
ILMN_1796962	protein phosphatase 3 regulatory subunit B alpha	PPP3R1
ILMN_1797001	DEAD (Asp-Glu-Ala-Asp) box polypeptide 58	DDX58
ILMN_1797310	ATPase H ⁺ transporting lysosomal 34kDa V1 subunit D	ATP6V1D
ILMN_1797341	AT rich interactive domain 1A (SWI-like)	ARID1A
ILMN_1797656	activating signal cointegrator 1 complex subunit 3	ASCC3
ILMN_1797731	membrane-spanning 4-domains subfamily A member 6A	MS4A6A
ILMN_1797822	sel-1 suppressor of lin-12-like 3 (C. elegans)	SEL1L3
ILMN_1797895	free fatty acid receptor 2	FFAR2
ILMN_1798085	EP300 interacting inhibitor of differentiation 2B	EID2B
ILMN_1798177	churchill domain containing 1	CHURC1
ILMN_1798181	interferon regulatory factor 7	IRF7
ILMN_1798233	proteasome (prosome macropain) subunit beta type 9	PSMB9
ILMN_1798270	single-pass membrane protein with coiled-coil domains 4	SMCO4
ILMN_1798360	atypical chemokine receptor 3	ACKR3
ILMN_1798706	G protein-coupled receptor 183	GPR183
ILMN_1799030	CKLF-like MARVEL transmembrane domain containing 2	CMTM2
ILMN_1799062	nuclear factor of kappa light polypeptide gene enhancer in B-cells	NFKB2
ILMN_1799103	small nuclear ribonucleoprotein polypeptides B and B1	SNRPB
ILMN_1799467	sterile alpha motif domain containing 9-like	SAMD9L

ILMN_1799601	UHRF1 binding protein 1-like	UHRF1BP1
ILMN_1799725	dedicator of cytokinesis 2	DOCK2
ILMN_1799765	Unnamed Transcript	
ILMN_1799856	Unnamed Transcript	
ILMN_1800451	mediator complex subunit 16	MED16
ILMN_1800540	CD55 molecule decay accelerating factor for complement (Cromer blo	CD55
ILMN_1800602	grancalcin EF-hand calcium binding protein	GCA
ILMN_1800912	zinc finger protein 106	ZNF106
ILMN_1800951	ataxin 1	ATXN1
ILMN_1801077	perilipin 2	PLIN2
ILMN_1801098	Unnamed Transcript	
ILMN_1801156	rearranged L-myc fusion	RLF
ILMN_1801205	glycoprotein (transmembrane) nmb	GNPMB
ILMN_1801216	S100 calcium binding protein P	S100P
ILMN_1801246	interferon induced transmembrane protein 1	IFITM1
ILMN_1801246	interferon induced transmembrane protein 1	IFITM1
ILMN_1801307	tumor necrosis factor (ligand) superfamily member 10	TNFSF10
ILMN_1801504	runt-related transcription factor 1	RUNX1
ILMN_1801616	epithelial membrane protein 1	EMP1
ILMN_1801710	amyloid beta (A4) precursor protein-binding family B member 1 in	APBB1IP
ILMN_1802257	phosphatidylcholine transfer protein	PCTP
ILMN_1802404	ATP-binding cassette sub-family C (CFTR/MRP) member 1	ABCC1
ILMN_1802457	MYC associated factor X	MAX
ILMN_1802831	myotubularin related protein 12	MTMR12
ILMN_1803476	potassium channel tetramerization domain containing 20	KCTD20
ILMN_1803615	megakaryocyte-associated tyrosine kinase	MATK
ILMN_1803788	lectin galactoside-binding soluble 3	LGALS3
ILMN_1803819	IQ motif containing GTPase activating protein 1	IQGAP1
ILMN_1803853	nucleolar protein 7 27kDa	NOL7
ILMN_1803882	vascular endothelial growth factor A	VEGFA
ILMN_1803925	myotubularin related protein 3	MTMR3
ILMN_1803945	HLA complex P5 (non-protein coding)	HCP5
ILMN_1803977	polypeptide N-acetylgalactosaminyltransferase 1	GALNT1
ILMN_1803988	myeloid cell leukemia 1	MCL1
ILMN_1803997	serologically defined colon cancer antigen 3	SDCCAG3
ILMN_1804174	Fc fragment of IgG low affinity IIb receptor (CD32)	FCGR2B
ILMN_1804448	musashi RNA-binding protein 2	MSI2
ILMN_1804854	catenin (cadherin-associated protein) alpha 1 102kDa	CTNNA1

ILMN_1805104	4-aminobutyrate aminotransferase	ABAT
ILMN_1805175	transforming growth factor alpha	TGFA
ILMN_1805228	leucine-rich alpha-2-glycoprotein 1	LRG1
ILMN_1805410	chromosome 15 open reading frame 48	C15orf48
ILMN_1806023	jun proto-oncogene	JUN
ILMN_1806184	mediator complex subunit 13	MED13
ILMN_1806408	acyl-CoA dehydrogenase very long chain	ACADVL
ILMN_1806906	signal sequence receptor gamma (translocon-associated protein gamma)	SSR3
ILMN_1806908	protein kinase C beta	PRKCB
ILMN_1806999	B9 protein domain 2	B9D2
ILMN_1807042	myristoylated alanine-rich protein kinase C substrate	MARCKS
ILMN_1807074	macrophage migration inhibitory factor (glycosylation-inhibiting fa)	MIF
ILMN_1807277	interferon gamma-inducible protein 30	IFI30
ILMN_1807277	interferon gamma-inducible protein 30	IFI30
ILMN_1807300	polycystic kidney disease 2 (autosomal dominant)	PKD2
ILMN_1807372	adenosine A2a receptor	ADORA2A
ILMN_1807596	ubiquitin associated protein 1	UBAP1
ILMN_1807662	insulin-like growth factor 2 receptor	IGF2R
ILMN_1807833	histocompatibility (minor) 13	HM13
ILMN_1808132	Fas cell surface death receptor	FAS
ILMN_1808405	major histocompatibility complex class II DQ alpha 1	HLA-DQA1
ILMN_1808548	Unnamed Transcript	
ILMN_1808587	zinc finger homeobox 3	ZFHX3
ILMN_1808707	fascin actin-bundling protein 1	FSCN1
ILMN_1808768	Rho-associated coiled-coil containing protein kinase 1	ROCK1
ILMN_1809027	ATP5S-like	ATP5SL
ILMN_1809040	low density lipoprotein receptor adaptor protein 1	LDLRAP1
ILMN_1809086	5'-3' exoribonuclease 1	XRN1
ILMN_1809139	AT hook containing transcription factor 1	AHCTF1
ILMN_1809147	family with sequence similarity 118 member A	FAM118A
ILMN_1809193	presenilin 1	PSEN1
ILMN_1809467	vesicle-associated membrane protein 5	VAMP5
ILMN_1809522	NACC family member 2 BEN and BTB (POZ) domain containing	NACC2
ILMN_1809582	vacuolar protein sorting 13 homolog C (S. cerevisiae)	VPS13C
ILMN_1809607	peptidylprolyl isomerase F	PPIF
ILMN_1809695	calcium/calmodulin-dependent protein kinase II gamma	CAMK2G
ILMN_1809866	WD repeat domain 74	WDR74

ILMN_1809931	N-myc downstream regulated 1	NDRG1
ILMN_1810058	RAB member of RAS oncogene family-like 2B	RABL2B
ILMN_1810214	jun D proto-oncogene	JUND
ILMN_1810254	meiosis inhibitor 1	MEI1
ILMN_1810274	homeobox B2	HOXB2
ILMN_1810289	myoferlin	MYOF
ILMN_1810385	ATR serine/threonine kinase	ATR
ILMN_1810418	lamin B receptor	LBR
ILMN_1810486	RAB34 member RAS oncogene family	RAB34
ILMN_1810680	Unnamed Transcript	
ILMN_1810838	metadherin	MTDH
ILMN_1810941	catechol-O-methyltransferase	COMT
ILMN_1811171	G protein-coupled receptor 132	GPR132
ILMN_1811258	v-rel avian reticuloendotheliosis viral oncogene homolog B	RELB
ILMN_1811328	dipeptidyl-peptidase 7	DPP7
ILMN_1811702	granulin	GRN
ILMN_1811933	serine hydroxymethyltransferase 1 (soluble)	SHMT1
ILMN_1811954	neuregulin 1	NRG1
ILMN_1811972	MYC binding protein 2 E3 ubiquitin protein ligase	MYCBP2
ILMN_1812139	Ras association (RalGDS/AF-6) domain family member 2	RASSF2
ILMN_1812191	chromosome 12 open reading frame 57	C12orf57
ILMN_1812384	nuclear receptor subfamily 3 group C member 1 (glucocorticoid re	NR3C1
ILMN_1812433	haptoglobin	HP
ILMN_1812995	cathepsin L	CTSL
ILMN_1813314	histone cluster 1 H2bk	HIST1H2B
ILMN_1813379	tumor necrosis factor receptor superfamily member 9	TNFRSF9
ILMN_1813399	ATPase Ca ⁺⁺ transporting plasma membrane 1	ATP2B1
ILMN_1813604	NADH dehydrogenase (ubiquinone) 1 beta subcomplex 7 18kDa	NDUFB7
ILMN_1813775	cyclin G associated kinase	GAK
ILMN_1813795	Ras association (RalGDS/AF-6) domain family member 5	RASSF5
ILMN_1813817	mitochondrial ribosomal protein L55	MRPL55
ILMN_1814305	sterile alpha motif domain containing 9	SAMD9
ILMN_1814650	trafficking protein particle complex 4	TRAPPC4
ILMN_1815023	Pim-1 proto-oncogene serine/threonine kinase	PIM1
ILMN_1815086	ninjurin 1	NINJ1
ILMN_1815148	mannosidase alpha class 2A member 2	MAN2A2
ILMN_1815283	Unnamed Transcript	
ILMN_1815527	hemoglobin delta	HBD
ILMN_1815656	serine incorporator 3	SERINC3
ILMN_1815733	eukaryotic translation initiation factor 5	EIF5

ILMN_1815758	GRB2-associated binding protein 2	GAB2
ILMN_1815890	interleukin 12 receptor beta 1	IL12RB1
ILMN_1837935	transportin 1	TNPO1
ILMN_2038775	tubulin beta 2A class IIa	TUBB2A
ILMN_2041190	coagulation factor II (thrombin) receptor-like 1	F2RL1
ILMN_2041327	mitochondrial ribosomal protein L37	MRPL37
ILMN_2044226	protein phosphatase 3 catalytic subunit alpha isozyme	PPP3CA
ILMN_2046024	dual specificity phosphatase 11 (RNA/RNP complex 1-interacting)	DUSP11
ILMN_2048607	ankyrin repeat domain 9	ANKRD9
ILMN_2048822	NudC domain containing 2	NUDCD2
ILMN_2049364	methyltransferase like 21A	METTTL21A
ILMN_2050911	solute carrier family 22 (organic cation/zwitterion transporter) m	SLC22A4
ILMN_2051900	EP300 interacting inhibitor of differentiation 2B	EID2B
ILMN_2052891	polycystic kidney disease 2 (autosomal dominant)	PKD2
ILMN_2053103	solute carrier family 40 (iron-regulated transporter) member 1	SLC40A1
ILMN_2053415	low density lipoprotein receptor	LDLR
ILMN_2054019	ISG15 ubiquitin-like modifier	ISG15
ILMN_2054442	zinc finger protein 146	ZNF146
ILMN_2055781	killer cell lectin-like receptor subfamily F member 1	KLRF1
ILMN_2056167	oligosaccharyltransferase complex subunit (non-catalytic)	OSTC
ILMN_2058782	interferon alpha-inducible protein 27	IFI27
ILMN_2059452	solute carrier family 12 (sodium/potassium/chloride transporter) m	SLC12A2
ILMN_2059886	tetratricopeptide repeat domain 38	TTC38
ILMN_2060115	sortilin-related receptor L(DLR class) A repeats containing	SORL1
ILMN_2062001	hook microtubule-tethering protein 3	HOOK3
ILMN_2062370	nucleolar protein 8	NOL8
ILMN_2063586	chloride intracellular channel 4	CLIC4
ILMN_2064898	cytochrome c oxidase assembly factor 3	COA3
ILMN_2067607	transmembrane protein 106B	TMEM106B
ILMN_2067708	transcription factor B2 mitochondrial	TFB2M
ILMN_2067709	transcription factor B2 mitochondrial	TFB2M
ILMN_2072178	enoyl CoA hydratase domain containing 3	ECHDC3
ILMN_2073157	amylase alpha 2B (pancreatic)	AMY2B
ILMN_2074401	LysM putative peptidoglycan-binding domain containing 3	LYSMD3
ILMN_2080637	zinc finger and BTB domain containing 44	ZBTB44
ILMN_2081988	LanC lantibiotic synthetase component C-like 1 (bacterial)	LANCL1

ILMN_2082762	small nucleolar RNA C/D box 68	SNORD68
ILMN_2082810	bromodomain containing 7	BRD7
ILMN_2083469	insulin receptor substrate 2	IRS2
ILMN_2083833	CCR4-NOT transcription complex subunit 6-like	CNOT6L
ILMN_2083946	transforming growth factor alpha	TGFA
ILMN_2084353	mannose-6-phosphate receptor (cation dependent)	M6PR
ILMN_2085012	transmembrane protein 176B	TMEM176 B
ILMN_2085760	actin related protein 2/3 complex subunit 1B 41kDa	ARPC1B
ILMN_2085862	solute carrier family 15 (oligopeptide transporter) member 3	SLC15A3
ILMN_2086077	jun B proto-oncogene	JUNB
ILMN_2086095	inhibitor of DNA binding 2 dominant negative helix-loop-helix prot	ID2
ILMN_2087303	XK Kell blood group complex subunit-related family member 8	XKR8
ILMN_2087575	zinc finger CCCH-type containing 4	ZC3H4
ILMN_2087646	H2.0-like homeobox	HLX
ILMN_2088437	chemokine (C-X3-C motif) receptor 1	CX3CR1
ILMN_2088612	exportin 4	XPO4
ILMN_2090105	transgelin 2	TAGLN2
ILMN_2090607	interferon regulatory factor 2	IRF2
ILMN_2092118	formyl peptide receptor 1	FPR1
ILMN_2094061	inositol(myo)-1(or 4)-monophosphatase 2	IMPA2
ILMN_2095840	K(lysine) acetyltransferase 6A	KAT6A
ILMN_2098126	chemokine (C-C motif) ligand 5	CCL5
ILMN_2100209	Unnamed Transcript	
ILMN_2100437	hemoglobin beta	HBB
ILMN_2101832	lysosomal protein transmembrane 4 beta	LAPTM4B
ILMN_2101920	heterogeneous nuclear ribonucleoprotein H1 (H)	HNRNPH1
ILMN_2105441	immunoglobulin J polypeptide linker protein for immunoglobulin alp	IGJ
ILMN_2106656	basic leucine zipper nuclear factor 1	BLZF1
ILMN_2107004	G protein-coupled receptor 1	GPR1
ILMN_2109197	erythrocyte membrane protein band 4.1-like 3	EPB41L3
ILMN_2109416	napsin B aspartic peptidase pseudogene	NAPSB
ILMN_2111739	mannosidase alpha class 2C member 1	MAN2C1
ILMN_2112049	DNL-type zinc finger	DNLZ
ILMN_2113126	ribonuclease RNase A family 3	RNASE3
ILMN_2113362	ADP-ribosylation factor-like 6 interacting protein 1	ARL6IP1
ILMN_2113738	minichromosome maintenance domain containing 2	MCMDC2
ILMN_2114422	nucleotide-binding oligomerization domain containing 1	NOD1

ILMN_2115218	ankyrin repeat domain 10	ANKRD10
ILMN_2115591	kelch domain containing 10	KLHDC10
ILMN_2115669	sema domain immunoglobulin domain (Ig) transmembrane domain (TM)	SEMA4C
ILMN_2116556	LSM5 homolog U6 small nuclear RNA associated (S. cerevisiae)	LSM5
ILMN_2117240	deoxycytidine kinase	DCK
ILMN_2117623	plexin C1	PLXNC1
ILMN_2118864	RAB1A member RAS oncogene family	RAB1A
ILMN_2119486	cyclin D binding myb-like transcription factor 1	DMTF1
ILMN_2120022	ADP-ribosylation factor-like 5B	ARL5B
ILMN_2121816	G protein-coupled receptor 137B	GPR137B
ILMN_2122511	collagen and calcium binding EGF domains 1	CCBE1
ILMN_2123312	endoplasmic reticulum aminopeptidase 2	ERAP2
ILMN_2123402	canopy FGF signaling regulator 2	CNPY2
ILMN_2123557	family with sequence similarity 73 member A	FAM73A
ILMN_2124064	v-rel avian reticuloendotheliosis viral oncogene homolog	REL
ILMN_2124155	ATPase class VI type 11B	ATP11B
ILMN_2126832	SEC24 family member A	SEC24A
ILMN_2127605	low density lipoprotein receptor-related protein 3	LRP3
ILMN_2129015	AF4/FMR2 family member 1	AFF1
ILMN_2129927	exostosin glycosyltransferase 1	EXT1
ILMN_2130078	CDKN2A interacting protein N-terminal like	CDKN2AIP
ILMN_2132599	ankyrin repeat domain 22	ANKRD22
ILMN_2133316	GTPase IMAP family member 7	GIMAP7
ILMN_2137464	dishevelled segment polarity protein 3	DVL3
ILMN_2137536	zinc finger ZZ-type containing 3	ZZZ3
ILMN_2137789	Kruppel-like factor 4 (gut)	KLF4
ILMN_2138435	mitochondrial ribosomal protein S27	MRPS27
ILMN_2138765	perilipin 2	PLIN2
ILMN_2139351	zinc finger protein 232	ZNF232
ILMN_2140207	Unnamed Transcript	
ILMN_2140990	calcium/calmodulin-dependent protein kinase I	CAMK1
ILMN_2141941	torsin A interacting protein 1	TOR1AIP1
ILMN_2144426	Unnamed Transcript	
ILMN_2145997	Sp4 transcription factor	SP4
ILMN_2147435	mannosidase alpha class 2A member 1	MAN2A1
ILMN_2148668	regulator of chromosome condensation (RCC1) and BTB (POZ) domain co	RCBTB2
ILMN_2148785	guanylate binding protein 1 interferon-inducible	GBP1
ILMN_2150258	ZFP36 ring finger protein-like 2	ZFP36L2

ILMN_2150294	FK506 binding protein 14 22 kDa	FKBP14
ILMN_2150851	serpin peptidase inhibitor clade B (ovalbumin) member 2	SERPINB2
ILMN_2150856	serpin peptidase inhibitor clade B (ovalbumin) member 2	SERPINB2
ILMN_2151277	lysophosphatidylglycerol acyltransferase 1	LPGAT1
ILMN_2151281	GABA(A) receptor-associated protein like 1	GABARA PL
ILMN_2151541	DnaJ (Hsp40) homolog subfamily C member 10	DNAJC10
ILMN_2153332	ataxin 1	ATXN1
ILMN_2154322	sema domain immunoglobulin domain (Ig) short basic domain secre	SEMA3E
ILMN_2154654	protein tyrosine phosphatase type IVA member 1	PTP4A1
ILMN_2154836	BTG family member 3	BTG3
ILMN_2156172	hexokinase 2	HK2
ILMN_2157957	general transcription factor IIIH polypeptide 1 62kDa	GTF2H1
ILMN_2158336	SH3-domain GRB2-like endophilin B2	SH3GLB2
ILMN_2159453	syntaxin binding protein 2	STXBP2
ILMN_2159694	major histocompatibility complex class II DR beta 4	HLA- DRB4
ILMN_2159694	major histocompatibility complex class II DR beta 4	HLA- DRB4
ILMN_2162972	lysozyme	LYZ
ILMN_2163819	kinesin family member 21B	KIF21B
ILMN_2165753	major histocompatibility complex class I A	HLA-A
ILMN_2168217	G protein-coupled receptor 183	GPR183
ILMN_2169439	integrin alpha V	ITGAV
ILMN_2169761	copine VIII	CPNE8
ILMN_2169983	ATPase family AAA domain containing 1	ATAD1
ILMN_2171289	SAM domain SH3 domain and nuclear localization signals 1	SAMSN1
ILMN_2172174	purine nucleoside phosphorylase	PNP
ILMN_2173004	RAB8B member RAS oncogene family	RAB8B
ILMN_2173451	glucose-6-phosphate isomerase	GPI
ILMN_2173909	zinc finger protein 14	ZNF14
ILMN_2174612	CCR4-NOT transcription complex subunit 8	CNOT8
ILMN_2176882	zinc finger protein 69	ZNF69
ILMN_2179018	NADH dehydrogenase (ubiquinone) 1 alpha/beta subcomplex 1 8kDa	NDUFAB1
ILMN_2180606	N(alpha)-acetyltransferase 50 NatE catalytic subunit	NAA50
ILMN_2182348	structural maintenance of chromosomes 3	SMC3
ILMN_2183510	mesencephalic astrocyte-derived neurotrophic factor	MANF
ILMN_2184262	2'-5'-oligoadenylate synthetase 3 100kDa	OAS3
ILMN_2184373	chemokine (C-X-C motif) ligand 8	CXCL8

ILMN_2185984	SAM and SH3 domain containing 1	SASH1
ILMN_2186061	6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase 3	PFKFB3
ILMN_2186108	DiGeorge syndrome critical region gene 6	DGCR6
ILMN_2186216	Unnamed Transcript	
ILMN_2189842	small nucleolar RNA H/ACA box 10	SNORA10
ILMN_2194649	transcriptional adaptor 1	TADA1
ILMN_2196078	SLAM family member 6	SLAMF6
ILMN_2196347	cyclin-dependent kinase inhibitor 1B (p27 Kip1)	CDKN1B
ILMN_2196479	5'-3' exoribonuclease 2	XRN2
ILMN_2196550	KIAA0226-like	KIAA0226
ILMN_2196588	Unnamed Transcript	
ILMN_2197101	DEAD (Asp-Glu-Ala-Asp) box polypeptide 19A	DDX19A
ILMN_2197846	hydroxyacyl-CoA dehydrogenase/3-ketoacyl-CoA thiolase/enoyl-CoA hyd	HADHB
ILMN_2198499	solute carrier family 25 member 51	SLC25A51
ILMN_2199439	carbonic anhydrase II	CA2
ILMN_2204297	adaptor-related protein complex 5 mu 1 subunit	AP5M1
ILMN_2204754	thioredoxin-related transmembrane protein 4	TMX4
ILMN_2204876	feline leukemia virus subgroup C cellular receptor family member 2	FLVCR2
ILMN_2204909	X-ray repair complementing defective repair in Chinese hamster cell	XRCC2
ILMN_2205350	NADH dehydrogenase (ubiquinone) complex I assembly factor 4	NDUFAF4
ILMN_2210298	coiled-coil domain containing 61	CCDC61
ILMN_2210601	ribonuclease L (2' 5'-oligoadenylate synthetase-dependent)	RNASEL
ILMN_2210934	nuclear receptor subfamily 3 group C member 2	NR3C2
ILMN_2211189	inhibitor of kappa light polypeptide gene enhancer in B-cells kina	IKBKAP
ILMN_2211672	translin-associated factor X	TSNAX
ILMN_2211780	solute carrier family 25 (mitochondrial carrier; adenine nucleotide)	SLC25A4
ILMN_2212763	intercellular adhesion molecule 3	ICAM3
ILMN_2214603	protein phosphatase 2 regulatory subunit B" gamma	PPP2R3C
ILMN_2214678	MAX dimerization protein 1	MXD1
ILMN_2215211	zinc finger protein 514	ZNF514
ILMN_2216157	guanine nucleotide binding protein (G protein) alpha 12	GNA12
ILMN_2218780	pyruvate dehydrogenase phosphatase catalytic subunit 1	PDP1
ILMN_2218856	Unnamed Transcript	
ILMN_2221408	ADP-ribosylation factor guanine nucleotide-exchange factor 1 (brefe	ARFGEF1
ILMN_2222074	protein tyrosine phosphatase non-receptor type 12	PTPN12

ILMN_2223130	SWI/SNF related matrix associated actin dependent regulator of c	SMARCA5
ILMN_2223720	ATM interactor	ATMIN
ILMN_2226015	leucine-rich repeat kinase 2	LRRK2
ILMN_2229940	protease-associated domain containing 1	PRADC1
ILMN_2229950	M-phase phosphoprotein 10 (U3 small nucleolar ribonucleoprotein)	MPHOSP1
ILMN_2230592	mitochondrial ribosomal protein L3	MRPL3
ILMN_2231242	high mobility group box 1	HMGB1
ILMN_2231928	MX dynamin-like GTPase 2	MX2
ILMN_2233783	CD38 molecule	CD38
ILMN_2235785	potassium voltage-gated channel subfamily H (eag-related) member	KCNH6
ILMN_2236625	urotensin 2	UTS2
ILMN_2236655	histocompatibility (minor) 13	HM13
ILMN_2236800	SON DNA binding protein	SON
ILMN_2241953	paired immunoglobulin-like type 2 receptor alpha	PILRA
ILMN_2245676	basic transcription factor 3	BTF3
ILMN_2247664	SON DNA binding protein	SON
ILMN_2252309	dipeptidyl-peptidase 7	DPP7
ILMN_2252705	vacuolar protein sorting 13 homolog B (yeast)	VPS13B
ILMN_2253286	protein kinase C zeta	PRKCZ
ILMN_2253300	transmembrane 9 superfamily member 1	TM9SF1
ILMN_2256050	serpin peptidase inhibitor clade A (alpha-1 antiproteinase antit)	SERPINA1
ILMN_2258234	kelch-like family member 5	KLHL5
ILMN_2258774	mitochondrial ribosomal protein L43	MRPL43
ILMN_2259292	Unnamed Transcript	
ILMN_2259495	BH3 interacting domain death agonist	BID
ILMN_2260082	NLR family apoptosis inhibitory protein	NAIP
ILMN_2261076	neural precursor cell expressed developmentally down-regulated 9	NEDD9
ILMN_2261416	CD3d molecule delta (CD3-TCR complex)	CD3D
ILMN_2263466	acyl-CoA dehydrogenase very long chain	ACADVL
ILMN_2264625	Nipped-B homolog (Drosophila)	NIPBL
ILMN_2266595	leukocyte immunoglobulin-like receptor subfamily A (with TM domain)	LILRA5
ILMN_2267135	ST3 beta-galactoside alpha-2 3-sialyltransferase 1	ST3GAL1
ILMN_2267914	CD68 molecule	CD68
ILMN_2268409	vacuolar protein sorting 13 homolog B (yeast)	VPS13B
ILMN_2271379	serologically defined colon cancer antigen 3	SDCCAG3
ILMN_2272074	TROVE domain family member 2	TROVE2

ILMN_2272967	trafficking protein particle complex 6B	TRAPPC6 B
ILMN_2273261	F-box protein 3	FBXO3
ILMN_2276996	chemokine (C-C motif) receptor 2	CCR2
ILMN_2277099	tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation prot	YWHAB
ILMN_2278561	Unnamed Transcript	
ILMN_2278729	eukaryotic translation initiation factor 3 subunit B	EIF3B
ILMN_2278850	RAB24 member RAS oncogene family	RAB24
ILMN_2280911	vacuolar protein sorting 41 homolog (S. cerevisiae)	VPS41
ILMN_2281502	dehydrogenase/reductase (SDR family) member 9	DHRS9
ILMN_2282641	thromboxane A synthase 1 (platelet)	TBXAS1
ILMN_2284794	proteasome (prosome macropain) subunit beta type 8	PSMB8
ILMN_2286014	cation channel sperm associated 2	CATSPER 2
ILMN_2288483	centrosomal protein 19kDa	CEP19
ILMN_2289924	trafficking protein kinesin binding 1	TRAK1
ILMN_2290118	multiple EGF-like-domains 9	MEGF9
ILMN_2292178	C-type lectin domain family 12 member A	CLEC12A
ILMN_2292187	chimerin 2	CHN2
ILMN_2293511	Unnamed Transcript	
ILMN_2293758	A kinase (PRKA) anchor protein 11	AKAP11
ILMN_2293872	protein kinase AMP-activated gamma 2 non-catalytic subunit	PRKAG2
ILMN_2294751	activating signal cointegrator 1 complex subunit 3	ASCC3
ILMN_2295252	chromosome 9 open reading frame 72	C9orf72
ILMN_2296057	caspase 2 apoptosis-related cysteine peptidase	CASP2
ILMN_2296677	chromosome 9 open reading frame 72	C9orf72
ILMN_2301624	microtubule-actin crosslinking factor 1	MACF1
ILMN_2304624	eukaryotic translation initiation factor 4H	EIF4H
ILMN_2306565	metaxin 2	MTX2
ILMN_2307450	zinc finger protein 302	ZNF302
ILMN_2309245	bridging integrator 1	BIN1
ILMN_2310896	NLR family pyrin domain containing 3	NLRP3
ILMN_2310968	RUN and FYVE domain containing 1	RUFY1
ILMN_2311796	testis derived transcript (3 LIM domains)	TES
ILMN_2312606	interferon regulatory factor 5	IRF5
ILMN_2312709	leucine carboxyl methyltransferase 1	LCMT1
ILMN_2312732	dipeptidyl-peptidase 8	DPP8
ILMN_2313889	zinc finger protein 682	ZNF682
ILMN_2313901	peptidylglycine alpha-amidating monooxygenase	PAM

ILMN_2313926	CDC42 small effector 2	CDC42SE 2
ILMN_2318011	proteasome (prosome macropain) subunit alpha type 3	PSMA3
ILMN_2318638	TGFB-induced factor homeobox 1	TGIF1
ILMN_2318643	TGFB-induced factor homeobox 1	TGIF1
ILMN_2319000	megakaryocyte-associated tyrosine kinase	MATK
ILMN_2319077	Fas cell surface death receptor	FAS
ILMN_2320964	adenosine deaminase RNA-specific	ADAR
ILMN_2322375	v-maf avian musculoaponeurotic fibrosarcoma oncogene homolog F	MAFF
ILMN_2322458	optic atrophy 1 (autosomal dominant)	OPA1
ILMN_2322806	calpastatin	CAST
ILMN_2323172	colony stimulating factor 3 receptor (granulocyte)	CSF3R
ILMN_2323302	SON DNA binding protein	SON
ILMN_2323366	succinate dehydrogenase complex subunit C integral membrane prot	SDHC
ILMN_2323526	WW domain containing adaptor with coiled-coil	WAC
ILMN_2325837	CD3d molecule delta (CD3-TCR complex)	CD3D
ILMN_2329429	GTPase IMAP family member 6	GIMAP6
ILMN_2329773	RAB27A member RAS oncogene family	RAB27A
ILMN_2329927	ATP-binding cassette sub-family G (WHITE) member 1	ABCG1
ILMN_2330845	N-ethylmaleimide-sensitive factor	NSF
ILMN_2331082	membrane-spanning 4-domains subfamily A member 7	MS4A7
ILMN_2331087	membrane-spanning 4-domains subfamily A member 7	MS4A7
ILMN_2331544	myelin basic protein	MBP
ILMN_2332368	vacuolar protein sorting 13 homolog B (yeast)	VPS13B
ILMN_2333107	amino-terminal enhancer of split	AES
ILMN_2333440	transmembrane 9 superfamily member 1	TM9SF1
ILMN_2334989	chaperonin containing TCP1 subunit 3 (gamma)	CCT3
ILMN_2335704	neutrophil cytosolic factor 4 40kDa	NCF4
ILMN_2336130	Unnamed Transcript	
ILMN_2336280	QKI KH domain containing RNA binding	QKI
ILMN_2336781	superoxide dismutase 2 mitochondrial	SOD2
ILMN_2337655	tryptophanyl-tRNA synthetase	WARS
ILMN_2337928	chemokine (C-X-C motif) receptor 5	CXCR5
ILMN_2338323	cell division cycle 25B	CDC25B
ILMN_2339835	prostaglandin-endoperoxide synthase 1 (prostaglandin G/H synthase a	PTGS1
ILMN_2339955	nuclear receptor subfamily 4 group A member 2	NR4A2
ILMN_2340721	transmembrane protein 134	TMEM134
ILMN_2342121	proteasome (prosome macropain) 26S subunit non-ATPase 13	PSMD13

ILMN_2342638	asialoglycoprotein receptor 2	ASGR2
ILMN_2343010	bolA family member 3	BOLA3
ILMN_2344455	GTPase activating protein (SH3 domain) binding protein 1	G3BP1
ILMN_2345142	sulfatase 2	SULF2
ILMN_2345353	leukocyte specific transcript 1	LST1
ILMN_2346831	mannosyl (alpha-1 6-)-glycoprotein beta-1 2-N-acetylglucosaminyltr	MGAT2
ILMN_2347592	neuromedin B	NMB
ILMN_2347798	interferon alpha-inducible protein 6	IFI6
ILMN_2347888	La ribonucleoprotein domain family member 4	LARP4
ILMN_2347917	embryonic ectoderm development	EED
ILMN_2347943	zinc finger protein 384	ZNF384
ILMN_2348090	mitochondrial ribosomal protein L55	MRPL55
ILMN_2348093	ATP synthase H ⁺ transporting mitochondrial Fo complex subunit F	ATP5J
ILMN_2348243	STE20-related kinase adaptor alpha	STRADA
ILMN_2349600	bromodomain containing 8	BRD8
ILMN_2349831	dicer 1 ribonuclease type III	DICER1
ILMN_2352009	acyl-CoA dehydrogenase very long chain	ACADVL
ILMN_2352303	Ras association (RalGDS/AF-6) domain family member 2	RASSF2
ILMN_2352326	CoA synthase	COASY
ILMN_2352580	methyl-CpG binding domain protein 1	MBD1
ILMN_2353358	lectin galactoside-binding soluble 8	LGALS8
ILMN_2353633	egf-like module containing mucin-like hormone receptor-like 2	EMR2
ILMN_2354391	Unnamed Transcript	
ILMN_2354478	cytoplasmic FMR1 interacting protein 2	CYFIP2
ILMN_2355225	Unnamed Transcript	
ILMN_2355462	cytoplasmic FMR1 interacting protein 1	CYFIP1
ILMN_2355463	cytoplasmic FMR1 interacting protein 1	CYFIP1
ILMN_2355953	leukocyte immunoglobulin-like receptor subfamily B (with TM and IT)	LILRB4
ILMN_2356654	lectin galactoside-binding soluble 8	LGALS8
ILMN_2357193	DEAD (Asp-Glu-Ala-Asp) box polypeptide 59	DDX59
ILMN_2357419	leukocyte immunoglobulin-like receptor subfamily A (with TM domain)	LILRA5
ILMN_2357809	GTPase activating protein (SH3 domain) binding protein 1	G3BP1
ILMN_2358069	MAD1 mitotic arrest deficient-like 1 (yeast)	MAD1L1
ILMN_2358652	nuclear RNA export factor 1	NXF1
ILMN_2359601	calcium/calmodulin-dependent protein kinase II gamma	CAMK2G
ILMN_2361104	zinc finger matrin-type 3	ZMAT3
ILMN_2361807	osteosarcoma amplified 9 endoplasmic reticulum lectin	OS9

ILMN_2362232	zinc finger protein 331	ZNF331
ILMN_2362293	F-box protein 38	FBXO38
ILMN_2362368	U2 small nuclear RNA auxiliary factor 1	U2AF1
ILMN_2362581	fibronectin type III domain containing 3A	FNDC3A
ILMN_2362858	paired immunoglobulin-like type 2 receptor alpha	PILRA
ILMN_2362902	Ras association (RalGDS/AF-6) domain family member 5	RASSF5
ILMN_2363426	MYC associated factor X	MAX
ILMN_2364022	solute carrier family 16 (monocarboxylate transporter) member 3	SLC16A3
ILMN_2364272	muscleblind-like splicing regulator 2	MBNL2
ILMN_2364674	tRNA phosphotransferase 1	TRPT1
ILMN_2364971	protein phosphatase 2 regulatory subunit B' gamma	PPP2R5C
ILMN_2365091	Fc fragment of IgA receptor for	FCAR
ILMN_2365479	sorting nexin 1	SNX1
ILMN_2365484	sorting nexin 1	SNX1
ILMN_2365528	HAUS augmin-like complex subunit 8	HAUS8
ILMN_2366212	CD79b molecule immunoglobulin-associated beta	CD79B
ILMN_2366634	pyruvate kinase muscle	PKM
ILMN_2367113	caspase 6 apoptosis-related cysteine peptidase	CASP6
ILMN_2367215	prolylcarboxypeptidase (angiotensinase C)	PRCP
ILMN_2367418	osteoclast associated immunoglobulin-like receptor	OSCAR
ILMN_2367681	sperm associated antigen 1	SPAG1
ILMN_2367782	StAR-related lipid transfer (START) domain containing 7	STARD7
ILMN_2368530	interleukin 32	IL32
ILMN_2368597	SMG7 nonsense mediated mRNA decay factor	SMG7
ILMN_2369785	small nuclear ribonucleoprotein D2 polypeptide 16.5kDa	SNRPD2
ILMN_2370336	membrane-spanning 4-domains subfamily A member 4A	MS4A4A
ILMN_2370573	XIAP associated factor 1	XAF1
ILMN_2370772	eukaryotic translation initiation factor 4 gamma 1	EIF4G1
ILMN_2370882	acyl-CoA synthetase long-chain family member 5	ACSL5
ILMN_2370976	myoferlin	MYOF
ILMN_2371280	colony stimulating factor 3 receptor (granulocyte)	CSF3R
ILMN_2371590	DEAD (Asp-Glu-Ala-Asp) box helicase 17	DDX17
ILMN_2371825	amylo-alpha-1 6-glucosidase 4-alpha-glucanotransferase	AGL
ILMN_2372136	prolyl 4-hydroxylase transmembrane (endoplasmic reticulum)	P4HTM
ILMN_2372413	BH3 interacting domain death agonist	BID
ILMN_2372974	signal-regulatory protein alpha	SIRPA
ILMN_2373266	splicing regulatory glutamine/lysine-rich protein 1	SREK1
ILMN_2373335	ligase III DNA ATP-dependent	LIG3
ILMN_2373831	butyrophilin subfamily 3 member A3	BTN3A3
ILMN_2374036	cathepsin L	CTSL

ILMN_2374362	abhydrolase domain containing 17B	ABHD17B
ILMN_2374383	tetraspanin 17	TSPAN17
ILMN_2374778	deoxyuridine triphosphatase	DUT
ILMN_2375002	mitogen-activated protein kinase kinase kinase kinase 4	MAP4K4
ILMN_2375003	mitogen-activated protein kinase kinase kinase kinase 4	MAP4K4
ILMN_2375879	vascular endothelial growth factor A	VEGFA
ILMN_2376204	lymphotoxin beta (TNF superfamily member 3)	LTB
ILMN_2376205	lymphotoxin beta (TNF superfamily member 3)	LTB
ILMN_2376431	chemokine (C-C motif) receptor 2	CCR2
ILMN_2377019	coronin actin binding protein 1B	CORO1B
ILMN_2377980	protein phosphatase 1 catalytic subunit alpha isozyme	PPP1CA
ILMN_2378316	nucleoporin like 1	NUPL1
ILMN_2379469	eukaryotic translation initiation factor 3 subunit B	EIF3B
ILMN_2379599	CD163 molecule	CD163
ILMN_2379718	RAB24 member RAS oncogene family	RAB24
ILMN_2379967	Fc fragment of IgA receptor for	FCAR
ILMN_2380418	bicaudal D homolog 2 (Drosophila)	BICD2
ILMN_2380605	myotubularin related protein 3	MTMR3
ILMN_2380850	serologically defined colon cancer antigen 3	SDCCAG3
ILMN_2380999	RecQ helicase-like	RECQL
ILMN_2382403	Fc fragment of IgG low affinity IIb receptor (CD32)	FCGR2B
ILMN_2382500	membrane-spanning 4-domains subfamily A member 14	MS4A14
ILMN_2383419	glucocorticoid modulatory element binding protein 1	GMEB1
ILMN_2383516	WD repeat domain 7	WDR7
ILMN_2384181	dehydrogenase/reductase (SDR family) member 9	DHRS9
ILMN_2384216	5'-3' exoribonuclease 1	XRN1
ILMN_2385647	aminolevulinic acid delta-synthase 1	ALAS1
ILMN_2386100	BUB3 mitotic checkpoint protein	BUB3
ILMN_2386982	protein kinase C zeta	PRKCZ
ILMN_2387078	myelin protein zero-like 2	MPZL2
ILMN_2387452	methyl-CpG binding domain protein 2	MBD2
ILMN_2387791	mucosa associated lymphoid tissue lymphoma translocation gene 1	MALT1
ILMN_2388090	mitogen-activated protein kinase 14	MAPK14
ILMN_2388507	v-akt murine thymoma viral oncogene homolog 1	AKT1
ILMN_2388547	epithelial stromal interaction 1 (breast)	EPSTI1
ILMN_2388585	golgi-associated PDZ and coiled-coil motif containing	GOPC
ILMN_2388955	family with sequence similarity 212 member B	FAM212B
ILMN_2389347	nuclear receptor subfamily 3 group C member 1 (glucocorticoid re	NR3C1
ILMN_2389582	heterogeneous nuclear ribonucleoprotein L	HNRNPL
ILMN_2389844	Sp3 transcription factor	SP3

ILMN_2389935	forty-two-three domain containing 1	FYTTD1
ILMN_2390114	adaptor-related protein complex 3 delta 1 subunit	AP3D1
ILMN_2390299	proteasome (prosome macropain) subunit beta type 8	PSMB8
ILMN_2390310	MIR22 host gene (non-protein coding)	MIR22HG
ILMN_2390853	cathepsin H	CTSH
ILMN_2390859	nuclear factor of kappa light polypeptide gene enhancer in B-cells	NFKB2
ILMN_2391512	N-acylethanolamine acid amidase	NAAA
ILMN_2391861	Unnamed Transcript	
ILMN_2391912	SEC14-like 1 (<i>S. cerevisiae</i>)	SEC14L1
ILMN_2392274	CD82 molecule	CD82
ILMN_2392569	formyl peptide receptor 2	FPR2
ILMN_2393046	NHL repeat containing 3	NHLRC3
ILMN_2393169	THO complex 5	THOC5
ILMN_2393712	cortactin	CTTN
ILMN_2393763	actin related protein 2/3 complex subunit 4 20kDa	ARPC4
ILMN_2395474	REV1 polymerase (DNA directed)	REV1
ILMN_2395827	RAS guanyl releasing protein 4	RASGRP4
ILMN_2396020	dual specificity phosphatase 6	DUSP6
ILMN_2396410	citrate synthase	CS
ILMN_2397028	serpin peptidase inhibitor clade B (ovalbumin) member 8	SERPINB8
ILMN_2397521	lysine (K)-specific demethylase 6B	KDM6B
ILMN_2398865	vacuolar protein sorting 13 homolog C (<i>S. cerevisiae</i>)	VPS13C
ILMN_2399140	RAB5C member RAS oncogene family	RAB5C
ILMN_2399174	trafficking protein kinesin binding 1	TRAK1
ILMN_2399363	C-type lectin domain family 4 member A	CLEC4A
ILMN_2401714	membrane-spanning 4-domains subfamily A member 1	MS4A1
ILMN_2401933	ATPase Ca ⁺⁺ transporting plasma membrane 1	ATP2B1
ILMN_2402131	TAF15 RNA polymerase II TATA box binding protein (TBP)-associated	TAF15
ILMN_2402463	serine hydroxymethyltransferase 1 (soluble)	SHMT1
ILMN_2402806	transient receptor potential cation channel subfamily C member 4	TRPC4AP
ILMN_2403228	C-type lectin domain family 12 member A	CLEC12A
ILMN_2403237	chimerin 2	CHN2
ILMN_2403946	fasciculation and elongation protein zeta 2 (zygin II)	FEZ2
ILMN_2403994	RALY heterogeneous nuclear ribonucleoprotein	RALY
ILMN_2404063	amyloid beta (A4) precursor protein	APP
ILMN_2404065	amyloid beta (A4) precursor protein	APP
ILMN_2404135	RIO kinase 3	RIOK3
ILMN_2404407	4-aminobutyrate aminotransferase	ABAT
ILMN_2405129	Rho guanine nucleotide exchange factor (GEF) 1	ARHGEF1

ILMN_2405297	notch 2	NOTCH2
ILMN_2406335	ankyrin repeat domain 17	ANKRD17
ILMN_2406501	superoxide dismutase 2 mitochondrial	SOD2
ILMN_2407389	glycoprotein (transmembrane) nmb	GPNMB
ILMN_2407464	Fas-activated serine/threonine kinase	FASTK
ILMN_2408543	plasminogen activator urokinase receptor	PLAUR
ILMN_2408851	Rho GTPase activating protein 30	ARHGAP30
ILMN_2410540	caspase 2 apoptosis-related cysteine peptidase	CASP2
ILMN_2410783	glucosidase alpha; acid	GAA
ILMN_2410826	2'-5'-oligoadenylate synthetase 1 40/46kDa	OAS1
ILMN_2410909	v-akt murine thymoma viral oncogene homolog 1	AKT1
ILMN_2411139	fibroblast growth factor (acidic) intracellular binding protein	FIBP
ILMN_2412380	TSC22 domain family member 1	TSC22D1
ILMN_2412807	dynactin 1	DCTN1
ILMN_2413527	vinculin	VCL
ILMN_2414325	tumor necrosis factor alpha-induced protein 8	TNFAIP8
ILMN_2414762	toll-like receptor 10	TLR10
ILMN_2415303	C-type lectin domain family 10 member A	CLEC10A
ILMN_2415898	DnaJ (Hsp40) homolog subfamily B member 14	DNAJB14
ILMN_3176989	HAUS augmin-like complex subunit 8	HAUS8
ILMN_3184724	cadherin-like and PC-esterase domain containing 1	CPED1
ILMN_3193306	transmembrane protein 251	TMEM251
ILMN_3222974	protein kinase C beta	PRKCB
ILMN_3224926	RNA binding motif protein 47	RBM47
ILMN_3225432	late endosomal/lysosomal adaptor MAPK and MTOR activator 3	LAMTOR3
ILMN_3229812	phosphatidylinositol 4-kinase catalytic alpha	PI4KA
ILMN_3232156	cadherin-like and PC-esterase domain containing 1	CPED1
ILMN_3232529	phosphatidylinositol 4-kinase catalytic alpha pseudogene 1	PI4KAP1
ILMN_3234997	MAP7 domain containing 1	MAP7D1
ILMN_3235096	small nucleolar RNA H/ACA box 28	SNORA28
ILMN_3235185	small nuclear ribonucleoprotein 200kDa (U5)	SNRNP200
ILMN_3235514	G protein-coupled receptor 183	GPR183
ILMN_3235922	chromosome 11 open reading frame 21	C11orf21
ILMN_3236156	oligosaccharyltransferase complex subunit (non-catalytic)	OSTC
ILMN_3236211	DiGeorge syndrome critical region gene 11 (non-protein coding)	DGCR11
ILMN_3237177	family with sequence similarity 175 member A	FAM175A

ILMN_3237641	adaptor-related protein complex 5 mu 1 subunit	AP5M1
ILMN_3237956	zinc finger CCCH-type containing 12C	ZC3H12C
ILMN_3238435	small nucleolar RNA H/ACA box 12	SNORA12
ILMN_3238560	interferon alpha-inducible protein 27-like 2	IFI27L2
ILMN_3238680	chromosome 7 open reading frame 55	C7orf55
ILMN_3238955	small nucleolar RNA C/D box 10	SNORD10
ILMN_3239965	indoleamine 2 3-dioxygenase 1	IDO1
ILMN_3240144	Unnamed Transcript	
ILMN_3241626	pyroglutamylated RFamide peptide receptor	QRFPR
ILMN_3242377	NACC family member 2 BEN and BTB (POZ) domain containing	NACC2
ILMN_3244319	coiled-coil domain containing 125	CCDC125
ILMN_3245116	golgi integral membrane protein 4	GOLIM4
ILMN_3245616	CWC22 spliceosome-associated protein	CWC22
ILMN_3246409	heterogeneous nuclear ribonucleoprotein H1 (H)	HNRNPH1
ILMN_3247018	small nucleolar RNA H/ACA box 67	SNORA67
ILMN_3247882	ERI1 exoribonuclease family member 3	ERI3
ILMN_3247998	signal transducing adaptor family member 1	STAP1
ILMN_3248890	small nucleolar RNA H/ACA box 24	SNORA24
ILMN_3250209	C-terminal binding protein 2	CTBP2
ILMN_3250243	methyltransferase like 21A	METTTL21 A
ILMN_3250899	Unnamed Transcript	
ILMN_3251085	retinoblastoma binding protein 4	RBBP4
ILMN_3251137	methyltransferase like 21A	METTTL21 A
ILMN_3251293	thioredoxin-related transmembrane protein 1	TMX1
ILMN_3251506	zinc finger protein 69	ZNF69
ILMN_3251572	family with sequence similarity 134 member A	FAM134A
ILMN_3251629	Unnamed Transcript	
ILMN_3251699	amyloid beta (A4) precursor protein-binding family B member 2	APBB2
ILMN_3253579	HAUS augmin-like complex subunit 8	HAUS8
ILMN_3258795	family with sequence similarity 13 member B	FAM13B
ILMN_3269775	transmembrane protein 251	TMEM251
ILMN_3272500	interferon alpha-inducible protein 27-like 1	IFI27L1
ILMN_3299520	protein kinase C beta	PRKCB
ILMN_3299804	zinc finger protein 638	ZNF638
ILMN_3300313	prolyl 4-hydroxylase transmembrane (endoplasmic reticulum)	P4HTM
ILMN_3301197	late endosomal/lysosomal adaptor MAPK and MTOR activator 3	LAMTOR 3

ILMN_3302919	Myoferlin	MYOF
ILMN_3306730	RNA binding motif protein 47	RBM47
ILMN_3307648	citrate synthase	CS
ILMN_3308138	RNA U4 small nuclear 2	RNU4-2
ILMN_3308265	microRNA 302c	MIR302C
ILMN_3310371	microRNA 939	MIR939

Appendix 2

Overlapping Genes Implicated Between Conserved Transcriptional Response to Adversity Studies		
Antoni- Affect		
	w/ Antoni (Cognitive Stress Behavioral Management Therapy)	ALAS2, AVPI1, C15orf48, C3AR1, CCL20, CCL3, CCL3L1, CCL4L2, CCL7, CXCR7, EMP1, GJB2, GPR132, GPR84, HLA-A29.1, IFIT3, IFNG, IL1A, IL1B, IL6, LMNA, LOC651524, LYPD3, MIR155HG, MMP9, MXD1, OLR1, PLAUR, PTGR2, PTGS2, SC5DL, SERPINB2, SPSB1, THBS1, TNFRSF21, ZNF331
	w/ Cole (Loneliness)	FOSB, HIST1H2BG, IL1B, PTGS2
	w/Cole (Rearing)	GZMB, SERPINB2, TKTL1
	w/ Contrast Score (Fredrickson and Vedhara)	FOSB, IFIT3, IL1A, IL1B, IL6, OASL, PTGS2, TNF
	w/ Miller (Caregiving stress)	ACSL1, ADORA2A, AHSA1, CRISPLD2, CXCL10, DYNLL1, EREG, FOLR3, FOSB, HSPA1B, HSPA5, HSPH1, KCNJ2, PHACTR1, SERPINB2, THBS1, TKTL1, TNF, ZNF331
	w/ O'Connor (Complicated Grief)	CLIC3, CXCL10, HBD, HLA-A29.1, HLA-DRB1, HLA-DRB5, IFIT3, IL18RAP, KIR3DS1, LOC731682, MYOM2, OASL, SCGB3A1
	w/ O'Connor (Non-complicated Grief)	C19orf59, CXCL10, FFAR2, GK, HBG1, HBG2, HLA-A29.1, HLA-DRB1, HLA-DRB5, IFIT3, IL1RN, MIR1974, MSC, MYOM2
	w/Powell (Socioeconomic status)	ACSL1, AQP9, ARL5B, AVPI1, C15orf48, C3AR1, CCL20, CCL3, CCL3L1, CLIC3, CRISPLD2, CXCL10, EMP1, EREG, ETS2, FASLG, FOSB, GJB2, GPR84, HBA2, HBD, HBG1, HBG2, HBM, HIST1H2BG, HLA-DRB1, HLA-DRB5, IL1A, IL1B, IL1RN, KCNJ2, LRRC50, MAP3K8, MMP9, MS4A7, MXD1, NFE2L2, OLR1, PLA2G7, PLAUR, PTGS2, RIN2, SERPINB2, SOD2, THBS1, TKTL1, TNF, ZC3H12A
	w/ Wingo (Anxiety disorder)	LOC440280, LOC728069, PLAUR, RAXL1, RILPL2, SAMSN1, TREM1
Antoni- Cognitive Stress		

Behavioral Management Therapy		
	w/ Cole (Loneliness)	G0S2, IFI44, IL1B, PTGS2, RGS1, STAT1, TNFSF10
	w/Cole (Rearing)	IFIT1, IFIT2, SERPINB2
	w/ Contrast Score (Fredrickson and Vedhara)	IFI44, IFI44L, IFIT1, IFIT2, IFIT3, IL1A, IL1B, IL6, MX2, OAS2, OAS3, PTGS2
	w/ Miller (Caregiving stress)	GNA15, HERC5, IFIT1, ISG15, MX2, PHLDA1, SERPINB2, STAT1, THBD, THBS1, TNFSF10, TRIM22, ZNF331
	w/ O'Connor (Complicated Grief)	IFI44, IFI44L, IFIT1, IFIT3
	w/ O'Connor (Non-complicated Grief)	CCL2, IFI44L, IFIT1, IFIT2, IFIT3, LAG3, RGS1, RSAD2, TMEM158
	w/Powell (Socioeconomic status)	AVPI1, C15orf48, C3AR1, C5AR1, CCL20, CCL3, CCL3L1, CCL3L3, CCL4L1, CD300LB, CXCL2, CYP1B1, EMP1, G0S2, GJB2
	w/ Wingo (Anxiety disorder)	ABCG1, C5AR1, CTSL1, PLAUR, STAT1, STAT2
Cole-Loneliness		
	w/Cole (Rearing)	IGKC, IL8, MS4A1, PF4
	w/ Contrast Score (Fredrickson and Vedhara)	FOSB, IFI27, IFI44, IGJ, IGLL1, IL1B, IL8, OAS1, PTGS2
	w/ Miller (Caregiving stress)	ARTS-1, ATXN1, BHLHB2, BIRC1, CCR2, CDKN1C, FOSB, HLA-DQA1, HLA-DQB1, HLA-DRB4, IGF2R, IL8, LR8
	w/ O'Connor (Complicated Grief)	DEFA1, IFI44, MS4A1
	w/ O'Connor (Non-complicated Grief)	C21orf7, CA2, CD79B, DEFA1, HIST1H3H, IFI27, IGLL1, MS4A1, POU2AF1, PPBP, RGS1, RPS26, TUBB1

	w/Powell (Socioeconomic status)	CD79B, DEFA1, DUSP2, EGR1, FOSB, G0S2, HCA112, HIST1H2BG, HIST1H3H, HLA-DQB1, IFI27, IL1B, IL8
	w/ Wingo (Anxiety disorder)	HGD, KIAA1033, OAS1, SFRS6, STAT1
Cole- Rearing		
	w/ Contrast Score (Fredrickson and Vedhara)	FOSL2, GBP1, IFIT1, IFIT2, IFITM3, IL8, IRF7
	w/ Miller (Caregiving stress)	ARL4C, CCL5, FOSL2, GBP1, IFIT1, IL8, LILRA2, SERPINB2, TKTL1
	w/ O'Connor (Complicated Grief)	GBP1, IFIT1, MS4A1
	w/ O'Connor (Non- complicated Grief)	IFIT1, IFIT2, LILRA2, MS4A1
	w/Powell (Socioeconomic status)	EGR2, IFIT1, IL8, SERPINB2, TKTL1, TLR4
	w/ Wingo (Anxiety disorder)	CD48
Contrast Score (Fredrickson and Vedhara)		
	w/ Miller (Caregiving stress)	FOSB, FOSL2, GBP1, IFI16, IFI35, IFIH1, IFIT1, IL8, MX2, TNF
	w/ O'Connor (Complicated Grief)	GBP1, IFI44, IFIT1, IFIT3, MX1, OAS3, OASL
	w/ O'Connor (Non- complicated Grief)	IFI27, IFIT1, IFIT2, IFIT3, IGLL1
	w/Powell (Socioeconomic status)	FOSB, IFI27, IFIT1, IFITM1, IL1A, IL1B, IL8, PTGS2, TNF
	w/ Wingo	IFIH1, OAS1, REL

	(Anxiety disorder)	
Miller (Caregiving stress)		
	w/ O'Connor (Complicated Grief)	CXCL10, GBP1, IFIT1, RETN
	w/ O'Connor (Non-complicated Grief)	CD9, CXCL10, FZD2, IFIT1, IMPA2, LILRA2, LMO2, MNDA, NFE2, NQO2, P2RY13, SLC31A2
	w/Powell (Socioeconomic status)	ABCA1, ACSL1, ANPEP, ATF3, BCL3, CD163, CDKN1A, CPVL, CRISPLD2, CXCL10, CYFIP1, EMR2, EREG, FER1L3, FLJ22662, FOSB, GNA15, GRN, HBEGF, HK3, HLA-DQB1, IFIT1, IL8, IRS2, ISG15, KCNJ2, KIAA0513, MAD1L1, MNDA, NKG7, NOTCH2, PHLDA1, PLXNC1, PRKAR1A, RAB31, RTN1, SERPINB2, SLC31A2, SNF1LK, THBS1, TIPARP, TKTL1, TNF, TNS3, VNN1
	w/ Wingo (Anxiety disorder)	ARFGEF1, ATP2B1, CCDC56, CD1D, CLTC, CNDP2, COMT, CORO1A, CSF1R, GNS, HAT1, IFIH1, LSM1, NEDD9, PAFAH1B1, PEX11B, PKM2, PPP2CB, PPP3CA, PSMA3, PSMB8, QARS, RABGAP1L, RASSF2, SLC7A5, STAT1, VCL, ZFP106, ZNF313
O'Connor (Complicated Grief)		
	w/ O'Connor (Non-complicated Grief)	CAMP, CD19, CEACAM8, CXCL10, DEFA1, DEFA1B, DEFA3, DEFA4, E2F5, ELANE, ERAP2, FCRL2, GSTM1, GSTM2, HLA-A29.1, HLA-DRB1, HLA-DRB5, IFI44L, IFIT1, IFIT3, LCN2, LGALS2, LOC653600, LTF, MIAT, MS4A1, MYOM2, OSBPL10, PGLYRP1, RNASE3, RPS23, S100P, SNHG8, TCL1A, TLR10, TNFRSF13B, UTS2, ZNF683
	w/Powell (Socioeconomic status)	ACTA2, CAMP, CD19, CEACAM6, CEACAM8, CLIC3, CXCL10, DEFA1, DEFA3, DEFA4, GSTT1, HBD, HLA-DRB1, HLA-DRB5, HOXB2, IFIT1, KIR2DL1, KLRF1, LCN2, PTGDS
	w/ Wingo (Anxiety disorder)	CD160, D4S234E, KLRF1, LOC648868
O'Connor (Non-		

complicated Grief)		
	w/Powell (Socioeconomic status)	ACRBP, ALPL, ASGR2, CAMP, CD19, CD24, CD33, CD36, CD79A, CD79B, CEACAM8, CLEC10A, CXCL10, DEFA1, DEFA3, DEFA4, F13A1, FCER2, FCGR1A, FCGR1B, FPR1, GBGT1, GNG11, GPR162, HBG1, HBG2, HIST1H3H, HLA-DOB, HLA-DRB1, HLA-DRB5, IFI27, IFIT1, IL1RN, KIAA1598, LCN2, LOC90925, MND A, MS4A6A, NAPSB, PDK4, PRSS23, SLC31A2, VPREB3, ZNF467
	w/ Wingo (Anxiety disorder)	ACRBP, DDX51, F13A1, LOC441377, LRRC25, SLAMF6
Powell (Socioeconomic status)		
	w/ Wingo (Anxiety disorder)	ACRBP, C5AR1, CPNE8, CSF3R, F13A1, GIMAP7, KLRF1, LOC283345, LOC649946, MAFB, MYC, PILRA, PIM3, PLAUR, PLEKHF1, PTMA, SLC1A7, TIMP2, WARS, ZNF232

Appendix 3

Correlation Among Continuous Social Environmental Factors					
	Loneliness	Major or Lifetime Discrimination	Chronic Burden	Perceived Stress	Social support
Loneliness	-	0.178	0.357	0.302	-0.329
Major or Lifetime Discrimination			0.211	0.052	-0.165
Chronic Burden			-	0.270	-0.249
Perceived Stress				-	-0.347
Social Support					-

Appendix 4

Transcripts (Genes) with p<0.05 in Aim 1 Linear Regression Analyses by Social Environmental Factor													
Adult SES Illumina Transcript ID # & Gene Symbol		Chronic Burden Illumina Transcript ID # & Gene Symbol		Childhood SES Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination Illumina Transcript ID # & Gene Symbol		Loneliness Illumina Transcript ID # & Gene Symbol		Perceived Stress Illumina Transcript ID # & Gene Symbol		Social Support Illumina Transcript ID # & Gene Symbol	
ILMN_1652394	RAB2A	ILMN_1651735	TGO LN2	ILMN_1654262	ZMA T3	ILMN_1652198	CCM2	ILMN_1652825	IL10 RA	ILMN_1651433	DCK	ILMN_1652825	IL10R A
ILMN_1652777	CDC42EP2	ILMN_1652085	MPH OSP H1	ILMN_1658494	RGC C	ILMN_1654545	CPSF1	ILMN_1653443	CDK2	ILMN_1653266	DNA JB14	ILMN_1657619	DNAJ B14
ILMN_1652806	ATP5J	ILMN_1654545	CPSF1	ILMN_1662488	MED23	ILMN_1654812	UNC93B1	ILMN_1653480	CCD C125	ILMN_1653283	APP	ILMN_1660661	TCP1
ILMN_1652906	GBGT1	ILMN_1654560	N/A	ILMN_1664094	P2RY13	ILMN_1655177	PI4KA	ILMN_1655422	RPL17	ILMN_1655414	TNFSF14	ILMN_1662451	FCER2
ILMN_1656310	IDO1	ILMN_1654583	CHD1	ILMN_1671731	AVPI1	ILMN_1656011	RGS1	ILMN_1656011	RGS1	ILMN_1657857	TMEM14C	ILMN_1662896	WDR11
ILMN_1659463	APAF1	ILMN_1655177	PI4KA	ILMN_1672650	PKM	ILMN_1656184	N/A	ILMN_1656486	DNAJ C10	ILMN_1657977	MSRB2	ILMN_1664094	P2RY13
ILMN_1660027	FCGR2B	ILMN_1655930	ELL2	ILMN_1688098	TBC1D4	ILMN_1661695	IRAK3	ILMN_1657977	MSRB2	ILMN_1662358	MX1	ILMN_1665243	FKBP14
ILMN_1662617	PPP2R3C	ILMN_1656761	TGIF1	ILMN_1688959	CD27	ILMN_1664016	ARHG EF18	ILMN_1659227	CD79A	ILMN_1664098	FAS TK	ILMN_1667893	TNS3

ILMN_1663484	N/A	ILMN_1657797	FIBP	ILMN_1689274	NIPA1	ILMN_1666364	COQ10A	ILMN_16660847	PFKF B3	ILMN_16667476	LTB R	ILMN_16669062	N/A
ILMN_16664068	ERGI C1	ILMN_16661833	ANK RD12	ILMN_1693220	AKA P11	ILMN_16666552	ZNF75 A	ILMN_16661646	BAN K1	ILMN_16668979	DNA JB14	ILMN_1670145	DFN A5
ILMN_16665217	CEP1 9	ILMN_16663664	MRP S10	ILMN_1693771	ASP H	ILMN_16666733	CXCL 8	ILMN_16661695	IRAK 3	ILMN_1672503	DPY SL2	ILMN_1671250	CLIC 4
ILMN_16667594	KLF1 0	ILMN_16664016	ARH GEF1 8	ILMN_1695711	FAM 105A	ILMN_16666742	C9orf7 2	ILMN_16662795	CA2	ILMN_1673352	IFIT M2	ILMN_1673586	SLC6 A6
ILMN_16667711	PLA2 G16	ILMN_16664449	ALG 5	ILMN_1696041	CDC 42	ILMN_16667460	SULF2	ILMN_16663664	MRP S10	ILMN_1674574	VNN 1	ILMN_1675156	CDC4 2
ILMN_16668484	LRR C47	ILMN_16665049	SPG1 1	ILMN_1696806	N/A	ILMN_16668345	OAF	ILMN_16665964	GAB 2	ILMN_1675756	KCN J15	ILMN_1683023	PDGF C
ILMN_16668979	DNA JB14	ILMN_16665192	NUD T6	ILMN_1700306	OCI AD2	ILMN_16669523	FOS	ILMN_16666932	FCG R2A	ILMN_1678546	PEX 11B	ILMN_1685057	SLC2 2A4
ILMN_16669062	N/A	ILMN_16665964	GAB 2	ILMN_1707137	C17orf97	ILMN_16669888	CSTA	ILMN_16667796	N/A	ILMN_1683786	N/A	ILMN_1686884	IL1R AP
ILMN_1670925	CYB 5D1	ILMN_16667068	ZC3H AV1	ILMN_1708041	PLE KHF 1	ILMN_1670439	FYTT D1	ILMN_16668134	GST M1	ILMN_1684585	ACS L1	ILMN_1689734	IL1R N
ILMN_1674394	APM AP	ILMN_16667460	SULF 2	ILMN_1710734	GZM K	ILMN_1671509	CCL3	ILMN_16668345	OAF	ILMN_1685009	ITG AM	ILMN_1694966	ASG R2
ILMN_1675156	CDC 42	ILMN_16667893	TNS3	ILMN_1713803	C17orf97	ILMN_1671891	PID1	ILMN_16668526	GVIN P1	ILMN_1686645	UTP 14C	ILMN_1696432	IDH1

ILMN_1678293	N/A	ILMN_1668484	LRR C47	ILMN_1717063	FBX O9	ILMN_1672660	MBP	ILMN_1669523	FOS	ILMN_1687785	PPA 2	ILMN_1696749	LMN A
ILMN_1679268	PELI 1	ILMN_1670145	DFN A5	ILMN_1717261	HLA-DRB 3	ILMN_1673113	F2RL1	ILMN_1670134	FADS 1	ILMN_1688152	IL27 RA	ILMN_1697469	SRSF 6
ILMN_1680687	NSF	ILMN_1670322	FCH O2	ILMN_1718718	MKK S	ILMN_1674574	VNN1	ILMN_1670145	DFN A5	ILMN_1688452	LCM T1	ILMN_1700340	ASG R2
ILMN_1681301	AIM2	ILMN_1671731	AVPI 1	ILMN_1719232	DGC R14	ILMN_1677511	PTGS2	ILMN_1670926	CHST 15	ILMN_1690476	QKI	ILMN_1703229	N/A
ILMN_1687410	OSBP L11	ILMN_1673023	EP40 0	ILMN_1721081	SP4	ILMN_1678707	TAF15	ILMN_1674574	VNN 1	ILMN_1693338	CYP 1B1	ILMN_1703617	AHS A1
ILMN_1693338	CYP1 B1	ILMN_1673478	FAM 13B	ILMN_1735151	EIF5 A2	ILMN_1679929	KLF13	ILMN_1675669	IBTK	ILMN_1694268	HES 6	ILMN_1703697	LAN CL1
ILMN_1695316	SLC3 9A8	ILMN_1675156	CDC 42	ILMN_1739674	N/A	ILMN_1680618	MYC	ILMN_1677511	PTGS 2	ILMN_1694587	EEF1 B2	ILMN_1703946	ADO RA2B
ILMN_1695590	ADR B2	ILMN_1679232	KIDI NS22	ILMN_1746426	TOM M70 A	ILMN_1682098	PSMA 4	ILMN_1677574	GAL NT6	ILMN_1694966	ASG R2	ILMN_1704713	CSN K1G1
ILMN_1696432	IDH1	ILMN_1679727	CLK1	ILMN_1749253	TUB D1	ILMN_1682717	IER3	ILMN_1679268	PELI 1	ILMN_1696584	ORM 1	ILMN_1706553	SMG 7
ILMN_1696975	USP1	ILMN_1679929	KLF1 3	ILMN_1751400	SKA P1	ILMN_1682781	TEAD 2	ILMN_1679727	CLK1	ILMN_1699878	RAB 27A	ILMN_1714650	RAS GRP4
ILMN_1699071	MAP 3K7C L	ILMN_1684982	PDK4	ILMN_1765801	GAA	ILMN_1683023	PDGF C	ILMN_1682147	HOO K2	ILMN_1701906	CD3 00C	ILMN_1714896	SART 3

ILMN_1699423	ZNF146	ILMN_1685009	ITGAM	ILMN_1768958	RASGRP1	ILMN_1685057	SLC22A4	ILMN_1682717	IER3	ILMN_1703650	TNIP1	ILMN_1720623	SYTL3
ILMN_1699878	RAB27A	ILMN_1685122	COL9A2	ILMN_1772387	TLR2	ILMN_1685289	C16orf58	ILMN_1682938	ARF3	ILMN_1708934	ADM	ILMN_1721868	KPNA2
ILMN_1700306	OCIA D2	ILMN_1685289	C16orf58	ILMN_1773935	TMEM165	ILMN_1685521	KLRF1	ILMN_1683023	PDGFC	ILMN_1710514	BCL3	ILMN_1722276	PAFAH1B1
ILMN_1700340	ASGR2	ILMN_1686135	CEP95	ILMN_1775327	PKM	ILMN_1685625	UCP2	ILMN_1683026	PSMB10	ILMN_1713162	GSTM2	ILMN_1724658	BNIP3
ILMN_1700584	IER2	ILMN_1687998	LPGAT1	ILMN_1775501	IL1B	ILMN_1686623	CSF1R	ILMN_1683127	ZNF281	ILMN_1714896	SART3	ILMN_1725244	HAT1
ILMN_1700695	SLC44A1	ILMN_1689274	NIPA1	ILMN_1780334	KCNJ2	ILMN_1686981	SULF2	ILMN_1683792	LAP3	ILMN_1726597	FAM65B	ILMN_1727762	CASP1
ILMN_1701111	GPCPD1	ILMN_1689400	CLK1	ILMN_1786065	UHRF1	ILMN_1688231	TREM1	ILMN_1685057	SLC22A4	ILMN_1727051	DDX19A	ILMN_1728478	CXCL16
ILMN_1703180	ETV3	ILMN_1690610	RALY	ILMN_1801616	EMP1	ILMN_1691717	RHBD F2	ILMN_1685445	NOTCH2NL	ILMN_1727965	ALDH3B1	ILMN_1732452	MAPKAPK3
ILMN_1703946	ADORA2B	ILMN_1690822	VAPA	ILMN_1802404	ABCC1	ILMN_1692962	CTDSP2	ILMN_1685678	EEF1B2	ILMN_1728047	AKR1A1	ILMN_1732575	SEC14L1
ILMN_1708416	ARL6IP1	ILMN_1691508	PLAUR	ILMN_1804448	MSI2	ILMN_1693242	ZNF296	ILMN_1687533	SEMA4D	ILMN_1728662	ALDH3B1	ILMN_1737394	LMNA
ILMN_1708934	ADM	ILMN_1692742	DEND3	ILMN_1807833	HM13	ILMN_1693352	MRPL20	ILMN_1687998	LPGAT1	ILMN_1730986	MALT1	ILMN_1737396	PSMD14

ILMN_1713266	FAM46C	ILMN_1692962	CTD SP2	ILMN_1815283	N/A	ILMN_1693352	MRPL20	ILMN_1689160	DPEP2	ILMN_1740015	CD14	ILMN_1739586	FEZ2
ILMN_1715169	HLA-DRB1	ILMN_1693045	TME D1	ILMN_2087646	HLX	ILMN_1694274	NDUF C2	ILMN_1691071	FCRLA	ILMN_1741165	SLC11A1	ILMN_1745788	CX3CR1
ILMN_1717163	F13A1	ILMN_1693136	VTI1 B	ILMN_2123557	FAM73A	ILMN_1694603	SMARCC1	ILMN_1691364	STAT1	ILMN_1741464	HOO K3	ILMN_1746846	TTLL4
ILMN_1718565	CDKN1C	ILMN_1693650	FES	ILMN_2159694	HLA-DRB4	ILMN_1695423	CD9	ILMN_1691693	N/A	ILMN_1744517	GNS	ILMN_1762899	EGR1
ILMN_1722622	CD163	ILMN_1695316	SLC39A8	ILMN_2159694_1	N/A	ILMN_1695640	PTPN22	ILMN_1691892	TAGLN2	ILMN_1745423	UTP3	ILMN_1777058	N/A
ILMN_1724658	BNIP3	ILMN_1696420	BRD7	ILMN_2186108	DGCR6	ILMN_1695711	FAM105A	ILMN_1695590	ADRB2	ILMN_1748473	GIMAP4	ILMN_1778136	ZMYND15
ILMN_1729691	SLC16A6	ILMN_1699071	MAP3K7CL	ILMN_2261416	CD3D	ILMN_1696432	IDH1	ILMN_1696360	CTSB	ILMN_1749641	FBXO3	ILMN_1785345	GPR84
ILMN_1732296	ID3	ILMN_1699695	TNFRSF21	ILMN_2319000	MATK	ILMN_1699908	IL12RB1	ILMN_1696419	STOM	ILMN_1750395	MBD2	ILMN_1785732	TNFAIP6
ILMN_1735453	FAM98A	ILMN_1699836	LSP1	ILMN_2325837	CD3D	ILMN_1700413	MAFF	ILMN_1696420	BRD7	ILMN_1756417	ANKRD37	ILMN_1789990	ARFGEF2
ILMN_1736068	CNO T8	ILMN_1703891	TBC1D9	ILMN_2333107	AES	ILMN_1700584	IER2	ILMN_1696432	IDH1	ILMN_1763837	ANPEP	ILMN_1800540	CD55
ILMN_1737003	N/A	ILMN_1703949	KPNB1	ILMN_2336130	N/A	ILMN_1702114	N/A	ILMN_1696933	NLRP3	ILMN_1766269	HM13	ILMN_1801616	EMP1

ILMN_ 173725 2	NRG 1	ILMN_ 170467 2	NAB P2	ILMN_2 338323	CDC 25B	ILMN_1 703650	TNIP1	ILMN_ 169990 8	IL12 RB1	ILMN_ 176719 3	CR1	ILMN_ 180392 5	MTM R3
ILMN_ 174763 0	DEK	ILMN_ 170479 7	CLE C10A	ILMN_2 355462	CYFI P1	ILMN_1 703891	TBC1 D9	ILMN_ 170014 7	VPRE B3	ILMN_ 176851 0	MA N2B 2	ILMN_ 180737 2	ADO RA2A
ILMN_ 174774 4	LHFP L2	ILMN_ 170568 6	NRG N	ILMN_2 355463	CYFI P1	ILMN_1 703949	KPNB 1	ILMN_ 170030 6	OCIA D2	ILMN_ 176929 9	MT MR1 1	ILMN_ 181027 4	HOX B2
ILMN_ 174888 3	CDK N2D	ILMN_ 170706 2	REV1	ILMN_2 368530	IL32	ILMN_1 707312	NFIL3	ILMN_ 170034 0	ASG R2	ILMN_ 176954 6	RIN2	ILMN_ 181048 6	RAB3 4
ILMN_ 175140 0	SKA P1	ILMN_ 170816 4	EIF3 A	ILMN_2 368597	SMG 7	ILMN_1 707551	AFMI D	ILMN_ 170058 4	IER2	ILMN_ 177292 9	ATP 5J	ILMN_ 181552 7	HBD
ILMN_ 175677 9	CLT C	ILMN_ 170893 4	ADM	ILMN_2 375879	VEG FA	ILMN_1 707815	N/A	ILMN_ 170171 1	BRD8	ILMN_ 177487 4	IL1R N	ILMN_ 208861 2	XPO4
ILMN_ 175998 3	DR1	ILMN_ 170903 2	FYC O1	ILMN_2 376204	LTB	ILMN_1 708164	EIF3A	ILMN_ 170269 1	TNF AIP3	ILMN_ 178421 8	DDX 23	ILMN_ 211566 9	SEM A4C
ILMN_ 176271 3	MCE MP1	ILMN_ 171149 0	ARH GAP2 6	ILMN_2 376205	LTB	ILMN_1 708416	ARL6I P1	ILMN_ 170394 6	ADO RA2B	ILMN_ 178500 5	NCF 4	ILMN_ 212901 5	AFF1
ILMN_ 176719 3	CR1	ILMN_ 171201 9	ANK RD17	ILMN_2 392274	CD82	ILMN_1 708881	RAB2 0	ILMN_ 170578 3	NXF1	ILMN_ 178665 8	BOL A3	ILMN_ 213259 9	ANK RD22
ILMN_ 176839 3	SNRP D1	ILMN_ 171295 9	DUS P2	ILMN_2 395827	RAS GRP 4	ILMN_1 709817	SLC35 A5	ILMN_ 170888 1	RAB2 0	ILMN_ 178841 6	ABH D17 C	ILMN_ 225823 4	KLH L5
ILMN_ 176858 2	PPP2 CB	ILMN_ 171366 8	TSN AX	ILMN_2 412380	TSC2 2D1	ILMN_1 712673	SASH 1	ILMN_ 171001 7	CD79 B	ILMN_ 178854 7	GCL M	ILMN_ 227699 6	CCR2

ILMN_ 176895 8	RAS GRP1	ILMN_ 171496 5	NFK B1			ILMN_1 713749	CORO 1A	ILMN_ 171202 6	NLRP 3	ILMN_ 179416 5	PGD	ILMN_ 228264 1	TBX AS1
ILMN_ 176954 6	RIN2	ILMN_ 171718 0	MTM R6			ILMN_1 714093	RMND 5A	ILMN_ 171257 7	FAM 174A	ILMN_ 179571 5	DPY D	ILMN_ 231089 6	NLRP 3
ILMN_ 177097 7	TME M134	ILMN_ 171863 3	LRP5 L			ILMN_1 715068	AQP9	ILMN_ 171294 4	AES	ILMN_ 180150 4	RUN X1	ILMN_ 232096 4	ADA R
ILMN_ 177296 4	CCL8	ILMN_ 171880 7	SMC 3			ILMN_1 715636	EIF3B	ILMN_ 171326 6	FAM 46C	ILMN_ 180225 7	PCT P	ILMN_ 234263 8	ASG R2
ILMN_ 177393 5	TME M165	ILMN_ 172205 9	SAFB			ILMN_1 716276	N/A	ILMN_ 171489 6	SART 3	ILMN_ 180870 7	FSC N1	ILMN_ 236258 1	FND C3A
ILMN_ 177396 3	GNA 15	ILMN_ 172283 8	MRP L46			ILMN_1 717973	N/A	ILMN_ 171863 3	LRP5 L	ILMN_ 180952 2	NAC C2	ILMN_ 240650 1	SOD2
ILMN_ 177532 7	PKM	ILMN_ 172396 9	PLCB 1			ILMN_1 718960	SERPI NB8	ILMN_ 171897 7	GAD D45B	ILMN_ 181083 8	MTD H	ILMN_ 323615 6	OSTC
ILMN_ 177941 0	VAP A	ILMN_ 172709 8	PPP1 R16B			ILMN_1 719316	TMED 3	ILMN_ 171990 5	TLR1 0	ILMN_ 181508 6	NINJ 1		
ILMN_ 178473 7	S1PR 4	ILMN_ 172816 3	CTD SP1			ILMN_1 719392	FH	ILMN_ 172015 8	ETS2	ILMN_ 181552 7	HBD		
ILMN_ 178517 5	SWA P70	ILMN_ 172847 1	ARF GEF1			ILMN_1 719627	SLC27 A3	ILMN_ 172077 1	STX1 1	ILMN_ 204422 6	PPP3 CA		
ILMN_ 178741 0	EIF6	ILMN_ 173104 8	TLR1			ILMN_1 720771	STX11	ILMN_ 172262 2	CD16 3	ILMN_ 205341 5	LDL R		

ILMN_ 178746 1	RUN X3	ILMN_ 173247 5	STY X			ILMN_1 722294	CPNE 8	ILMN_ 172298 1	TLR5	ILMN_ 206200 1	HOO K3		
ILMN_ 178841 6	ABH D17C	ILMN_ 173322 1	SEM A4D			ILMN_1 722811	CDKN 1B	ILMN_ 172449 5	SEST D1	ILMN_ 208281 0	BRD 7		
ILMN_ 178928 3	PPP2 R5C	ILMN_ 173702 5	PLCL 2			ILMN_1 723035	OLR1	ILMN_ 172524 4	HAT1	ILMN_ 208383 3	CNO T6L		
ILMN_ 179175 9	CXC L10	ILMN_ 173739 6	PSM D14			ILMN_1 724250	GRN	ILMN_ 172727 1	WAR S	ILMN_ 213753 6	ZZZ 3		
ILMN_ 179607 5	VPS4 1	ILMN_ 173781 3	ERI3			ILMN_1 726288	TMEM 106B	ILMN_ 173063 9	SLC2 2A15	ILMN_ 217128 9	SAM SN1		
ILMN_ 179782 2	SEL1 L3	ILMN_ 173890 9	TRO VE2			ILMN_1 728163	CTDS P1	ILMN_ 173112 3	RNF7	ILMN_ 217217 4	PNP		
ILMN_ 179808 5	EID2 B	ILMN_ 174074 2	URO D			ILMN_1 728698	GDE1	ILMN_ 173268 8	DUT	ILMN_ 221615 7	GNA 12		
ILMN_ 179870 6	GPR1 83	ILMN_ 174188 1	C9orf 72			ILMN_1 729976	CHAM P1	ILMN_ 173487 8	CD79 A	ILMN_ 227872 9	EIF3 B		
ILMN_ 180060 2	GCA	ILMN_ 174507 5	RPLP 0			ILMN_1 730986	MALT 1	ILMN_ 173702 5	PLCL 2	ILMN_ 229605 7	CAS P2		
ILMN_ 180115 6	RLF	ILMN_ 174535 6	CXC L9			ILMN_1 731233	GZMH	ILMN_ 173739 6	PSM D14	ILMN_ 231096 8	RUF Y1		
ILMN_ 180381 9	IQGA P1	ILMN_ 174577 2	ASC C3			ILMN_1 735453	FAM9 8A	ILMN_ 173751 4	KYN U	ILMN_ 235595 3	LILR B4		

ILMN_ 180417 4	FCG R2B	ILMN_ 174936 8	HIST 1H3H			ILMN_1 737254	USP1	ILMN_ 173842 4	CDC4 2	ILMN_ 236402 2	SLC 16A3		
ILMN_ 180737 2	ADO RA2 A	ILMN_ 174941 9	PRK AG2			ILMN_1 740171	DUSP 11	ILMN_ 173863 2	PRK AR1 A	ILMN_ 237996 7	FCA R		
ILMN_ 180840 5	HLA- DQA 1	ILMN_ 175007 5	DMT F1			ILMN_1 740875	FPR2	ILMN_ 174191 7	OSC AR	ILMN_ 238341 9	GME B1		
ILMN_ 180870 7	FSCN 1	ILMN_ 175080 5	ARH GAP3 0			ILMN_1 741148	ALDO A	ILMN_ 174240 0	CEP3 50	ILMN_ 239227 4	CD8 2		
ILMN_ 181502 3	PIM1	ILMN_ 175224 9	PIEZ O1			ILMN_1 745423	UTP3	ILMN_ 174318 7	C6orf 120	ILMN_ 239371 2	CTT N		
ILMN_ 181573 3	EIF5	ILMN_ 175260 6	N/A			ILMN_1 745788	CX3C R1	ILMN_ 174578 8	CX3C R1	ILMN_ 241113 9	FIBP		
ILMN_ 208063 7	ZBT B44	ILMN_ 175306 4	TTC1 3			ILMN_1 745994	GAS7	ILMN_ 174631 7	NUP L1	ILMN_ 323499 7	MAP 7D1		
ILMN_ 210192 0	HNR NPH1	ILMN_ 175311 1	NAM PT			ILMN_1 747355	N/A	ILMN_ 174642 6	TOM M70 A	ILMN_ 326977 5	TME M25 1		
ILMN_ 211566 9	SEM A4C	ILMN_ 175442 1	NDU FAF1			ILMN_1 756417	ANKR D37	ILMN_ 174750 4	AHC TF1				
ILMN_ 212415 5	ATP1 1B	ILMN_ 175734 7	KIAA 0930			ILMN_1 756723	DPP7	ILMN_ 174807 7	DDX 59				
ILMN_ 212992 7	EXT1	ILMN_ 175814 6	SIRP A			ILMN_1 758939	RIPK2	ILMN_ 175160 7	FOSB				

ILMN_ 215483 6	BTG3	ILMN_ 175877 8	CNT RL			ILMN_1 759008	ZNF68 9	ILMN_ 175252 6	RNF1 44B				
ILMN_ 216821 7	GPR1 83	ILMN_ 175891 8	BRD 2			ILMN_1 759084	INTS8	ILMN_ 175641 7	ANK RD37				
ILMN_ 217128 9	SAM SN1	ILMN_ 175995 2	PSM A5			ILMN_1 760849	NETO 2	ILMN_ 175680 6	MCL 1				
ILMN_ 217217 4	PNP	ILMN_ 176297 2	CHD 9			ILMN_1 762674	NUP43	ILMN_ 175841 8	TNFS F13B				
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Appendix 5

Transcripts (Genes) Selected via Elastic Net in Aim 1 Analyses by Social Environmental Factor											
Adult SES Illumina Transcript ID # & Gene Symbol		Chronic Burden Illumina Transcript ID # & Gene Symbol		Childhood SES Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination Illumina Transcript ID # & Gene Symbol		Loneliness Illumina Transcript ID # & Gene Symbol		Social Support Illumina Transcript ID # & Gene Symbol	
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ILMN_16 61409	FAM20 8A	ILMN_16 89160	DPEP 2			ILMN_16 85521	KLRF1	ILMN_16 53480	CCDC1 25	ILMN_16 96432	IDH1
ILMN_16 62617	PPP2R 3C	ILMN_16 89734	IL1RN			ILMN_16 95640	PTPN22	ILMN_16 56011	RGS1	ILMN_17 03946	ADOR A2B
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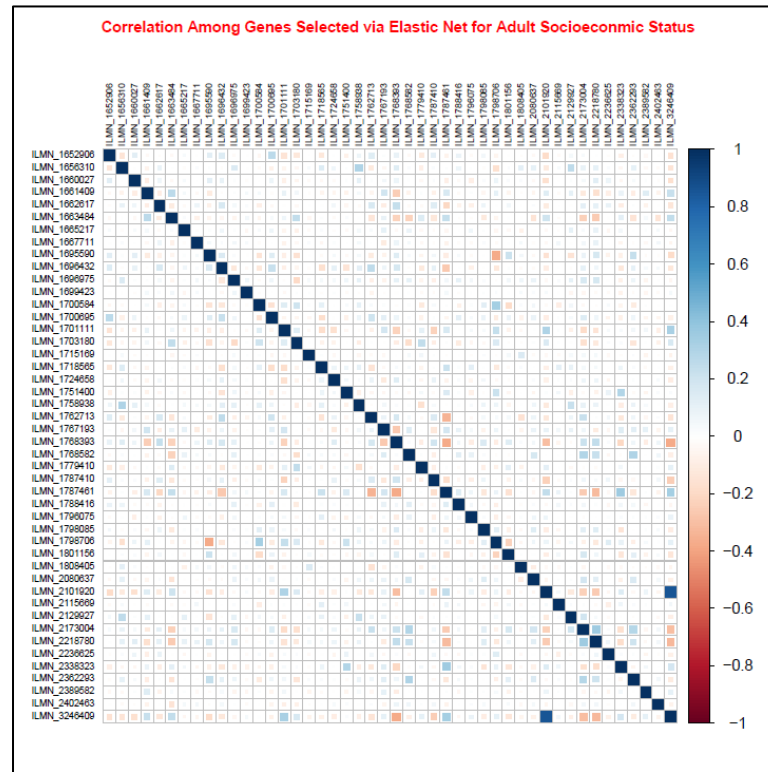
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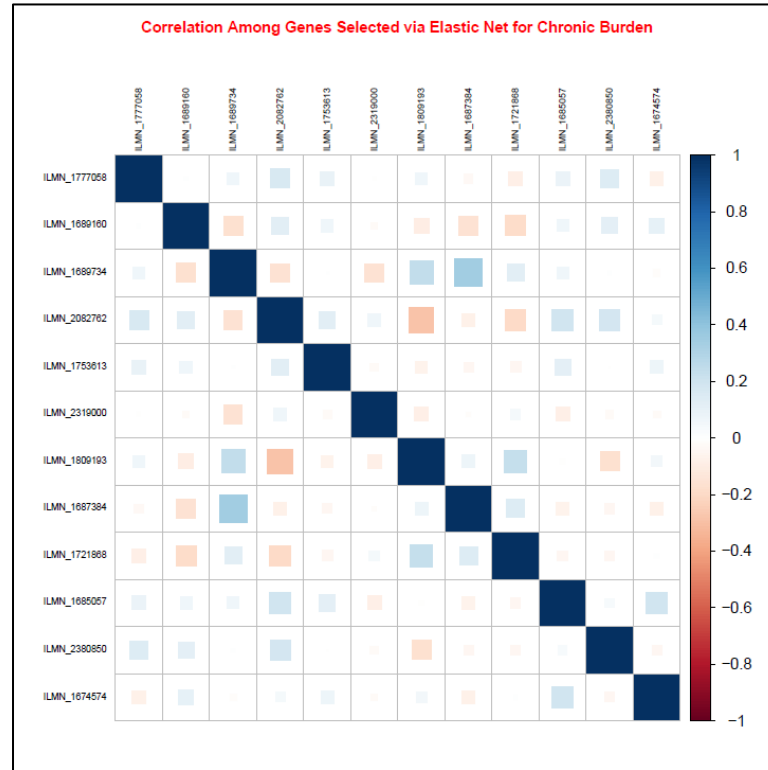
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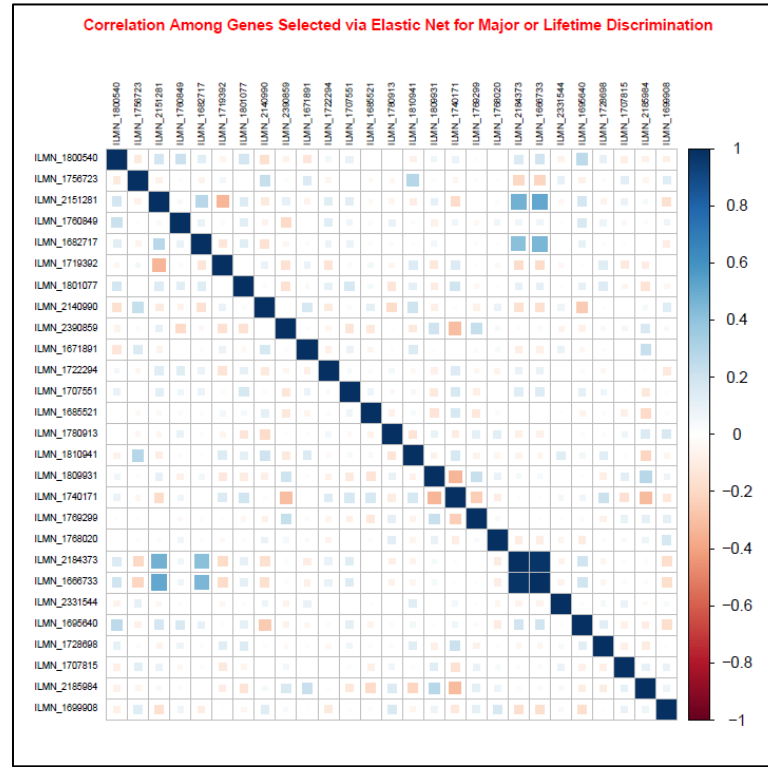
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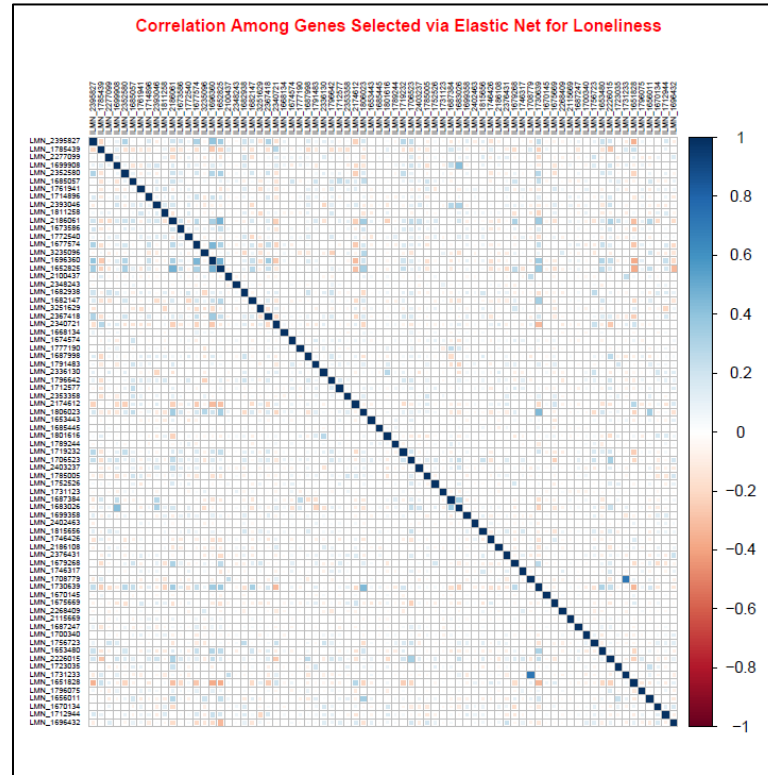
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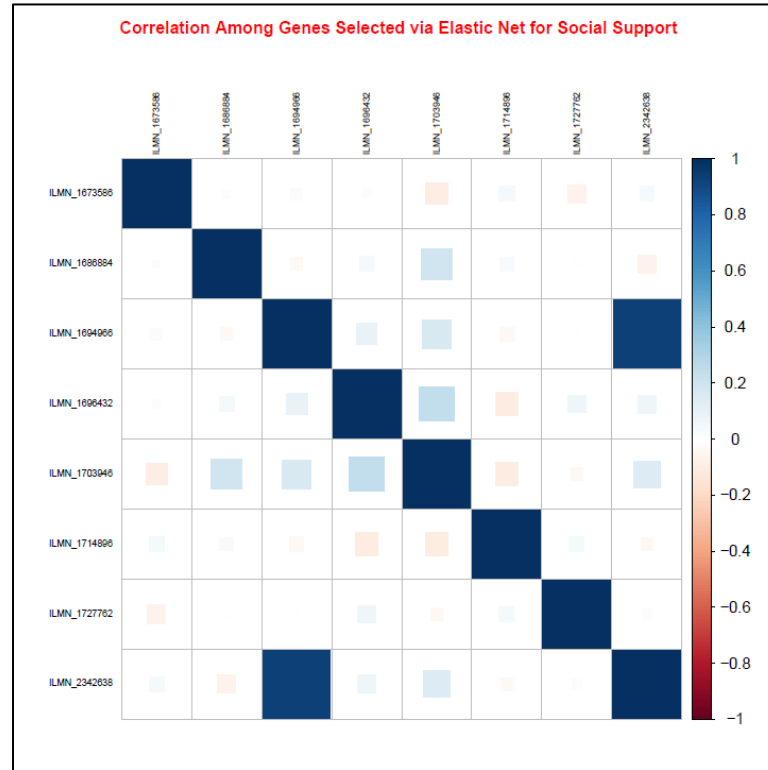
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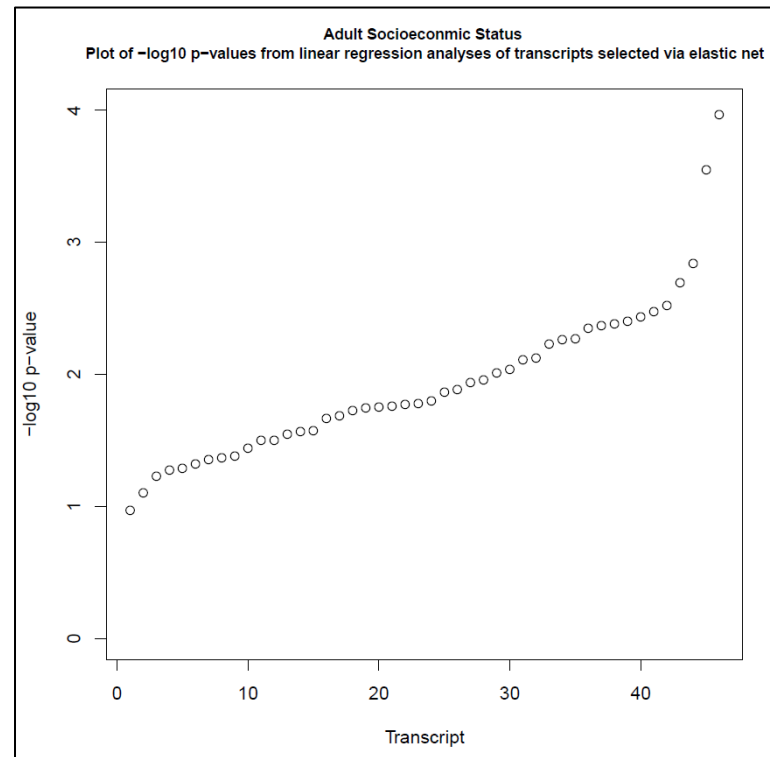
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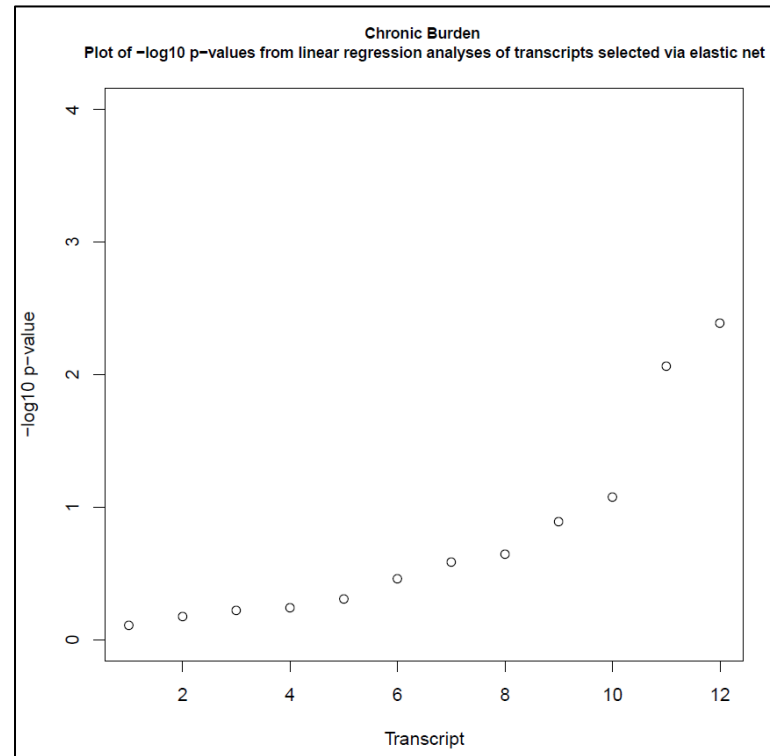
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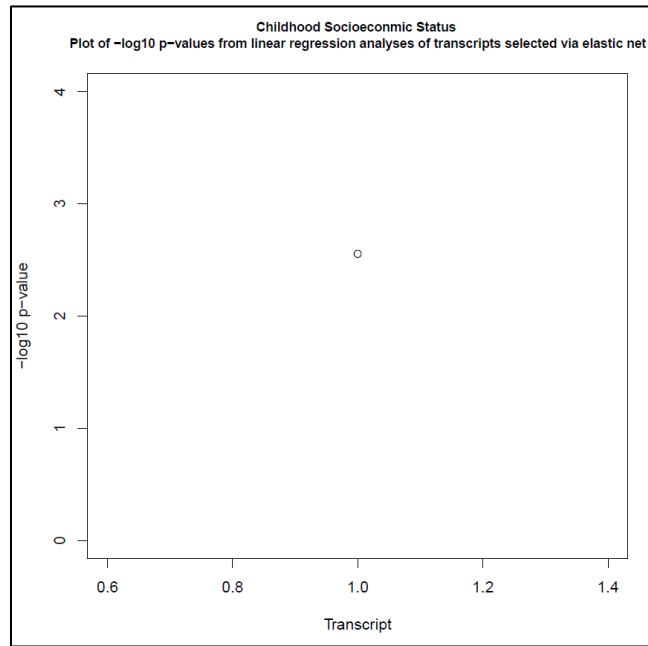
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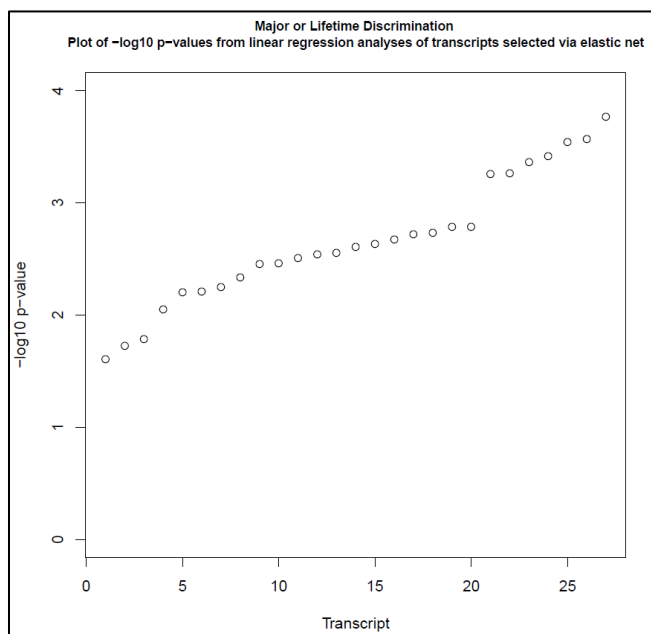
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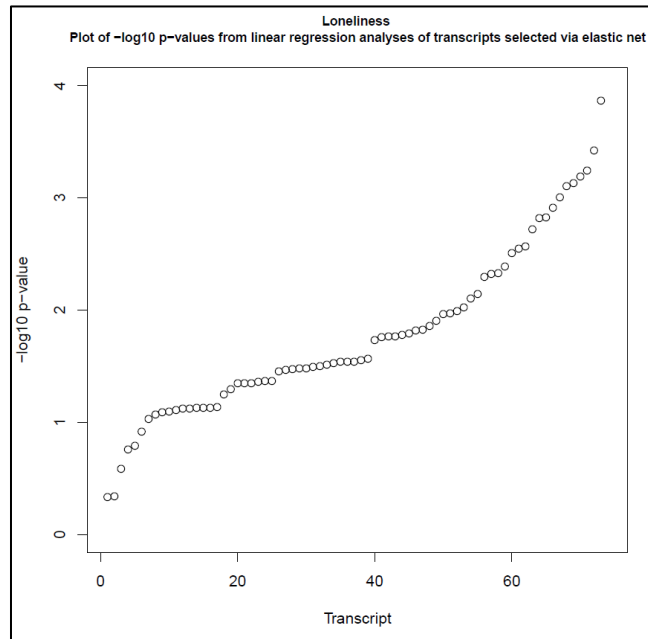
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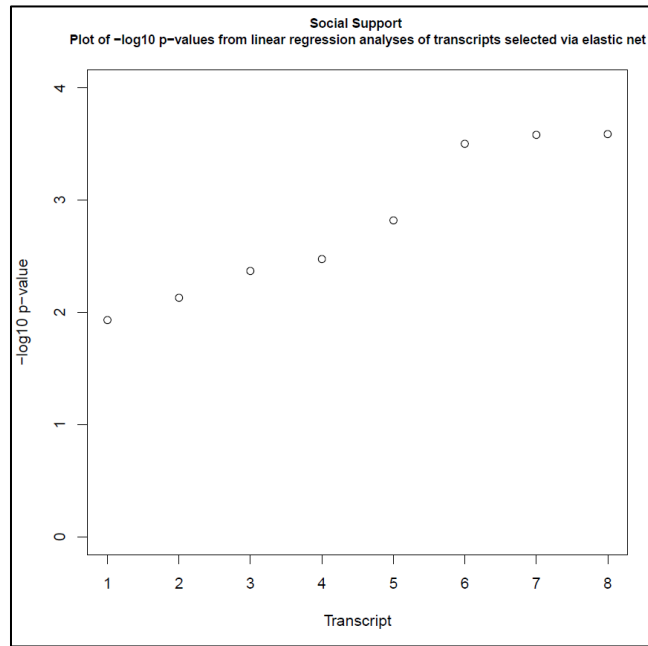
Appendix 14



Appendix 15



Appendix 16



Appendix 17

Gene List for Each Investigated Gene Ontology Biological Processes	
Biological Process	Genes in Biological Process
Chronic Inflammation	IDO1, AHCY, VNN1, THBS1, CAMP, IL1RN, CEBPB, TNFAIP3, ADORA2B, TNF, CHAMP1, CX3CR1, CCL5, UNC13D, IL1RN, IL1B, LTA, CX3CR1, CCL5, IDO1
Immune Response	ZFPM1, CTNNA1, PNKD, IL4R, CCM2, SECTM1, ERCC1, IPO7, PIK3AP1, ST6GAL1, CLEC10A, APP, IGSF6, SIKE1, IL18BP, PTPRC, PDPK1, BCL2L1, CD1C, HMGB2, ADCY9, CLEC7A, RASA3, TRIM32, UNC93B1, ABR, TNFSF14, TNFAIP1, TRIM27, RGS1, DHX9, IDO1, DUSP5, TRIM6, CRIP1, ACTR3, RNF168, SPPL3, SPG21, SRPK2, RPS6KA5, VAV3, CFH, AHCY, RSAD2, MAP2K2, OAS1, KLRG1, RGCC, MRE11A, DEAF1, POLR2K, RARA, CD79A, TNFRSF8, SEC61A1, ISG20, FCGR2B, SPN, CLCF1, SPTBN1, IRAK3, MEF2A, MX1, FCER2, PPP2R3C, GPM3, PLEKHA1, STOML2, LFNG, ELMO1, IFIT3, RPSA, SERINC3, C6orf89, NUB1, GPER1, CLEC4C, TUBB, CD180, RAB27A, GAB2, WASL, HLA-H, TRIM56, PSMB6, SP2, CRK, RBX1, CXCL8, FCGR2A, INS, ZC3HAV1, MAPK3, LPPR2, LTBR, CDC37, WIPF1, BATF, C1QBP, SRPK2, FOS, BCR, CNPY3, TNK2, TCF12, SH2B2, ICOS, NCR3LG1, POLR2L, EXOSC6, SERPING1, IRF5, PPP3CA, AGO1, CCL3, TRIM35, F12, DUSP22, PROS1, TNFRSF10C, TAB2, SPPL2B, OAS1, MBP, ABR, CRCP, CMKLR1, F2RL1, IFITM2, CD97, PAG1, CASP8, ATG12, POLR3F, OAS2, NFKBIB, KLHL7, VNN1, OASL, ZAP70, MUL1, CDC42, GAP, OAS1, ICOSLG, APOBEC3C, ARRB2, LYST, NCAM1, PAK2, ADCY3, TYK2, RNF8, CYFIP2, PVR, DUSP6, CCL21, ANGPT1, UBE2D2, LTF, NR1H2, TRIM21, DHX58, CCR1, LEF1, PELI1, TRPM4, ADARB1, COL4A3BP, APOBEC3A, PAXIP1, STX4, TXN, CTSB, LIG4, MAP2K3, SPNS1, MYO1F, ICAM4, AIM2, SIGLEC7, RAB27A, PTPN1, BIRC3, FOXO3, OASL, POLR3A, PRKAR2A, RNF19B, PSMA4, FOXP1, CXCL2, PSMB10, UNG, FTH1, JAK2, BAX, IP6K2, HFE, CAMK2D, FKBP1A, TNFSF12, FKBP1A, WASF2, IL2RB, SAMSN1, TRAT1, TNFRSF1A, ITGAM, BAD, KLRF1, CBLB, NFATC3, CCL23, THBS1, DEFB1, CSF1R, MT2A, SIGLEC7, CRK, HLX, IL1RAP, IFI6, SNAP23, SEMA4D, ETS1, PSMC4, PIK3C3, PEBP1, IL27RA, TREM1, LST1, CAMP, PPP1R9B, CD27, COLEC12, UBA3, POLR2H, IL1RN, C5AR1, CD81, STAT1, NFKBIB, TREML1, CCR6, STAT2, DHX9, FCRLA, STAT1, JAG1, MAPKAP1, LAT, PSMA1, LCN2, CCM2, MYO1G, IRAK4, PHB, TBX21, CEBPB, SUSP2, CD300A, FES, TRIM68, LILRA6, CD34, CTSL, PTPRA, SRPK2, SEMA3C, HNRNPC, PTPN22, AGO2, LILRB2, CDC42, PHB2, SNX6, CTSC, CTSB, IL6R, FGF23, IFIT5, NLRP3, ANG, PRKCZ, NCF1,

TNFRSF14, PLA2G6, MAP2K6, PANX1, PLD2, TRIM38, LGMN, NCOR2, NLRX1, ITK, TNFRSF10B, IK, PPP2R5D, UBE2D2, TNFRSF21, BAIAP2, RAB27A, IL12RB1, VPREB3, ATP6V0A2, HLA-DOB, CLEC7A, RNF135, GBP1, SH2D1B, EHHADH, TRAF3IP2, IFIT3, CD300C, SNCA, DLG1, FKBP1A, RPS6KA2, CD244, TNFAIP3, SEMA4A, PSMD1, UBE2L6, NEDD4, PSTPIP1, ARHGEF2, AIF1, TNIP1, FCGR3A, RASA2, ADORA2B, PSMA6, PSMD13, MARK3, CLEC10A, ACTG1, RELA, PIK3CA, IL7, BAIAP2, FTH1, TLR4, AGO4, EIF2AK2, FCGR2A, ELANE, MB21D1, MAPK1, PKN2, NFATC1, NFIL3, ARPC4, IFIT1, MICB, DAPK1, ADAM8, IRF1, ABL1, ADM, TAB1, OAS2, MRE11A, MAPK7, RAC2, KCNN4, CD79B, BCL3, APOBEC3F, RC3H2, C2, SLAMF7, IFI16, PRAM1, COCH, CLEC2D, PNKD, MYB, ANKRD17, NLRP3, CARD9, NFKBIB, VAMP2, PRKCB, ABL1, CORO1A, SERINC3, TSC2, PTK2, PSEN2, TNRC6A, RASGRP4, ITGB1, NFKB1, AQP9, CCR7, HLA-DRB1, RPS6KA1, LGALS9, PIK3R4, PTPN22, PAQR3, BCL10, PRKCB, NLRC5, ADAMDEC1, FBXO9, CLSTN1, HLA-DRB3, PROCR, VAV1, PRKCE, CASP9, ATG5, PVRL2, TRIM11, TSPAN32, LST1, CD1D, MSRB1, WDR83, TLR10, CCL2, GMNN, IL25, MAPK14, STX11, SHC1, SAMHD1, EXOSC9, MED1, CARD11, DEFB123, VPS72, CDKN1B, TLR5, BST2, SIRT2, CD1A, MAP4K2, NLRX1, OTUB1, IFI44L, LGALS1, MAP3K14, ORAI1, IL15, SELL, LY96, CD59, PCBP2, ZC3HAV1, TICAM1, RASA1, UBE2D2, MOV10, RFX1, CD1B, CPAMD8, LILRA5, PIK3R2, PSME1, TNRC6B, AKT1S1, IKBKB, CD4, SYNCRIP, CASP1, CFI, KRAS, TNF, PSMD4, CXCL16, FCGR3B, NOTCH1, ZC3HAV1, CHAMP1, RNF216, SRC, TRIM27, KBTBD7, LGALS3, DUSP22, AZU1, MALT1, TLR1, PARP9, GZMH, PTPRJ, KLB, HIST2H2BE, PTK2B, CRHR1, MAPKAPK3, PSMD9, LILRB5, SEMA4D, PSMD8, C1RL, PRKCQ, SPG21, TFEB, IGF2BP1, SPPL2A, ICAM4, GPR65, LILRA1, NBN, CD79A, THEMIS2, ITPR2, POU2F2, CD74, TRIM28, NCKIPSD, OAS2, PAG1, MDM2, PLCL2, NRG1, BCL6, DDX41, PSMD14, MSH2, KYNU, MAPK14, C1QA, FOXP1, PIGU, CDC42, MYD88, PRKAR1A, BAD, PTPN6, TRIM26, LIF, SNX4, FOXO1, RNF135, MNDA, ATG9A, SIRT1, NMI, TNRC6A, CD14, PLCG1, ELMO1, PRF1, MAP3K8, SLC11A1, OSCAR, SERPINB9, BCL2L1, TAPBP, GRB2, MEF2C, CR1, XAF1, CLEC6A, XRCC6, TOX4, ERAP2, VASP, TNFAIP8L2, INPP5D, PSMB5, EP300, CLEC1B, PLSCR1, CXCL9, IFI35, OAS3, IRF9, CX3CR1, POLR2F, IRAK2, NRROS, SARM1, CTNNB1, KYNU, CD6, POLR1C, TRIM8, C5, PTAFR, ITGA4, ZP3, RNF125, PSMB8, LTB4R, MILR1, CD33, ATF2, RELT, PSMB3, GRB2, SNAP23, S100A12, HLA-DPB1, TLR6, ITGAL, CUL1, GPX1, TAP1, CD300E, RBCK1, SKAP1, TNFSF9, ADAM15, STK11, C12orf4, IFNAR1, STAT5A, PRKD2, CSK, IRF4, IKBKE, EIF2AK4, NFATC3, LAMTOR2, IL5RA, ST6GAL1, ADAM15, LY75, CTNNB1,

NCF4, TRAFD1, TSC1, TNFSF13B, PRKRA, CNIH1, FADD, SUPT6H, RNF31, CTSK, RIPK2, DUSP10, TAP2, P2RX7, DPP8, ARPC1A, PSMA5, NAIP, PIK3R1, TSPAN32, ANG, KLHL7, MRE11A, AGO3, RPS6KB2, CD74, HLA-DMB, TNFSF8, UBB, NOD2, PTPRA, HLA-F, EGR1, CAMK2B, STAT6, PIK3CB, RIPK3, TNFRSF1B, IFNGR2, ANKHD1, IFNAR2, HLA-E, S100A13, TOLLIP, IRF2, ADAM17, ZBP1, REL, SNCA, PIK3CD, BPI, PSMB8, SMAD6, SOS1, CR1, POLR1C, PPBP, IL10RB, POLR1D, POLR3B, BIRC2, FOXP1, CD8A, SFTPD, LAIR1, KLHL6, RASGRP1, MTOR, UBE2L6, CCR2, BST1, PIK3CG, RPS6KA3, PNMA1, SLCO1B1, PRKACB, CLEC4E, PRKCA, IL1R2, HLA-DPA1, TLR2, FRS3, ZNF395, CCL8, NFKBIA, CCL3L1, CCL5, CIITA, UNC13D, HRAS, GBP2, CFB, PNKD, CCR2, IL1RN, KCNH8, IL1B, CYLD, NRAS, PIN1, ADAR, CFD, STAT1, C1orf177, CCR2, ITGB7, TAP2, PRKCSH, ELMO2, PTPN11, HLA-B, BTLA, CD44, ENC1, TRIM22, CD40, GZMA, TP53, GSK3B, GAS6, PELI2, CLEC5A, OSM, CD83, TUBB4B, PPP2R5D, SOCS3, MAP2K7, LYN, FYN, IFIH1, PGLYRP4, GAB1, FCRLB, LAMP1, DUSP7, CD19, CD300LB, TAPBP, PDE4B, PIK3R6, EPRS, CD164, TRAF6, TNFSF13, TGFBR3, ELMO1, CCM2, VIMP, CDKN1A, CLEC2B, GNPDA1, S1PR4, GAS6, CD36, FASN, LILRB3, NCF4, SWAP70, SLC26A6, CD58, SOCS5, CD79B, C1QC, POLR3H, GPRC5B, CASP9, CD37, LILRA3, CNIH1, PSME2, ICAM2, ANKRD17, CDKN1A, GPX1, C3AR1, CASP8, TRIB3, C19orf10, CXCL1, UBE2D1, MAPK14, SIT1, GCH1, MLST8, GNL1, ITPR1, FER, MAP2K3, IFNAR2, PDE4D, CXCL10, NFAM1, RASGRP4, AIF1, STX7, CD7, DAPK3, CTSC, TAX1BP1, JAK1, UBA7, VAV2, RASAL3, FGR, DDX60, RNASE1, SIGLEC9, GPER1, LTA, PTGER4, CD36, HIST1H2BK, C1QB, PSMD3, FYB, NCF2, PSEN1, PPP3R1, DDX58, FGF18, ERCC1, TRIM5, LIMK1, TSC1, PRKDC, DUSP3, CASP10, IL6ST, FFAR2, KLRD1, IRF7, LLGL1, PSMB9, CSF2RB, GPR183, PLA2G6, FZD5, NFKB2, KLRD1, DOCK2, PPARG, HMOX1, CD55, PSMD11, PRKCD, BCL2, IFITM1, TNFSF10, POLR3K, APBB1IP, ATF1, APOBEC3G, BTN3A1, CRK, LAT2, MATK, ADA, CAPZA1, VIMP, LGALS3, FCGR2B, SPRED1, PTPRC, SMAGP, MEFV, SIN3A, PSME1, JUN, PRKCB, MIF, IFI30, PADI4, LY86, SNX6, FAS, HLA-DQA1, SBNO2, CLEC4D, PSMC3, PSEN1, XBP1, CREBBP, CAMK2G, AGO3, ARPC2, PDPK1, RELB, PML, NRG1, VASP, ICAM1, SHC1, GCH1, TNFRSF10B, CTSL, DUSP22, HIST1H2BK, TNFRSF9, IL16, CNR1, TRIM25, NR1H3, PSMB7, TICAM1, ITPR3, SERINC3, PLCG2, GAB2, IL12RB1, TUBB2A, F2RL1, PPP3CA, KBTBD7, PARP9, ISG15, SUGT1, PAG1, KLRF1, PSMA2, IFI27, SYK, FTH1, PTK6, KLRB1, IRS2, PHLPP2, ARPC1B, HLX, CX3CR1, TNFSF4, IRF2, SPTAN1, PSMC6, CCL5, DHX36, MAP2K4, MAP2K4, ADAMDEC1, TNFSF15, CD300LF, RNASE3, NOD1, GBP5, RIPK1, ADAM17, ETS1, ERAP2, FCER1G, REL, SKAP2, MAVS, FCGR3B0,

	<p>CD58, GBP1, ADCY4, SOCS3, GBF1, STXBP2, HLA-DRB4, HLA-A, XRCC6, MR1, GPR183, TNIP2, LAMP3, SAMSN1, CXCL5, PNP, IKBKB, GPI, PRELID1, LIME1, ANXA1, OAS3, CXCL8, VAMP8, CLEC4G, SLAMF6, CDKN1B, POLR3A, VIPR1, TREML4, C10orf54, RNASEL, IKBKAP, ICAM3, LEF1, PPP2R3C, HMGB2, RABGEF1, RABGEF1, HMGB1, MX2, GPR65, MYO10, ECSIT, CD38, UBE2D3, TSC1, BCL2, LAT2, FYN, FOXP1, LGMN, PRKCZ, PRKDC, TRIM34, AGER, NAIP, CD3D, CACTIN, LILRA5, MAPKAP1, IL15, CCR2, YWHAB, ERCC1, FYB, PSMB8, SP100, PSMC4, VAV3, TIRAP, COL4A3BP, TANK, SLC26A6, CREBBP, ANG, ANG, PTPN2, PTPN2, APOBEC3F, CD46, NLRP3, IRF5, EXOSC9, DPP8, NLRP1, ELMO2, PSMA3, MATK, FAS, CAMK2D, ADAR, BAX, TRIM4, CD3D, LAT2, IP6K2, ECM1, RAB27A, CNIH1, PTK2B, TNFRSF10B, PRKD2, MBP, RNASE1, PRKDC, CREB1, CREB1, IL18BP, NCF4, CD1E, GCH1, PVRL2, CXCR5, LILRB5, MAPK7, NCOR2, PTPRC, PDE4B, PSMD13, IL7R, PSMD4, PTGES2, PTGES2, LST1, IFI6, CACTIN, SOCS5, WIPF1, KL, LGALS8, CD8A, CYFIP2, OTUB1, CYFIP1, CYFIP1, LILRB4, LGALS8, IL1RAP, LILRA5, CAMK2G, PPP2R5D, MAPKAP1, MAPK8, FCAR, CD79B, OSCAR, CD40, FGR, IL32, UBA52, IL15, XAF1, TANK, LIG4, BTN3A3, CTSL, PRKACB, TAX1BP1, CD37, LTB, LTB, CCR2, ERCC1, CASP8, MAP3K7, CD74, SUMO1, FCAR, SMAGP, FYN, CRCP, FCGR2B, CREB1, TRAF3, ITGB1, GPER1, NFATC1, ST6GAL1, ADAM15, PRKCZ, CCR6, DEAF1, MALT1, PDPK1, MAPK14, AKT1, ACTR2, TSPAN32, PSMB8, SP100, CTSH, NFKB2, SLC26A6, TRAF6, ICAM4, ARPC4, PLEKHA1, FBXW11, PRKACA, PSMC4, ASS1, ARRB2, RASGRP4, DUSP6, HCST, KDM6B, PYCARD, PML, SIRT2, TNFSF13, CLEC4A, AP1G1, AP1G1, PTPN2, MAPK9, PRKCSH, MS4A1, DUSP10, DUSP10, MAPK3, APP, APP, PSEN2, LAT, NOTCH2, BIRC3, RBCK1, ANKRD17, SPTBN4, S100A13, RNF135, IL24, OAS1, AKT1, ATG9A, PTPRA, APOBEC3F, LGALS9, CD97, TLR10, CLSTN1, CLEC10A, CD96, IP6K2, RPS6, PRKCB, LAMTOR3, CNRIP1, MUL1, GPR183, RASAL3, POLR2E, IDO1, USP18, GAPT, SIGLEC14, NDUFA2, PIK3R6, RAPGEF2, FCGR1C, MED1, PCBP2, PCBP2, FTH1, IP6K2, PRKCB, LAMTOR3, APOBEC3D, PGLYRP2, ADRBK1, BTF3P11</p>
Inflammatory Response	<p>TPST1, IL4R, CCM2, PIK3AP1, HMGB2, CLEC7A, ABR, STAB1, IDO1, RPS6KA5, MGLL, CFH, AHCY, KLRG1, DEAF1, TNFRSF8, ADRA2A, SPN, STAT3, RPSA, PTGS1, GPER1, CD180, CXCL8, INS, HYAL3, LTBR, C1QBP, C5AR2, FOS, BCR, SERPING1, POLG2, CCL3, F12, MTA1, PROS1, ABR, F2RL1, CD97, HRH1, TFRC, VNN1, SLIT2, NR1D2, CCL21, PTGS2, CHST1, CCR1, GART, CXCR2, MAP2K3, ALOX5, SPNS1, HIF1A, AIM2, BIRC3, FOXP1, CXCL2, IER3, JAK2, HFE, TNFRSF1A, ITGAM, NFATC3, CCL23, THBS1, DEFB1, CSF1R,</p>

IL1RAP, RXRA, DHX8, SNAP23, ETS1, CTNNBIP1, ZNF580, CAMP, PPP1R9B, CD27, IL1RN, C5AR1, LTA4H, CXXC1, LAT, CCM2, PHB, CEBPB, HNRNPC, ADRB2, PHB2, SNX6, GHRL, IL6R, KLK1, ORM1, NLRP3, PRKCZ, NCF1, TNFRSF14, NFX1, NR1D2, NLRX1, TNFRSF21, CLEC7A, PLA2G7, EHHADH, ZYX, TNFAIP3, PSTPIP1, FEM1C, S1PR3, AIF1, TNIP1, ADORA2B, PSMA6, SEH1L, RELA, TLR4, EIF2AK2, ELANE, ADAM8, SEH1L, MAPK7, AOA, C2, IFI16, NLRP3, VAMP2, NFKB1, ZNF580, CCR7, HLA-DRB1, CEBPA, LGALS9, NLRP12, CLSTN1, LIPA, NFRKB, NFKBIZ, TLR10, FEM1A, CCL2, IL25, MAPK14, ZFP36, CASP5, CD163, TLR5, OLR1, NLRX1, LXN, SETD6, IL15, LY96, CD59, TICAM1, IKBKB, CASP1, CFI, TNF, IL17RA, NOTCH1, CHAMP1, AZU1, TLR1, CD163, PRKCQ, THEMIS2, LIAS, SIGIRR, BRD4, BCL6, MAPK14, FOXP1, MYD88, MGLL, SNX4, NMI, NLRP12, CD14, FPR2, SLC11A1, CR1, NFX1, PLSCR1, CXCL9, CX3CR1, FOLR2, IRAK2, NRROS, CD6, C5, PTAFR, ZP3, ADORA1, LTB4R, RELT, SNAP23, S100A12, TLR6, MAPK13, ITGAL, GPX1, CAMK1D, MCPH1, HNRNPA0, STAT5A, NFATC3, RPS6KA4, APOL3, LY75, CALCRL, P2RX1, NLRP12, RIPK2, DUSP10, P2RX7, NAIP, ACVR1, ATRN, NOD2, HIF1A, NFKBID, ABCF1, XCR1, TNFRSF1B, TOLLIP, REL, B4GALT1, PIK3CD, CR1, PPBP, IL10RB, PRDX2, POLB, BIRC2, FOXP1, RASGRP1, ELF3, PLAA, CCR2, NFKBIZ, PIK3CG, PNMA1, BRD4, CD47, PRKCA, ATRN, TLR2, CCL8, CCL3L1, CCL5, CIITA, UNC13D, CFB, CCR2, IL1RN, PROK2, LTB4R2, IL1B, CCR2, CASP4, MMP24, CD44, CD40, KLF4, OSM, SOCS3, LYN, HDAC9, ASH1L, CCM2, VIMP, FASN, TNFAIP6, GPRC5B, NOV, GPX1, C3AR1, C19orf10, CXCL1, MAPK14, IL17C, MAS1, METRNL, MAP2K3, NFE2L2, STK39, CXCL10, NFAM1, AIF1, GART, CHST2, RNASE1, GPER1, LTA, CAMK1D, PTGER4, IL6ST, ALOX5AP, FFAR2, IL17RC, NFKB2, PPARG, HMOX1, CD55, PRKCD, CXCR4, METRNL, HDAC9, ADA, VIMP, TUSC2, NFX1, MEFV, PGAP3, CELA1, MIF, NDST1, ADORA2A, LY86, SNX6, SIGIRR, FAS, KCNJ10, SBNO2, TSPAN2, PLA2G4C, RELB, CSRP1, ICAM1, HP, TNFRSF9, CNR1, NR1H3, PRDX5, TICAM1, SDC1, F2RL1, NPY5R, SYK, ACP5, UCN, CX3CR1, TNFSF4, CCL5, NOD1, GBP5, ETS1, FCER1G, REL, KLF4, SOCS3, LYZ, TNIP2, CXCL5, IKBKB, ANXA1, CXCL8, TSPAN2, VAMP8, HMGB2, TBC1D23, RABGEF1, RABGEF1, HMGB1, FOXP1, PRKCZ, SERPINA1, AGER, METRNL, NAIP, IL15, CCR2, SP100, TIRAP, PTPN2, PTPN2, CD46, NLRP3, NLRP1, FAS, CXCR4, APOL2, APOL2, ECM1, RNASE1, MAPK7, PTGS1, MEN1, F11R, METRNL, KL, ATRN, IL1RAP, GBA, PRCP, CD40, IL15, ZYX, CCR2, CD163, HIF1A, SEH1L, PRDX2, GPER1, PRKCZ, DEAF1, MAPK14, AKT1, SP100, NFKB2, FPR2, ABCF1, LIAS, ASS1, KDM6B,

	PYCARD, PTPN2, MS4A1, DUSP10, DUSP10, STAT3, LAT, BIRC3, F11R, IL24, HDAC9, AKT1, STAT3, LGALS9, CD97, TLR10, CLSTN1, IDO1, NDUFA2, EIF2AK1, PGLYRP2
Regulation of Inflammatory Response	CCM2 , PIK3AP1, ABR, IDO1, MGLL, CFH, DEAF1, SPN, RPSA, GPER1, INS, C1QBP, BCR, SERPING1, CCL3, F12, MTA1, PROS1, ABR, SLIT2, NR1D2, PTGS2, BIRC3, FOXP1, IER3, JAK2, TNFRSF1A, CCL23, ETS1, PPP1R9B, CCM2, PHB, HNRNPC, ADRB2, PHB2, SNX6, GHRL, NLRP3, NR1D2, NLRX1, PLA2G7, EHHADH, ZYX, TNFAIP3, FEM1C, TNIP1, ADORA2B, PSMA6, RELA, TLR4, ELANE, ADAM8, MAPK7, AOA, C2, NLRP3, VAMP2, NFKB1, CCR7, HLA-DRB1, NLRP12, TLR10, FEM1A, MAPK14, ZFP36, CASP5, NLRX1, SETD6, IL15, CD59, CASP1, CFI, TNF, IL17RA, BRD4, BCL6, MAPK14, FOXP1, MYD88, MGLL, SNX4, NLRP12, CR1, TNFAIP8L2, CX3CR1, CD6, C5, ZP3, ADORA1, S100A12, MAPK13, GPX1, MCPH1, STAT5A, CALCRL, NLRP12, DUSP10, NOD2, TNFRSF1B, CR1, BIRC2, FOXP1, CCR2, PIK3CG, BRD4, CD47, PRKCA, TLR2, CCL3L1, CCL5, CFB, CCR2, IL1B, CCR2, CASP4, KLF4, OSM, SOCS3, LYN, ASH1L, CCM2, VIMP, TNFAIP6, GPRC5B, NOV, GPX1, MAPK14, MAS1, METRNL, STK39, CXCL10, GPER1, LTA, PTGER4, IL6ST, ALOX5AP, FFAR2, IL17RC, PPARG, CD55, PRKCD, METRNL, ADA, VIMP, MEFV, PGAP3, ADORA2A, SNX6, SBNO2, CNR1, NR1H3, NPY5R, ACP5, CX3CR1, TNFSF4, CCL5, GBP5, ETS1, FCER1G, KLF4, SOCS3, ANXA1, VAMP8, TBC1D23, RABGEF1, RABGEF1, FOXP1, AGER, METRNL, IL15, CCR2, PTPN2, PTPN2, CD46, NLRP3, NLRP1, MAPK7, METRNL, GBA, IL15, ZYX, CCR2, GPER1, DEAF1, MAPK14, PTPN2, DUSP10, DUSP10, BIRC3, TLR10, IDO1, PGLYRP2

Appendix 18

Overlapping Genes Between Investigated Gene Ontology Biological Processes		
Chronic Inflammation		
	Immune Response	IDO1, AHCY, VNN1, THBS1, CAMP, IL1RN, CEBPB, TNFAIP3, ADORA2B, TNF, CHAMP1, CX3CR1, CCL5, UNC13D, IL1RN, IL1B, LTA, CX3CR1, CCL5, IDO1
	Inflammatory Response	IDO1, AHCY, VNN1, THBS1, CAMP, IL1RN, CEBPB, TNFAIP3, ADORA2B, TNF, CHAMP1, CX3CR1, CCL5, UNC13D, IL1RN, IL1B, LTA, CX3CR1, CCL5, IDO1
	Regulation of the Inflammatory Response	IDO1, TNFAIP3, ADORA2B, TNF, CX3CR1, CCL5, IL1B, LTA, CX3CR1, CCL5, IDO1
Immune Response		
	Inflammatory Response	IL4R, CCM2, PIK3AP1, HMGB2, CLEC7A, ABR, IDO1, RPS6KA5, CFH, AHCY, KLRG1, DEAF1, TNFRSF8, SPN, RPSA, GPER1, CD180, CXCL8, INS, LTBR, C1QBP, FOS, BCR, SERPING1, CCL3, F12, PROS1, ABR, F2RL1, CD97, VNN1, CCL21, CCR1, MAP2K3, SPNS1, AIM2, BIRC3, FOXP1, CXCL2, JAK2, HFE, TNFRSF1A, ITGAM, NFATC3, CCL23, THBS1, DEFB1, CSF1R, IL1RAP, SNAP23, ETS1, CAMP, PPP1R9B, CD27, IL1RN, C5AR1, LAT, CCM2,

		<p>PHB, CEBPB, HNRNPC, PHB2, SNX6, IL6R, NLRP3, PRKCZ, NCF1, TNFRSF14, NLRX1, TNFRSF21, CLEC7A, EHHADH, TNFAIP3, PSTPIP1, AIF1, TNIP1, ADORA2B, PSMA6, RELA, TLR4, EIF2AK2, ELANE, ADAM8, MAPK7, C2, IFI16, NLRP3, VAMP2, NFKB1, CCR7, HLA-DRB1, LGALS9, CLSTN1, TLR10, CCL2, IL25, MAPK14, TLR5, NLRX1, IL15, LY96, CD59, TICAM1, IKBKB, CASP1, CFI, TNF, NOTCH1, CHAMP1, AZU1, TLR1, PRKCQ, THEMIS2, BCL6, MAPK14, FOXP1, MYD88, SNX4, NMI, CD14, SLC11A1, CR1, PLSCR1, CXCL9, CX3CR1, IRAK2, NRROS, CD6, C5, PTAFR, ZP3, LTB4R, RELT, SNAP23, S100A12, TLR6, ITGAL, GPX1, STAT5A, NFATC3, LY75, RIPK2, DUSP10, P2RX7, NAIP, NOD2, TNFRSF1B, TOLLIP, REL, PIK3CD, CR1, PPBP, IL10RB, BIRC2, FOXP1, RASGRP1, CCR2, PIK3CG, PNMA1, PRKCA, TLR2, CCL8, CCL3L1, CCL5, CIITA, UNC13D, CFB, CCR2, IL1RN, IL1B, CCR2, CD44, CD40, OSM, SOCS3, LYN, CCM2, VIMP, FASN, GPRC5B, GPX1, C3AR1, C19orf10, CXCL1, MAPK14, MAP2K3, CXCL10, NFAM1, AIF1, RNASE1, GPER1, LTA, PTGER4, IL6ST, FFAR2, NFKB2, PPARG,</p>
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		<p>HMOX1, CD55, PRKCD, ADA, VIMP, MEFV, MIF, LY86, SNX6, FAS, SBNO2, RELB, ICAM1, TNFRSF9, CNR1, NR1H3, TICAM1, F2RL1, SYK, CX3CR1, TNFSF4, CCL5, NOD1, GBP5, ETS1, FCER1G, REL, SOCS3, TNIP2, CXCL5, IKBKB, ANXA1, CXCL8, VAMP8, HMGB2, RABGEF1, RABGEF1, HMGB1, FOXP1, PRKCZ, AGER, NAIP, IL15, CCR2, SP100, TIRAP, PTPN2, PTPN2, CD46, NLRP3, NLRP1, FAS, ECM1, RNASE1, MAPK7, KL, IL1RAP, CD40, IL15, CCR2, GPER1, PRKCZ, DEAF1, MAPK14, AKT1, SP100, NFKB2, ASS1, KDM6B, PYCARD, PTPN2, MS4A1, DUSP10, DUSP10, LAT, BIRC3, IL24, AKT1, LGALS9, CD97, TLR10, CLSTN1, IDO1, NDUFA2, PGLYRP2</p>
	<p>Regulation of the Inflammatory Response</p>	<p>CCM2, PIK3AP1, ABR, IDO1, CFH, DEAF1, SPN, RPSA, GPER1, INS, C1QBP, BCR, SERPING1, CCL3, F12, PROS1, ABR, BIRC3, FOXP1, JAK2, TNFRSF1A, CCL23, ETS1, PPP1R9B, CCM2, PHB, HNRNPC, PHB2, SNX6, NLRP3, NLRX1, EHHADH, TNFAIP3, TNIP1, ADORA2B, PSMA6, RELA, TLR4, ELANE, ADAM8, MAPK7, C2, NLRP3, VAMP2, NFKB1, CCR7, HLA-DRB1, TLR10, MAPK14, NLRX1, IL15, CD59, CASP1, CFI, TNF, BCL6,</p>

		<p>MAPK14, FOXP1, MYD88, SNX4, CR1, TNFAIP8L2, CX3CR1, CD6, C5, ZP3, S100A12, GPX1, STAT5A, DUSP10, NOD2, TNFRSF1B, CR1, BIRC2, FOXP1, CCR2, PIK3CG, PRKCA, TLR2, CCL3L1, CCL5, CFB, CCR2, IL1B, CCR2, OSM, SOCS3, LYN, CCM2, VIMP, GPRC5B, GPX1, MAPK14, CXCL10, GPER1, LTA, PTGER4, IL6ST, FFAR2, PPARG, CD55, PRKCD, ADA, VIMP, MEFV, SNX6, SBNO2, CNR1, NR1H3, CX3CR1, TNFSF4, CCL5, GBP5, ETS1, FCER1G, SOCS3, ANXA1, VAMP8, RABGEF1, RABGEF1, FOXP1, AGER, IL15, CCR2, PTPN2, PTPN2, CD46, NLRP3, NLRP1, MAPK7, IL15, CCR2, GPER1, DEAF1, MAPK14, PTPN2, DUSP10, DUSP10, BIRC3, TLR10, IDO1, PGLYRP2</p>
Inflammatory Response		
	Regulation of the Inflammatory Response	<p>CCM2, PIK3AP1, ABR, IDO1, MGLL, CFH, DEAF1, SPN, RPSA, GPER1, INS, C1QBP, BCR, SERPING1, CCL3, F12, MTA1, PROS1, ABR, SLIT2, NR1D2, PTGS2, BIRC3, FOXP1, IER3, JAK2, TNFRSF1A, CCL23, ETS1, PPP1R9B, CCM2, PHB, HNRNPC, ADRB2, PHB2, SNX6, GHRL, NLRP3, NR1D2, NLRX1, PLA2G7, EHHADH, ZYX, TNFAIP3, FEM1C, TNIP1, ADORA2B, PSMA6, RELA, TLR4, ELANE, ADAM8,</p>

		<p>MAPK7, AOA1, C2, NLRP3, VAMP2, NFKB1, CCR7, HLA-DRB1, NLRP12, TLR10, FEM1A, MAPK14, ZFP36, CASP5, NLRX1, SETD6, IL15, CD59, CASP1, CFI, TNF, IL17RA, BRD4, BCL6, MAPK14, FOXP1, MYD88, MGLL, SNX4, NLRP12, CR1, CX3CR1, CD6, C5, ZP3, ADORA1, S100A12, MAPK13, GPX1, MCPH1, STAT5A, CALCRL, NLRP12, DUSP10, NOD2, TNFRSF1B, CR1, BIRC2, FOXP1, CCR2, PIK3CG, BRD4, CD47, PRKCA, TLR2, CCL3L1, CCL5, CFB, CCR2, IL1B, CCR2, CASP4, KLF4, OSM, SOCS3, LYN, ASH1L, CCM2, VIMP, TNFAIP6, GPRC5B, NOV, GPX1, MAPK14, MAS1, METRNL, STK39, CXCL10, GPER1, LTA, PTGER4, IL6ST, ALOX5AP, FFAR2, IL17RC, PPARG, CD55, PRKCD, METRNL, ADA, VIMP, MEFV, PGAP3, ADORA2A, SNX6, SBNO2, CNR1, NR1H3, NPY5R, ACP5, CX3CR1, TNFSF4, CCL5, GBP5, ETS1, FCER1G, KLF4, SOCS3, ANXA1, VAMP8, TBC1D23, RABGEF1, RABGEF1, FOXP1, AGER, METRNL, IL15, CCR2, PTPN2, PTPN2, CD46, NLRP3, NLRP1, MAPK7, METRNL, GBA, IL15, ZYX, CCR2, GPER1, DEAF1, MAPK14, PTPN2, DUSP10, DUSP10, BIRC3, TLR10, IDO1, PGLYRP2</p>
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Appendix 19

List of Transcripts (Genes) with p<0.05 in Aim 2 Linear Regression Analyses by Social Environmental Factor and Biological Process									
Major or Lifetime Discrimination and Chronic Inflammation Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination and Immune Response Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination and Inflammatory Response Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination and Regulation of the Inflammatory Response Illumina Transcript ID # & Gene Symbol		Loneliness and Chronic Inflammation Illumina Transcript ID # & Gene Symbol	
ILMN_1674 574	VNN1	ILMN_1696 488	FGF23	ILMN_16521 98	CCM2	ILMN_16521 98	CCM2	ILMN_1674 574	VNN1
ILMN_1729 976	CHAMP1	ILMN_1699 727	BAIAP2	ILMN_16603 15	SPN	ILMN_16603 15	SPN	ILMN_1703 946	ADORA2B
ILMN_1745 788	CX3CR1	ILMN_1699 908	IL12RB1	ILMN_16656 47	CD180	ILMN_16715 09	CCL3	ILMN_1745 788	CX3CR1
ILMN_2088 437	CX3CR1	ILMN_1701 507	EHHADH	ILMN_16667 33	CXCL8	ILMN_16728 78	ABR	ILMN_1795 464	LTA
		ILMN_1702 501	RPS6KA2	ILMN_16695 23	FOS	ILMN_16768 44	NR1D2	ILMN_2088 437	CX3CR1
		ILMN_1702 787	SEMA4A	ILMN_16715 09	CCL3	ILMN_16827 17	IER3		
		ILMN_1703 477	ARHGAP2	ILMN_16728 78	ABR	ILMN_16963 80	GHRL		
		ILMN_1703 650	TNIP1	ILMN_16731 13	F2RL1	ILMN_16986 68	NR1D2		
		ILMN_1704 961	ACTG1	ILMN_16745 74	VNN1	ILMN_17015 07	EHHA1		
		ILMN_1707 312	NFIL3	ILMN_16768 44	NR1D2	ILMN_17036 50	TNIP1		

		ILMN_1710 514	BCL3	ILMN_16827 17	IER3	ILMN_17457 88	CX3CR 1		
		ILMN_1713 732	ABL1	ILMN_16866 23	CSF1R	ILMN_17489 15	S100A1 2		
		ILMN_1713 749	CORO1 A	ILMN_16963 80	GHRL	ILMN_17647 88	TNFRS F1B		
		ILMN_1715 068	AQP9	ILMN_16984 02	NFX1	ILMN_17681 94	BIRC2		
		ILMN_1715 885	PTPN22	ILMN_16986 68	NR1D2	ILMN_17732 45	CCL3L 1		
		ILMN_1716 071	PAQR3	ILMN_17015 07	EHHA DH	ILMN_17805 46	OSM		
		ILMN_1718 070	CASP9	ILMN_17036 50	TNIP1	ILMN_17810 01	SOCS3		
		ILMN_1718 265	ATG5	ILMN_17157 15	CEBP A	ILMN_17811 55	LYN		
		ILMN_1718 621	TSPAN3 2	ILMN_17230 35	OLR1	ILMN_17913 28	STK39		
		ILMN_1723 625	MAP4K 2	ILMN_17291 61	NOTC H1	ILMN_17987 90	IL17RC		
		ILMN_1726 565	PIK3R2	ILMN_17299 76	CHAM P1	ILMN_18005 40	CD55		
		ILMN_1729 161	NOTCH 1	ILMN_17457 88	CX3C R1	ILMN_18011 05	PRKCD		
		ILMN_1729 976	CHAMP 1	ILMN_17472 51	LTB4R	ILMN_20884 37	CX3CR 1		
		ILMN_1730 986	MALT1	ILMN_17489 15	S100A 12	ILMN_22305 79	RABGE F1		
		ILMN_1731 233	GZMH	ILMN_17495 91	ITGAL	ILMN_23077 44	CD46		
		ILMN_1732 071	HIST2H 2B	ILMN_17532 79	HNRN PA0	ILMN_23130 79	NLRP1		

		ILMN_1738 675	PTPN6	ILMN_17589 39	RIPK2				
		ILMN_1745 788	CX3CR1	ILMN_17647 88	TNFRS F1B				
		ILMN_1746 704	TRIM8	ILMN_17662 75	PIK3C D				
		ILMN_1747 251	LTB4R	ILMN_17681 94	BIRC2				
		ILMN_1748 797	GRB2	ILMN_17721 24	ATRNL				
		ILMN_1748 915	S100A12	ILMN_17732 45	CCL3L 1				
		ILMN_1749 591	ITGAL	ILMN_17805 46	OSM				
		ILMN_1751 095	CD300E	ILMN_17810 01	SOCS3				
		ILMN_1754 121	CSK	ILMN_17811 55	LYN				
		ILMN_1754 507	IRF4	ILMN_17848 71	FASN				
		ILMN_1756 920	ADAM1 5	ILMN_17905 34	MAP2 K3				
		ILMN_1758 474	PRKRA	ILMN_17913 28	STK39				
		ILMN_1758 939	RIPK2	ILMN_17987 90	IL17R C				
		ILMN_1760 303	PIK3R1	ILMN_17990 62	NFKB2				
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		ILMN_1761 049	AGO3	ILMN_18011 05	PRKC D				

		ILMN_1762 766	PTPRA	ILMN_18046 10	NFX1				
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		ILMN_1768 194	BIRC2	ILMN_21843 73	CXCL8				
		ILMN_1768 598	LAIR1	ILMN_22305 79	RABG EF1				
		ILMN_1772 876	ZNF395	ILMN_23077 44	CD46				
		ILMN_1773 245	CCL3L1	ILMN_23130 79	NLRP1				
		ILMN_1777 519	ITGB7	ILMN_23908 59	NFKB2				
		ILMN_1777 794	PRKCS H	ILMN_23926 35	ABCF1				
		ILMN_1779 324	GZMA	ILMN_24122 14	LGAL S9				
		ILMN_1779 558	GAS6						
		ILMN_1780 465	CLEC5A						
		ILMN_1780 546	OSM						
		ILMN_1781 001	SOCS3						
		ILMN_1781 155	LYN						

		ILMN_1781 207	FYN						
		ILMN_1782 292	LAMP1						
		ILMN_1783 695	EPRS						
		ILMN_1784 871	FASN						
		ILMN_1785 175	SWAP70						
		ILMN_1786 024	POLR3H						
		ILMN_1790 534	MAP2K 3						
		ILMN_1798 706	GPR183						
		ILMN_1799 062	NFKB2						
		ILMN_1799 725	DOCK2						
		ILMN_1800 540	CD55						
		ILMN_1801 105	PRKCD						
		ILMN_1804 277	SPRED1						
		ILMN_1804 279	PTPRC						
		ILMN_1805 996	SIN3A						
		ILMN_1806 023	JUN						

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		ILMN_2055 781	KLRF1						
		ILMN_2079 655	KLRB1						
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		ILMN_2184 373	CXCL8						
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		ILMN_2230 579	RABGE F1						
		ILMN_2246 510	TSC1						
		ILMN_2307 744	CD46						
		ILMN_2313 079	NLRP1						
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		ILMN_2350 970	SOCS5						

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		ILMN_2399 622	AP1G1						
		ILMN_2404 063	APP						
		ILMN_2405 297	NOTCH 2						
		ILMN_2411 794	PTPRA						
		ILMN_2412 214	LGALS9						
		ILMN_3238 803	RASAL3						
		ILMN_3301 197	LAMTO R3						
		ILMN_3307 926	ADRBK 1						

Appendix 20

List of Transcripts (Genes) Selected via Elastic Net in Aim 2 Analyses by Social Environmental Factor and Biological Process											
Major or Lifetime Discrimination and Chronic Inflammation Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination and Immune Response Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination and Inflammatory Response Illumina Transcript ID # & Gene Symbol		Major or Lifetime Discrimination and Regulation of the Inflammatory Response Illumina Transcript ID # & Gene Symbol		Loneliness and Chronic Inflammation Illumina Transcript ID # & Gene Symbol		Chronic Burden and Chronic Inflammation Illumina Transcript ID # & Gene Symbol	
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ILMN_17 29976	CHAMP1	ILMN_16 53166	CLEC10A	ILMN_16 60315	SPN	ILMN_16 60315	SPN	ILMN_17 03946	ADORA2B	ILMN_16 86116	THBS1
ILMN_17 45788	CX3CR1	ILMN_16 53652	PTPRC	ILMN_16 65647	CD180	ILMN_16 71509	CCL3	ILMN_17 45788	CX3CR1	ILMN_16 89734	IL1RN
ILMN_20 88437	CX3CR1	ILMN_16 54586	RASA3	ILMN_16 66733	CXCL8	ILMN_16 72878	ABR	ILMN_17 95464	LTA	ILMN_17 03946	ADORA2B
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		ILMN_16 58636	MRE11A	ILMN_16 71509	CCL3	ILMN_16 82717	IER3			ILMN_20 88437	CX3CR1
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		ILMN_16 61695	IRAK3	ILMN_16 74574	VNN1	ILMN_17 01507	EHHA1			ILMN_16 74574	VNN1
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		ILMN_16 69523	FOS	ILMN_16 96380	GHRL	ILMN_17 64788	TNFR SF1B				
		ILMN_16 69832	TCF12	ILMN_16 98402	NFX1	ILMN_17 68194	BIRC2				
		ILMN_16 71509	CCL3	ILMN_16 98668	NR1D 2	ILMN_17 73245	CCL3L 1				
		ILMN_16 71809	DUSP2 2	ILMN_17 01507	EHHA DH	ILMN_17 80546	OSM				
		ILMN_16 72660	MBP	ILMN_17 03650	TNIP1	ILMN_17 81001	SOCS3				
		ILMN_16 72878	ABR	ILMN_17 15715	CEBP A	ILMN_17 81155	LYN				
		ILMN_16 73113	F2RL1	ILMN_17 23035	OLR1	ILMN_17 91328	STK39				
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		ILMN_16 76385	PAK2	ILMN_17 45788	CX3C R1	ILMN_18 01105	PRKC D				
		ILMN_16 77466	DUSP6	ILMN_17 47251	LTB4R	ILMN_20 88437	CX3C R1				
		ILMN_16 81591	PTPN1	ILMN_17 48915	S100A 12	ILMN_22 30579	RABG EF1				
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		ILMN_16 99908	IL12R B1	ILMN_17 84871	FASN						
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		ILMN_17 03650	TNIP1	ILMN_18 00540	CD55						
		ILMN_17 04961	ACTG 1	ILMN_18 01105	PRKC D						

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		ILMN_17 13732	ABL1	ILMN_18 10093	TSPA N2						
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		ILMN_17 29161	NOTC H1								
		ILMN_17 29976	CHAM P1								
		ILMN_17 30986	MALT 1								
		ILMN_17 31233	GZMH								

		ILMN_17 32071	HIST2 H2B								
		ILMN_17 38675	PTPN6								
		ILMN_17 45788	CX3C R1								
		ILMN_17 46704	TRIM8								
		ILMN_17 47251	LTB4R								
		ILMN_17 48797	GRB2								
		ILMN_17 48915	S100A 12								
		ILMN_17 49591	ITGAL								
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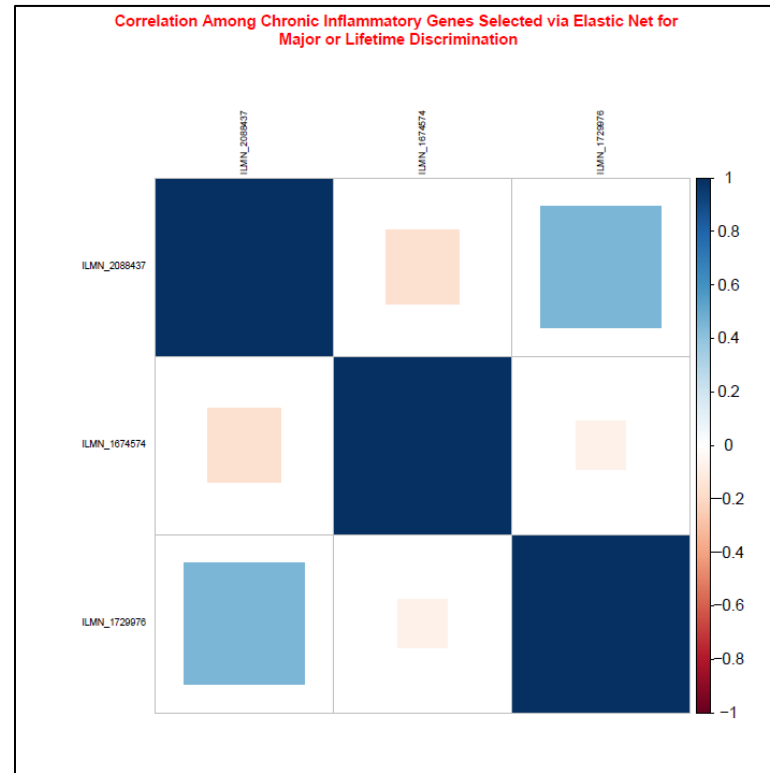
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		ILMN_17 68598	LAIR1								
		ILMN_17 72876	ZNF39 5								
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		ILMN_17 77519	ITGB7								
		ILMN_17 77794	PRKC SH								
		ILMN_17 79324	GZMA								
		ILMN_17 79558	GAS6								
		ILMN_17 80465	CLEC5 A								
		ILMN_17 80546	OSM								
		ILMN_17 81001	SOCS3								

		ILMN_17 81155	LYN								
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		ILMN_17 83695	EPRS								
		ILMN_17 84871	FASN								
		ILMN_17 85175	SWAP 70								
		ILMN_17 86024	POLR3 H								
		ILMN_17 90534	MAP2 K3								
		ILMN_17 98706	GPR18 3								
		ILMN_17 99062	NFKB 2								
		ILMN_17 99725	DOCK 2								
		ILMN_18 00540	CD55								
		ILMN_18 01105	PRKC D								
		ILMN_18 04277	SPRE D1								
		ILMN_18 04279	PTPRC								
		ILMN_18 05996	SIN3A								

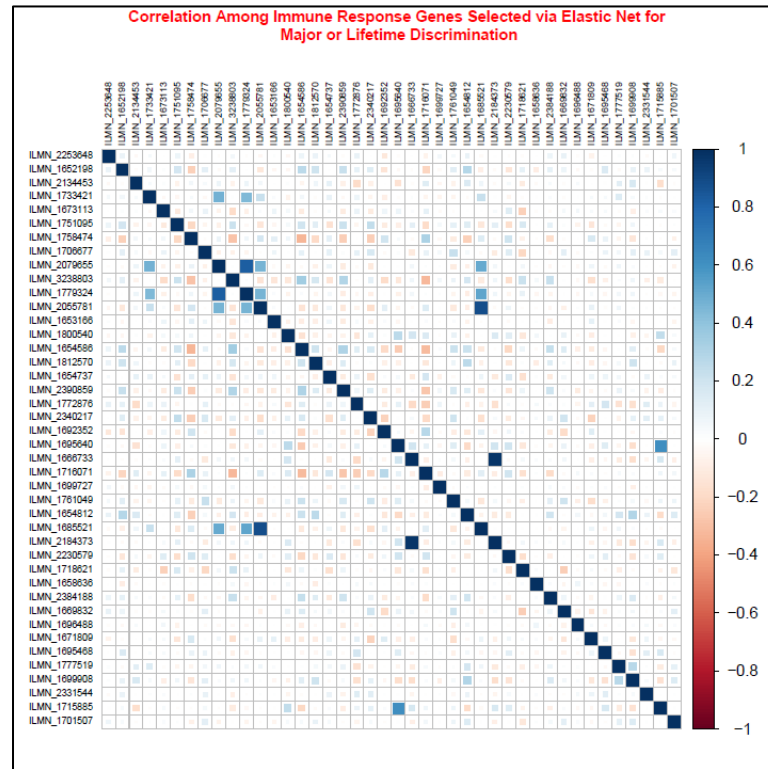
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		ILMN_18 07277	IFI30								
		ILMN_18 08979	CLEC4 D								
		ILMN_18 12001	VASP								
		ILMN_20 55781	KLRF1								
		ILMN_20 79655	KLRB 1								
		ILMN_20 88437	CX3C R1								
		ILMN_21 84373	CXCL 8								
		ILMN_22 11189	IKBK AP								
		ILMN_22 30579	RABG EF1								
		ILMN_22 46510	TSC1								
		ILMN_23 07744	CD46								
		ILMN_23 13079	NLRP1								
		ILMN_23 31544	MBP								
		ILMN_23 40217	PTPRC								
		ILMN_23 40259	PDE4B								

		ILMN_23 50970	SOCS5								
		ILMN_23 65091	FCAR								
		ILMN_23 79967	FCAR								
		ILMN_23 83934	ITGB1								
		ILMN_23 84188	NFAT C1								
		ILMN_23 90859	NFKB 2								
		ILMN_23 99622	AP1G1								
		ILMN_24 04063	APP								
		ILMN_24 05297	NOTC H2								
		ILMN_24 11794	PTPR A								
		ILMN_24 12214	LGAL S9								
		ILMN_32 38803	RASA L3								
		ILMN_33 01197	LAMT OR3								
		ILMN_33 07926	ADRB K1								

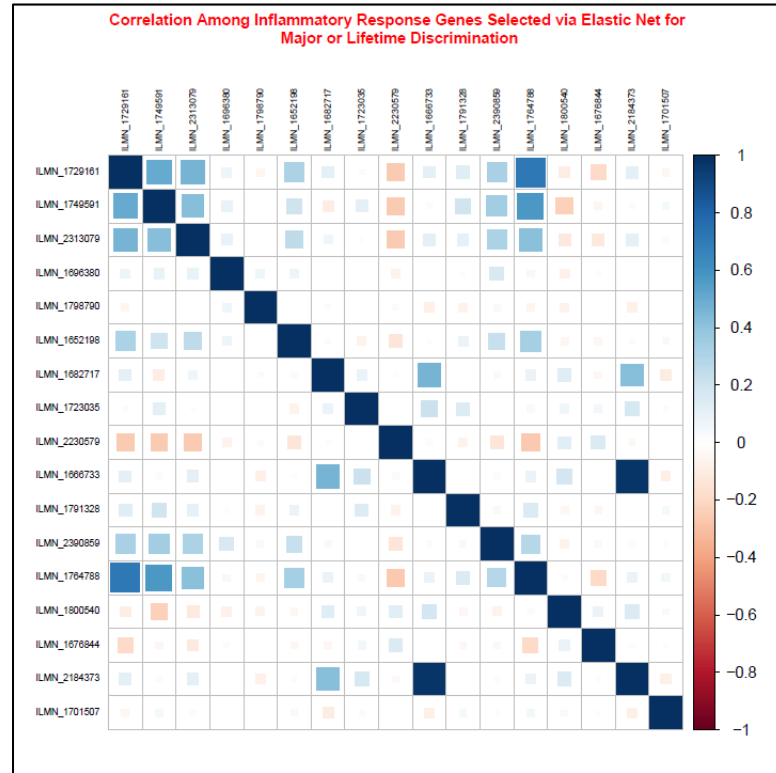
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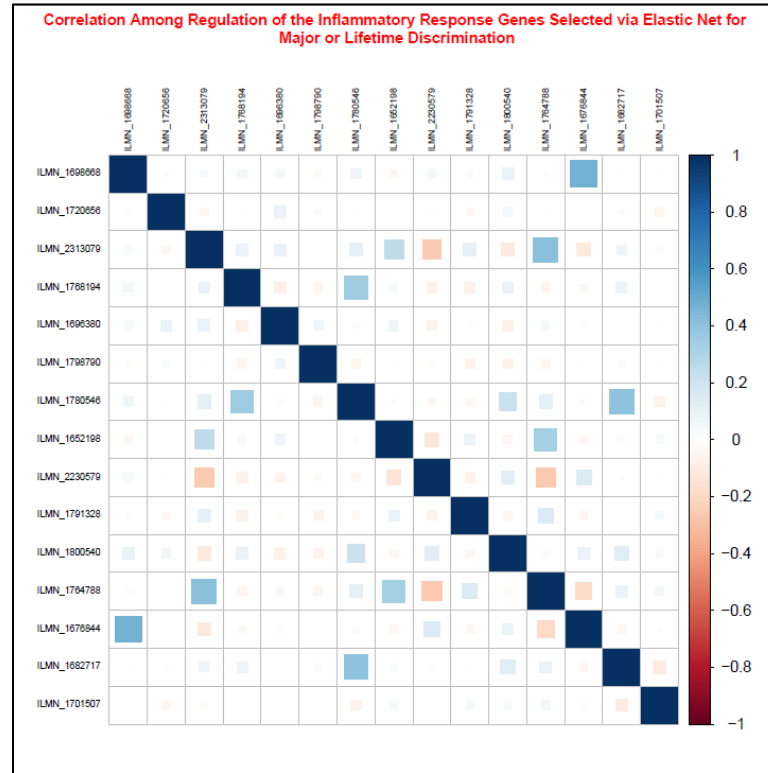
Appendix 22



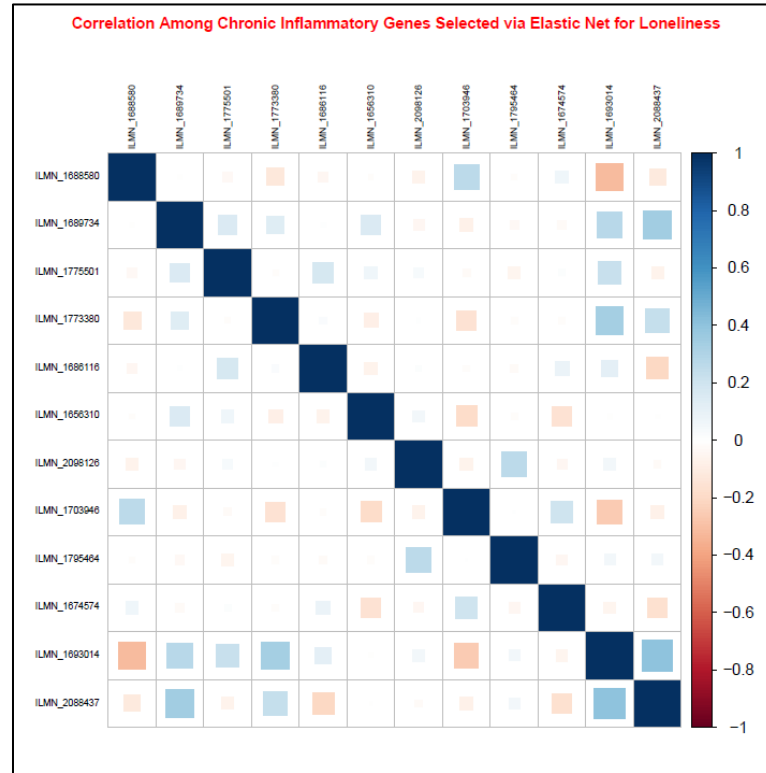
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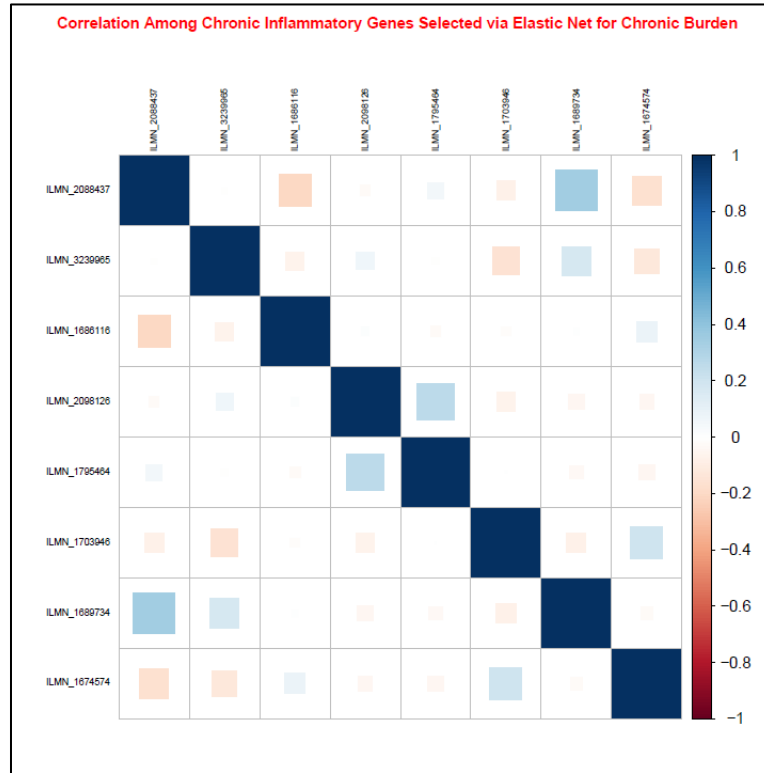
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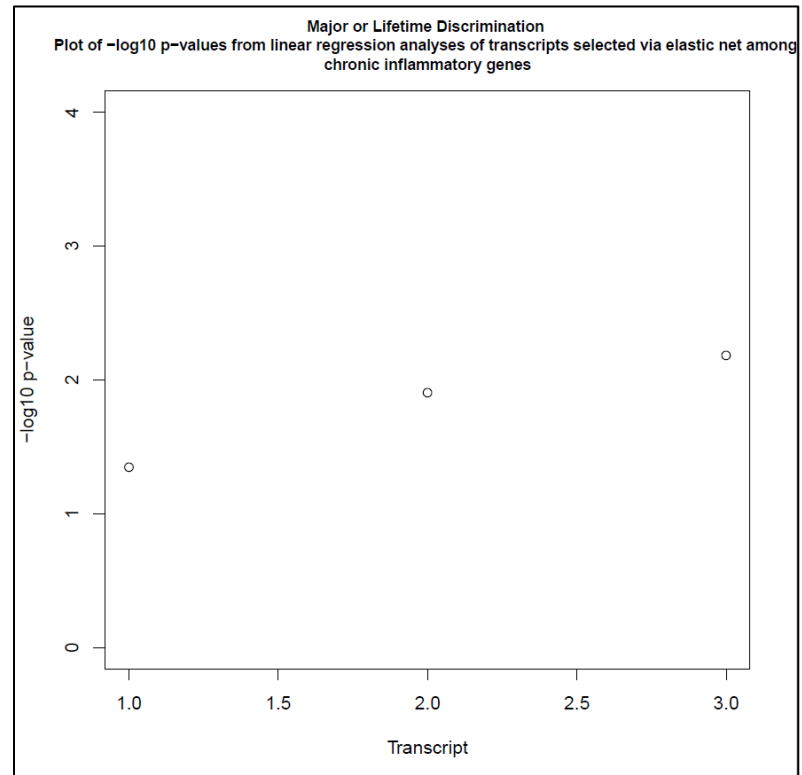
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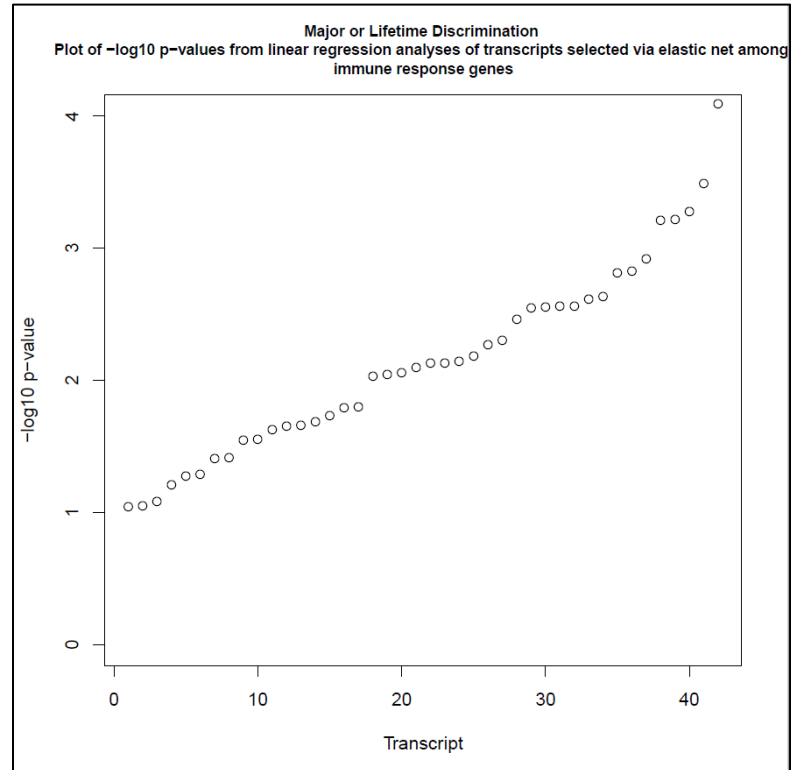
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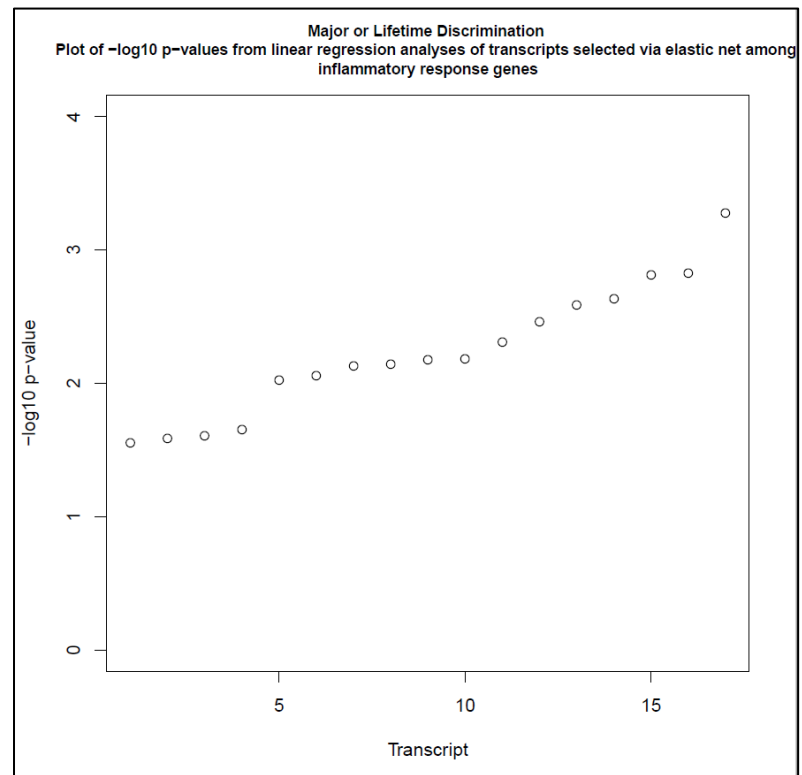
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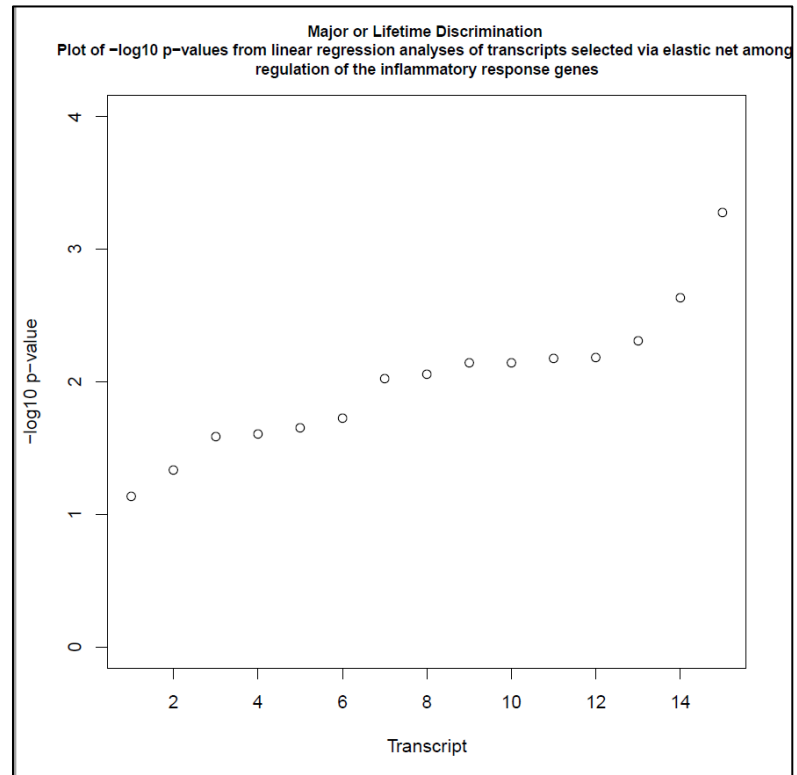
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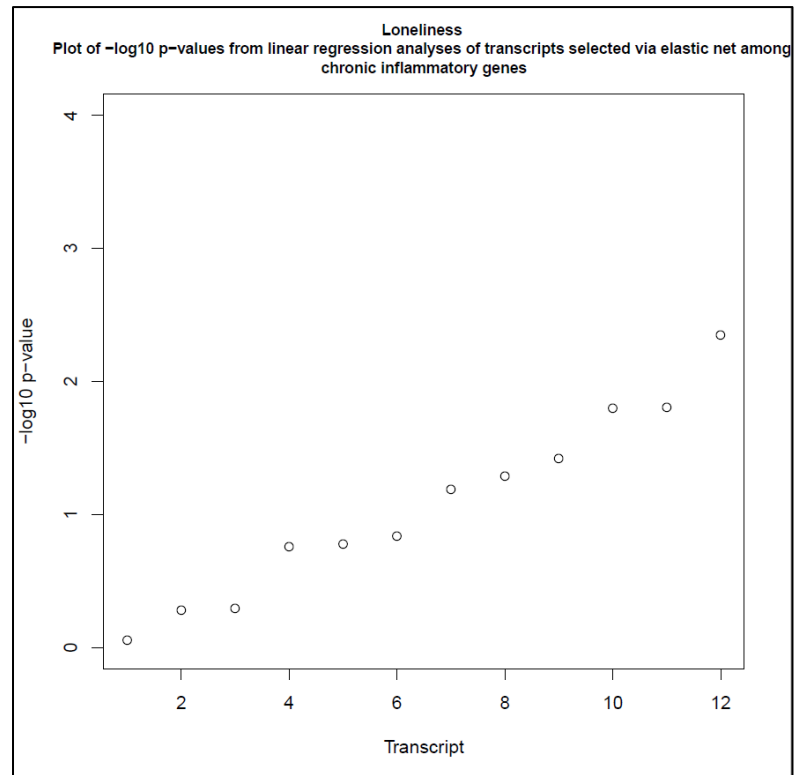
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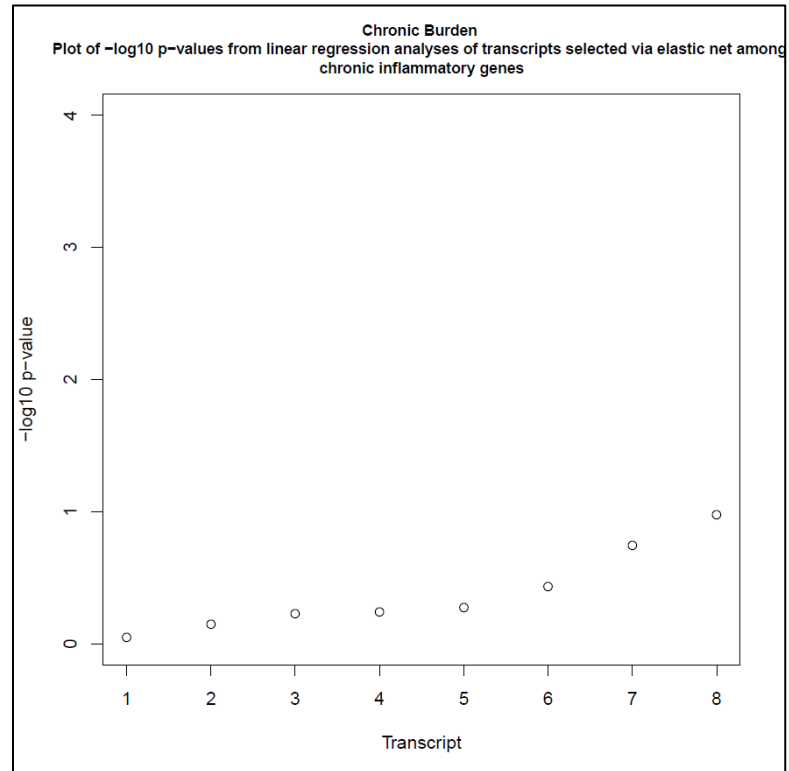
Appendix 30



Appendix 31



Appendix 32



Appendix 33

Genes with Racial/Ethnic Differences by Biological Process		
Race/ethnic comparison	Biological Process	Genes
Black/White		
	Chronic Inflammation	AHCY , VNN1, THBS1, IL1RN, CEBPB, ADORA2B, TNF, CX3CR1, CCL5, IL1RN , IL1B, LTA, CX3CR1, CCL5
	Immune Response	PNKD, IL4R, PIK3AP1, HMGB2, ADCY9, RASA3, TNFSF14, DUSP5, CRIP1, AHCY, OAS1, RGCC, RARA, CD79A, SPN, CLCF1, IRAK3, FCER2, PPP2R3C, PLEKHA1, STOML2, LFNG, CLEC4C, CD180, FCGR2A, MAPK3, C1QBP, EXOSC6, IRF5, CCL3, DUSP22, OAS1, F2RL1, IFITM2, CD97, PAG1, CASP8, VNN1, OAS1, NCAM1, ADCY3, CYFIP2, DUSP6, NR1H2, TRIM21, DHX58, TRPM4, ADARB1, COL4A3BP, TXN, CTSG, SPNS1, SIGLEC7, PTPN1, POLR3A, FOXP1, PSMB10, FTH1, HFE, TNFSF12, FKBP1A, ITGAM, THBS1, DEFB1, CSF1R , MT2A, SIGLEC7, ETS1, PEBP1, TREM1, CD81, STAT1, CCR6, FCRLA, STAT1, CCM2, CEBPB, FES, TRIM68, CTSL, AGO2, CTSB, NLRP3, MAP2K6, TNFRSF10, TNFRSF21, VPREB3, RNF135, SH2D1B, EHHADH, CD300C, RPS6KA2, PSMD1, UBE2L6,

		<p> NEDD4, AIF1, RASA2, ADORA2B, PSMA6, MARK3, FTH1, TLR4, ELANE, NFATC1, ADM, RAC2, CD79B, MYB, NLRP3, PRKCB, PTK2, ITGB1, CCR7, PAQR3, TSPAN32, LST1, CD1D, MSRB1, CCL2, VPS72, TLR5, SIRT2, CD1A, MAP4K2, OTUB1, LGALS1, SELL, RASA1, CD1B, CPAMD8, CD4, KRAS, TNF, PSMD4, CXCL16, FCGR3B, SRC, TRIM27, LGALS3, DUSP22, PTPRJ, PSMD9, SEMA4D, LILRA1, NBN, CD79A, THEMIS2, POU2F2, PAG1, BCL6, DDX41, MYD88, FOXO1, RNF135, ELMO1, PRF1, MAP3K8, CLEC6A, TNFAIP8L, PSMB5, PLSCR1, OAS3, IRF9, CX3CR1, POLR2F, IRAK2, NRROS, CTNNB1, POLR1C, TRIM8, C5, RNF125, LTB4R, MILR1, CD33, ATF2, HLA-DPB1, GPX1, SKAP1, C12orf4, CSK, IRF4, IKBKE, LAMTOR2, ST6GAL1, LY75, CTNNB1, TNFSF13B, DPP8, PSMA5, PIK3R1, TSPAN32, CD74, HLA-DMB, TNFSF8, STAT6, RIPK3, IFNGR2, S100A13, ADAM17, BPI, SOS1, POLR1D, POLR3B, SFTPD, RASGRP1, CCR2, PIK3CG, RPS6KA3, PNMA1, HLA-DPA1, CCL3L1, HRAS, PNKD, CCR2, IL1RN, IL1B, CFD, CCR2, ITGB7, ELMO2, CD40, PELI2, CLEC5A, CD83, LYN, FYN, DUSP7, CD19, CCM2, CDKN1A, CD36, NCF4, SWAP70, CD79B, LILRA3, CDKN1A, CASP8, TRIB3, MLST8, GNL1, </p>
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		<p> AIF1, CD7, VAV2, RNASE1, SIGLEC9, GPER1, LTA, PTGER4, CD36, HIST1H2B, PPP3R1, ERCC1, IL6ST, FFAR2, KLRD1, GPR183, KLRD1, HMOX1, BTN3A1, LAT2, ADA, SPRED1, PTPRC, SMAGP, SIN3A, PSME1, JUN, LY86, CLEC4D, XBP1, RELB, TNFRSF10, CTSL, DUSP22, HIST1H2B, TNFRSF9, IL16, TRIM25, NR1H3, PSMB7, F2RL1, PAG1, IFI27, SYK, FTH1, PHLPP2, SPTAN1, TNFSF15, CD300LF, RNASE3, NOD1, GBP5, ADAM17, ETS1, ERAP2, SKAP2, STXBP2, HLA-DRB4, GPR183, CXCL5, PRELID1, ANXA1, VAMP8, CLEC4G, SLAMF6, TREML4, RNASEL, ICAM3, HMGB2, MYO10, ECSIT, LAT2,FOXP1, LGMN, TRIM34, CD3D, CCR2, YWHAB, ERCC1, COL4A3BP, APOBEC3F, NLRP3, IRF5, ELMO2, PSMA3, TRIM4, CD3D, LAT2, RAB27A, TNFRSF10, RNASE1, CREB1, IL18BP, NCF4, CXCR5, LILRB5, PDE4B, IL7R, PSMD4, PTGES2, PTGES2, SOCS5, KL, LGALS8, CYFIP2, OTUB1, LGALS8, CD79B, CD40, IL32, UBA52, XAF1, BTN3A3, CTSL, PRKACB, CD37, LTB, LTB, CCR2 ERCC1,CASP8, SMAGP, CRCP, ITGB1, GPER1, NFATC1, PRKCZ, TSPAN32, PLEKHA1, ASS1, PYCARD, CLEC4A, DUSP10, MAPK3, S100A13, RNF135, CD97, CD96, CNRIP1, SIGLEC14, NDUFA2, PIK3R6, FCGR1C, FTH1, IP6K2 </p>
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	Inflammatory Response	<p>IL4R, PIK3AP1, HMGB2, AHCY, SPN, CD180, HYAL3, C1QBP, POLG2, CCL3, F2RL1, CD97, VNN1,CCR1, GART, CXCR2, SPNS1, FOXP1, HFE, ITGAM, THBS1, DEFB1, CSF1R, ETS1, LTA4H, CCM2, CEBPB, ADRB2, GHRL, ORM1, NLRP3, NR1D2, TNFRSF21, PLA2G7, EHHADH, AIF1, ADORA2B, PSMA6, TLR4, ELANE, NLRP3, NFKB1, CCR7, NFKBIZ, CCL2, ZFP36, CASP5, TLR5, LXN, TNF, THEMIS2, BCL6, FOXP1, MYD88, PLSCR1, CX3CR1, IRAK2, NRROS, C5, ADORA1, LTB4R, SNAP23, S100A12, GPX1, CAMK1D, MCPH1, APOL3, LY75, CALCRL, P2RX1, ACVR1, B4GALT1, PRDX2, POLB, RASGRP1, ELF3, CCR2, NFKBIZ, PIK3CG, PNMA1, CD47, CCL3L1, CCR2, IL1RN, PROK2, IL1B, CCR2, CD40, KLF4, LYN, CCM2, TNFAIP6, NOV, METRNL, STK39, AIF1, GART, RNASE1, GPER1, LTA, CAMK1D, PTGER4, IL6ST, FFAR2, HMOX1, METRNL, ADA, NDST1, LY86, SIGIRR, TSPAN2, PLA2G4C, RELB, HP, TNFRSF9, NR1H3, F2RL1, SYK, CX3CR1, NOD1, GBP5, ETS1, KLF4, LYZ, CXCL5, ANXA1, VAMP8, HMGB2, FOXP1, METRNL, CCR2, NLRP3, APOL2, RNASE1, METRNL, KL, ATRN, CD40, tCCR2, HIF1A, SEH1L, PRDX2, GPER1, PRKCZ, ASS1, PYCARD, MS4A1, DUSP10, F11R, CD97, NDUFA2</p>
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	Regulation of the Inflammatory Response	PIK3AP1, SPN, C1QBP, CCL3, FOXP1, ETS1, CCM2, ADRB2, GHRL, NLRP3, NR1D2, PLA2G7, EHHADH, ADORA2B, PSMA6, TLR4, ELANE, NLRP3, NFKB1, CCR7, ZFP36, CASP5, TNF, BCL6, MAPK14, FOXP1, MYD88, TNFAIP8L, CX3CR1, CD6, C5, ADORA1, S100A12, GPX1, MCPH1, CALCRL, CCR2, PIK3CG, CD47, CCL3L1, CCL5, CCR2, IL1B, CCR2, KLF4, LYN, CCM2, TNFAIP6, NOV, METRNL, STK39, GPER1, LTA, PTGER4, IL6ST, FFAR2, METRNL, ADA, NR1H3, CX3CR1, CCL5, GBP5, ETS1, KLF4, ANXA1, VAMP8, FOXP1, METRNL, CCR2, NLRP3, METRNL, CCR2, GPER1, DUSP10
Black/Hispanic	Chronic Inflammation	AHCY, VNN1, TNFAIP3, TNF, CX3CR1, CCL5, IL1B, LTA, CCL5
	Immune Response	PNKD, IL4R, RGS1, DUSP5, CRIP1, RPS6KA5, OAS1, RGCC, SPN, CLCF1, SPTBN1, IRAK3, FCER2, PLEKHA1, LFNG, CD180, CXCL8, MAPK3, BATF, EXOSC6, DUSP22, OAS1, F2RL1, VNN1, GAPT, OAS1, ADCY3, CYFIP2, LEF1, TRPM4, ADARB1, CTSG, SPNS1, ICAM4, IP6K2, HFE, TRAT1, DEFB1, SIGLEC7, ETS1, CD27, CCM2, FES, TRIM68, NLRP3, MAP2K6, ITK, TNFRSF10, RNF135, SH2D1B, CD300C, RPS6KA2, TNFAIP3, PSMD1, CLEC10A, PKN2, NFATC1, NFIL3, IRF1, ABL1, RAC2, CD79B, NLRP3, CORO1A, ITGB1, CCR7,

		<p>TSPAN32, CD1D, CCL2, EXOSC9, CDKN1B, TLR5, BST2, SIRT2, MAP4K2, OTUB1, CPAMD8, LILRA5, TNF, CXCL16, FCGR3B, SRC, LGALS3, HIST2H2B, MAPKAPK3, PSMD9, NBN, THEMIS2, TRIM28, PAG1, BCL6, RNF135, OSCAR, CLEC6A, PSMB5, CD6, C5, CD33, HLA-DPB1, RBCK1, SKAP1, CSK, IRF4, IKBKE, EIF2AK4, LY75, PRKRA, P2RX7, NAIP, EGR1, IFNGR2, IFNAR2, SOS1, POLR1D, CD8A, SFTPD, RASGRP1, CCR2, BST1, PNMA1, HLA-DPA1, CCL5, HRAS, PNKD, CCR2, IL1B, PIN1, CFD, CCR2, ELMO2, CD44, GZMA, OSM, TUBB4B, SOCS3, LYN, PDE4B, CCM2, S1PR4, CD36, CD79B, LILRA3, CDKN1A, TRIB3, GNL1, CD7, RNASE1, SIGLEC9, GPER1, LTA, CD36, HIST1H2B, PPP3R1, FFAR2, PPARG, HMOX1, IFITM1, LAT2, ADA, SMAGP, JUN, MIF, PADI4, CTSL, DUSP22, HIST1H2B, TNFRSF9, NR1H3, F2RL1, PAG1, KLRB1, ARPC1B, CCL5, RNASE3, ETS1, STXBP2, HLA-DRB4, GPR183, CXCL5, PRELID1, LIME1, CXCL8, CLEC4G, CDKN1B, TREML4, LEF1, FYN, NAIP, CD3D, CCR2, YWHAB, PSMC4, APOBEC3F, NLRP3, TRIM4, CD3D, LAT2, RNASE1, CREB1, PDE4B, IL7R, CD8A, CYFIP2, OTUB1, LILRA5, CD79B, IL32, BTN3A3, CTSL, LTB, LTB, CCR2, SMAGP, CRCP, ITGB1, GPER1, CCR6, TSPAN32, ICAM4, TNFSF13,</p>
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		CLEC4A, AP1G1, AP1G1, BIRC3, RNF135, CD96, IP6K2, GAPT, MED1, IP6K2, PGLYRP2
	Inflammatory Response	IL4R, RPS6KA5, SPN, CD180, CXCL8, HYAL3, F2RL1, VNN1, PTGS2, SPNS1, HIF1A, HFE, DEFB1, ETS1, CD27, LTA4H, CCM2, NLRP3, NR1D2, PLA2G7, EHHADH, TNFAIP3, ELANE, NLRP3, CCR7, CCL2, CASP5, TLR5, LXN, TNF, THEMIS2, BCL6, NLRP12, CD6, C5, ADORA1, MCPH1, APOL3, LY75, RIPK2, P2RX7, NAIP, HIF1A, PRDX2, POLB, RASGRP1, CCR2, PNMA1, CD47, CCL5, CCR2, PROK2, IL1B, CCR2, CD44, OSM, SOCS3, LYN, CCM2, MAP2K3, NFE2L2, RNASE1, GPER1, LTA, FFAR2, PPARG, HMOX1, CXCR4, ADA, MIF, TSPAN2, PLA2G4C, HP, TNFRSF9, NR1H3, PRDX5, F2RL1, CCL5, ETS1, CXCL5, CXCL8, TSPAN2, NAIP, CCR2, NLRP3, CXCR4, RNASE1, CCR2, HIF1A, SEH1L, PRDX2, GPER1, DUSP10, BIRC3, PGLYRP2
	Regulation of the Inflammatory Response	SPN, RPSA, F12, PTGS2, ETS1, CCM2, NLRP3, NR1D2, PLA2G7, EHHADH, TNFAIP3, ELANE, NLRP3, CCR7, CASP5, TNF, BCL6, MYD88, NLRP12, CD6, C5, ADORA1, MCPH1, CCR2, CD47, CCL5, CCR2, IL1B, CCR2, OSM, SOCS3, LYN, CCM2, GPX1, GPER1, LTA, FFAR2, PPARG, ADA, NR1H3, CCL5, ETS1, CCR2, NLRP3, CCR2, GPER1, DUSP10, BIRC3, PGLYRP2

Hispanic/White	Chronic Inflammation	AHCY, VNN1, THBS1, CAMP, CEBPB, TNFAIP3, ADORA2B, TNF
	Immune Response	CLEC10A, HMGB2, ADCY9, ABR, TNFSF14, RGS1, CRIP1, RPS6KA5, AHCY, RGCC, CD79A, STOML2, CD180, WASL, CRK, RBX1, CXCL8, C1QBP, FOS, IRF5, CCL3, CD97, PAG1, POLR3F, VNN1, NCAM1, PAK2, CCR1, LEF1, TRPM4, COL4A3BP, TXN, BIRC3, FOXO3, POLR3A, CXCL2, PSMB10, FTH1, JAK2, CAMK2D, TNFSF12, TRAT1, TNFRSF1A, CBLB, NFATC3, SIGLEC7, IFI6, PEBP1, TREM1, LST1, C5AR1, NFKBIB, FCRLA, PHB, CEBPB, AGO2, PHB2, TRIM38, LGMN, IK, TNFRSF21, RNF135, CD300C, RPS6KA2, CD244, PSMD1, AIF1, RASA2, ADORA2B, MARK, FTH1, PKN2, NFIL3, ARPC4, MAPK7, CD79B, BCL3, PNKD, MYB, TNRC6A, ITGB1, PAQR3, LST1, CD1D, MSRB1, EXOSC9, VPS72, CDKN1B, BST2, LGALS1, SELL, RASA1, KRAS, PSMD4, HIST2H2B, SEMA4D, PSMD8, LILRA1, CD79A, THEMIS2, POU2F2, PAG1, BCL6, DDX41, FOXP1, FOXO1, RNF135, MND A, SIRT1, MAP3K8, TNFAIP8L, EP300, POLR2F, IRAK2, NRROS, CTN NB1, POLR1C, RNF125, MILR1, PSMB3, HLA-DPB1, ADAM15, IRF4, EIF2AK4, LAMTOR2, ST6GAL1, CTN NB1, DPP8, ARPC1A, PSMA5, NAIP, PIK3R1, TSPAN32, HLA-DMB, UBB, PIK3CB, IFNGR2, S100A13, REL, BPI,

		<p>SOS1, BIRC2, BST1, PIK3CG, PNMA1, NFKBIA, CCL3L1, PNKD, PIN1, CCR2, ITGB7, CD44, CD40, PELI2, OSM, CD83, SOCS3, CD19, TRAF6, CDKN1A, SWAP70, SOCS5, CD79B, POLR3H, GPX1, C19orf10, MLST8, FER, IFNAR2, AIF1, RNASE1, PTGER4, CD36, NCF2, PPP3R1, FFAR2, GPR183, POLR3K, LAT2, VIMP, PTPRC, SMAGP, SIN3A, PSME1, LY86, CLEC4D, PSMC3, XBP1, AGO3, TNFRSF10, CTSL, TNFRSF9, IL16, NR1H3, PSMB7, IL12RB1, ISG15, PAG1, PSMA2, PHLPP2, TNFSF15, CD300LF, NOD1, ADAM17, ERAP2, REL, SOCS3, MR1, GPR183, GPI, PRELID1, CXCL8, CLEC4G, SLAMF6, CDKN1B, POLR3A, ICAM3, LEF1, ECSIT, FYN, LGMN, TRIM34, NAIP, CD3D, ERCC1, COL4A3BP, EXOSC9, DPP8, PSMA3, CAMK2D, CD3D, LAT2, RAB27A, TNFRSF10, RNASE1, CREB1, CREB1, IL18BP, NCF4, MAPK7, PDE4B, PSMD13, PSMD4, PTGES2, LST1, WIPF1, KL, LGALS8, CYFIP1, CYFIP1, LGALS8, CD79B, CD40, UBA52, XAF1, BTN3A3, CTSL, PRKACB, ERCC1, CASP8, SMAGP, CRCP, ITGB1, PRKCZ, CCR6, ARPC4, PLEKHA1, FBXW11, DUSP6, PYCARD, MAPK3, BIRC3, RBCK1, RNF135, CD97, CLEC10A, IP6K2, RPS6, GPR183, NDUFA2, FTH1</p>
	Inflammatory Response	<p>HMGB2, ABR, RPS6KA5, AHCY, CD180, CXCL8, HYAL3, C1QBP, FOS, CCL3,</p>

		<p>CD97, VNN1, PTGS2, CCR1, CXCR2, BIRC3, FOXP1, CXCL2, JAK2, TNFRSF1A, NFATC3, THBS1, C5AR1, PHB, CEBPB, PHB2, GHRL, ORM1, TNFRSF21, PLA2G7, AIF1, ADORA2B, MAPK7, NFKB1, CEBPA, NFKBIZ, ZFP36, OLR1, THEMIS2, LIAS, BCL6, FOXP1, NLRP12, CD14, IRAK2, NRROS, CAMK1D, MCPH1, APOL3, P2RX1, NAIP, ACVR1, REL, PIK3CD, PRDX2, POLB, BIRC2, PIK3CG, PNMA1, ATRN, CCL3L1, CCR2, CD44, CD40, KLF4, OSM, SOCS3, FASN, TNFAIP6, NOV, GPX1, C19orf10, METRNL, NFE2L2, STK39, AIF1, GART, RNASE1, CAMK1D, PTGER4, FFAR2, CXCR4, VIMP, TUSC2, NDST1, LY86, SIGIRR, PLA2G4C, TNFRSF9, NR1H3, PRDX5, NOD1, REL, KLF4, SOCS3, LYZ, CXCL8, NAIP, CXCR4, RNASE1, MAPK7, METRNL, KL, ATRN, CD40, PRDX2, PRKCZ, PYCARD, BIRC3, F11R, CD97, NDUFA2</p>
	<p>Regulation of the Inflammatory Response</p>	<p>ABR, C1QBP, CCL3, PTGS2, BIRC3, FOXP1, JAK2, TNFRSF1A, PHB, PHB2, GHRL, PLA2G7, TNFAIP3, ADORA2B, MAPK7, NFKB1, ZFP36, BCL6, FOXP1, NLRP12, TNFAIP8L, ADORA1, MCPH1, CALCRL, BIRC2, PIK3CG, CCL3L1, CCR2, KLF4, OSM, SOCS3, TNFAIP6, NOV, GPX1, METRNL, STK39, PTGER4, FFAR2, VIMP, NR1H3, KLF4, SOCS3, MAPK7, METRNL, BIRC3</p>

Appendix 34

Overlapping Genes Significantly Associated with at Least Two Race/Ethnic Comparisons by Biological Process		
Biological Process	Racial/Ethnic Comparisons	Genes
Chronic Inflammation		
	Black/White – Hispanic White	AHCY, VNN1, THBS1, CEBPB, ADORA2B, TNF
	Black/White - Black/Hispanic	AHCY, VNN1, TNF, CX3CR1, CCL5, IL1B, LTA, CCL5
	Black/Hispanic - Hispanic/White	AHCY, VNN1, TNFAIP3, TNF
	All	AHCY, VNN1, TNF
Immune Response		
	Black/White – Hispanic White	HMGB2, ADCY9, TNFSF14, CRIP1, AHCY, RGCC, CD79A, STOML2, CD180, C1QBP, IRF5, CCL3, CD97, PAG1, VNN1, NCAM1, TRPM4, COL4A3B, TXN, POLR3A, PSMB10, FTH1, TNFSF12, SIGLEC7, PEBP1, TREM1, FCRLA, CEBPB, AGO2, TNFRSF2, RNF135, CD300C, RPS6KA2, PSMD1, AIF1, RASA2, ADORA2B, MARK3, FTH1, CD79B, MYB, ITGB1, PAQR3, LST1, CD1D, MSRB1, VPS72, LGALS1, SELL, RASA1, KRAS, PSMD4, SEMA4D, LILRA1, CD79A, THEMIS2, POU2F2, PAG1, BCL6, DDX41, FOXO1, RNF135, MAP3K8, TNFAIP8, POLR2F, IRAK2, NRROS, CTNNB1, POLR1C, RNF125, MILR1, HLA-DPB, IRF4, LAMTOR2, ST6GAL1, CTNNB1,

		<p>DPP8, PSMA5, PIK3R1, TSPAN32, HLA-DMB, IFNGR2, S100A13, BPI, SOS1, PIK3CG, PNMA1, CCL3L1, PNKD, CCR2, ITGB7, CD40, PELI2, CD83, CD19, CDKN1A, SWAP70, CD79B, MLST8, AIF1, RNASE1, PTGER4, CD36, PPP3R1, FFAR2, GPR183, LAT2, PTPRC, SMAGP, SIN3A, PSME1, LY86, CLEC4D, XBP1, TNFRSF1, CTSL, TNFRSF9, IL16, NR1H3, PSMB7, PAG1, PHLPP2, TNFSF15, CD300LF, NOD1, ADAM17, ERAP2, GPR183, PRELID1, CLEC4G, SLAMF6, ICAM3, ECSIT, LGMN, TRIM34, CD3D, ERCC1, COL4A3B, PSMA3, CD3D, LAT2, RAB27A, TNFRSF1, RNASE1, CREB1, IL18BP, NCF4, PDE4B, PSMD4, PTGES2, KL, LGALS8, LGALS8, CD79B, CD40, UBA52, XAF1, BTN3A3, CTSL, PRKACB, ERCC1, CASP8, SMAGP, CRCP, ITGB1, PRKCZ, PLEKHA1, PYCARD, MAPK3, RNF135, CD97, NDUFA2, FTH1</p>
	<p>Black/White - Black/Hispanic</p>	<p>PNKD, IL4R, DUSP5, CRIP1, OAS1, RGCC, SPN, CLCF1, IRAK3, FCER2, PLEKHA1, LFNG, CD180, MAPK3, EXOSC6, DUSP22, OAS1, F2RL1, VNN1, OAS1, ADCY3, CYFIP2, TRPM4, ADARB1, CTSG, SPNS1, HFE, DEFB1, SIGLEC7, ETS1, CCM2, FES, TRIM68, NLRP3, MAP2K6,</p>

		<p>TNFRSF1, RNF135, SH2D1B, CD300C, RPS6KA2, PSMD1, NFATC1, RAC2, CD79B, NLRP3, ITGB1, CCR7, TSPAN32, CD1D, CCL2, TLR5, SIRT2, MAP4K2, OTUB1, CPAMD8, TNF, CXCL16, FCGR3B, SRC, LGALS3, PSMD9, NBN, THEMIS2, PAG1, BCL6, RNF135, CLEC6A, PSMB5, C5, CD33, HLA-DPB, SKAP1, CSK, IRF4, IKBKE, LY75, IFNGR2, SOS1, POLR1D, SFTPD, RASGRP1, CCR2, PNMA1, HLA-DPA, HRAS, PNKD, CCR2, IL1B, CFD, CCR2, ELMO2, LYN, CCM2, CD36, CD79B, LILRA3, CDKN1A, TRIB3, GNL1, CD7, RNASE1, SIGLEC9, GPER1, LTA, CD36, HIST1H2, PPP3R1, FFAR2, HMOX1, LAT2, ADA, SMAGP, JUN, CTSL, DUSP22, HIST1H2, TNFRSF9, NR1H3, F2RL1, PAG1, RNASE3, ETS1, STXBP2, HLA-DRB, GPR183, CXCL5, PRELID1, CLEC4G, TREML4, CD3D, CCR2, YWHAB, APOBEC3, NLRP3, TRIM4, CD3D, LAT2, RNASE1, PDE4B, IL7R, CYFIP2, OTUB1, CD79B, IL32, BTN3A3, CTSL, LTB, LTB, CCR2, SMAGP, CRCP, ITGB1, GPER1, TSPAN32, CLEC4A, RNF135, CD96, IP6K2</p>
	Black/Hispanic - Hispanic/White	<p>RGS1, CRIP1, RPS6KA5, RGCC, CD180, CXCL8, VNN1, LEF1, TRPM4, TRAT1, SIGLEC7, RNF135, CD300C,</p>

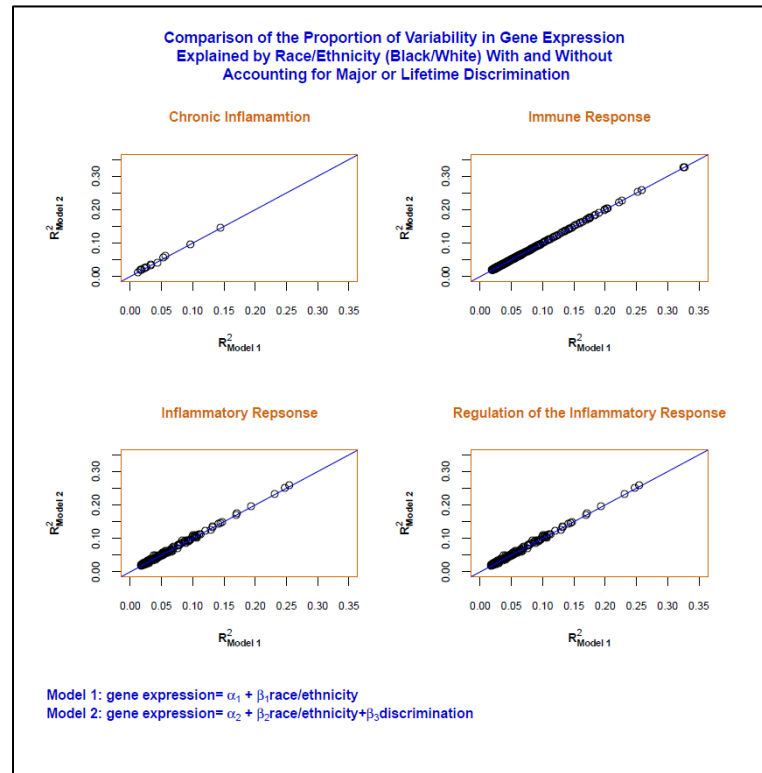
		RPS6KA2, PSMD1, PKN2, NFIL3, CD79B, ITGB1, CD1D, EXOSC9, CDKN1B, BST2, HIST2H2, THEMIS2, PAG1, BCL6, RNF135, HLA-DPB, IRF4, EIF2AK4, NAIP, IFNGR2, SOS1, BST1, PNMA1, PNKD, PIN1, CCR2, CD44, OSM, SOCS3, CD79B, RNASE1, CD36, PPP3R1, FFAR2, LAT2, SMAGP, CTSL, TNFRSF9, NR1H3, PAG1, GPR183, PRELID1, CXCL8, CLEC4G, CDKN1B, LEF1, FYN, NAIP, CD3D, CD3D, LAT2, RNASE1, CREB1, PDE4B, CD79B, BTN3A3, CTSL, SMAGP, CRCP, ITGB1, CCR6, BIRC3, RNF135, IP6K2
	All	CRIP1, RGCC, CD180, VNN1, TRPM4, SIGLEC7, RNF135, CD300C, RPS6KA2, PSMD1, CD79B, ITGB1, CD1D, THEMIS2, PAG1, BCL6, RNF135, HLA DPB, IRF4, IFNGR2, SOS1, PNMA1, PNKD, CCR2, CD79B, RNASE1, CD36, PPP3R1, FFAR2, LAT2, SMAGP, CTSL, TNFRSF9, NR1H3, PAG1, GPR183, PRELID1, CLEC4G, CD3D CD3D, LAT2, RNASE1, PDE4B, CD79B, BTN3A3, CTSL, SMAGP, CRCP, ITGB1, RNF135
Inflammatory Response		
	Black/White – Hispanic White	HMGB2, AHCY, CD180, HYAL3, C1QBP, CCL3, CD97, VNN1, CCR1, CXCR2, FOXP1, THBS1, CEBPB, GHRL, ORM1, TNFRSF2, PLA2G7,

		AIF1, ADORA2B, NFKB1, NFKBIZ, ZFP36, THEMIS2, BCL6, FOXP1, IRAK2, NRROS, CAMK1D, MCPH1, APOL3, P2RX1, ACVR1, PRDX2, POLB, PIK3CG, PNMA1, CCL3L1, CCR2, CD40, KLF4, TNFAIP6, NOV, METRNL, STK39, AIF1, GART, RNASE1, CAMK1D, PTGER4, FFAR2, NDST1, LY86, SIGIRR, PLA2G4C, TNFRSF9, NR1H3, NOD1, KLF4, LYZ, RNASE1, METRNL, KL, ATRN, CD40, PRDX2, PRKCZ, PYCARD, F11R, CD97, NDUFA2
	Black/White - Black/Hispanic	IL4R, SPN, CD180, HYAL3, F2RL1, VNN1, SPNS1, HFE, DEFB1, ETS1, LTA4H, CCM2, NLRP3, NR1D2, PLA2G7, EHHADH, ELANE, NLRP3, CCR7, CCL2, CASP5, TLR5, LXN, TNF, THEMIS2, BCL6, C5, ADORA1, MCPH1, APOL3, LY75, PRDX2, POLB, RASGRP1, CCR2, PNMA1, CD47, CCR2, PROK2, IL1B, CCR2, LYN, CCM2, RNASE1, GPER1, LTA, FFAR2, HMOX1, ADA, TSPAN2, PLA2G4C, HP, TNFRSF9, NR1H3, F2RL1, ETS1, CXCL5, CCR2, NLRP3, RNASE1, CCR2, HIF1A, SEH1L, PRDX2, GPER1, DUSP10
	Black/Hispanic - Hispanic/White	RPS6KA5, CD180, CXCL8, HYAL3, VNN1, PTGS2, PLA2G7, THEMIS2, BCL6, NLRP12, MCPH1, APOL3, NAIP, PRDX2, POLB, PNMA1, CCR2,

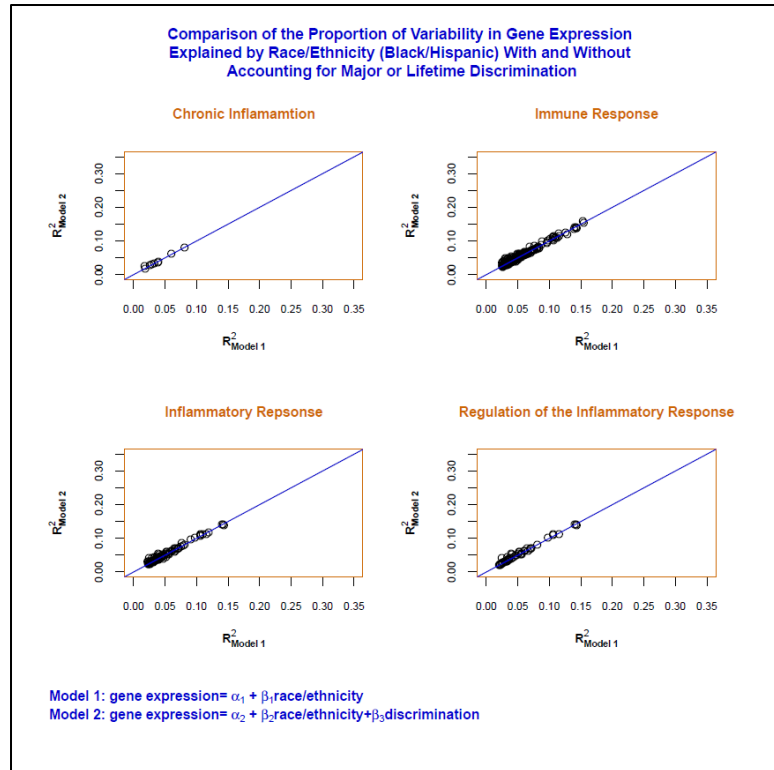
		CD44, OSM, SOCS3, NFE2L2, RNASE1, FFAR2, CXCR4, PLA2G4C, TNFRSF9, NR1H3, PRDX5, CXCL8, NAIP, CXCR4, RNASE1, PRDX2, BIRC3
	All	CD180, HYAL3, VNN1, PLA2G7, THEMIS2, BCL6, MCPH1, APOL3, PRDX2, POLB, PNMA1, CCR2, RNASE1, FFAR2, PLA2G4C, TNFRSF9, NR1H3, RNASE1, PRDX2
Regulation of the Inflammatory Response		
	Black/White – Hispanic White	C1QBP, CCL3, FOXP1, GHRL, PLA2G7, ADORA2B, NFKB1, ZFP36, BCL6, FOXP1, TNFAIP8, ADORA1, MCPH1, CALCRL, PIK3CG, CCL3L1, CCR2, KLF4, TNFAIP6, NOV, METRNL, STK39, PTGER4, FFAR2, NR1H3, KLF4, METRNL
	Black/White - Black/Hispanic	SPN, ETS1, CCM2, NLRP3, NR1D2, PLA2G7, EHHADH, ELANE, NLRP3, CCR7, CASP5, TNF, BCL6, MYD88, CD6, C5, ADORA1, MCPH1, CCR2, CD47, CCL5, CCR2, IL1B, CCR2, LYN, CCM2, GPER1, LTA, FFAR2, ADA, NR1H3, CCL5, ETS1, CCR2, NLRP3, CCR2, GPER1, DUSP10
	Black/Hispanic - Hispanic/White	PTGS2, PLA2G7, TNFAIP3, BCL6, NLRP12, ADORA1, MCPH1, CCR2, OSM, SOCS3, GPX1, FFAR2, NR1H3, BIRC3

	All	PLA2G7, BCL6, ADORA1, MCPH1, CCR2, FFAR2, NR1H3
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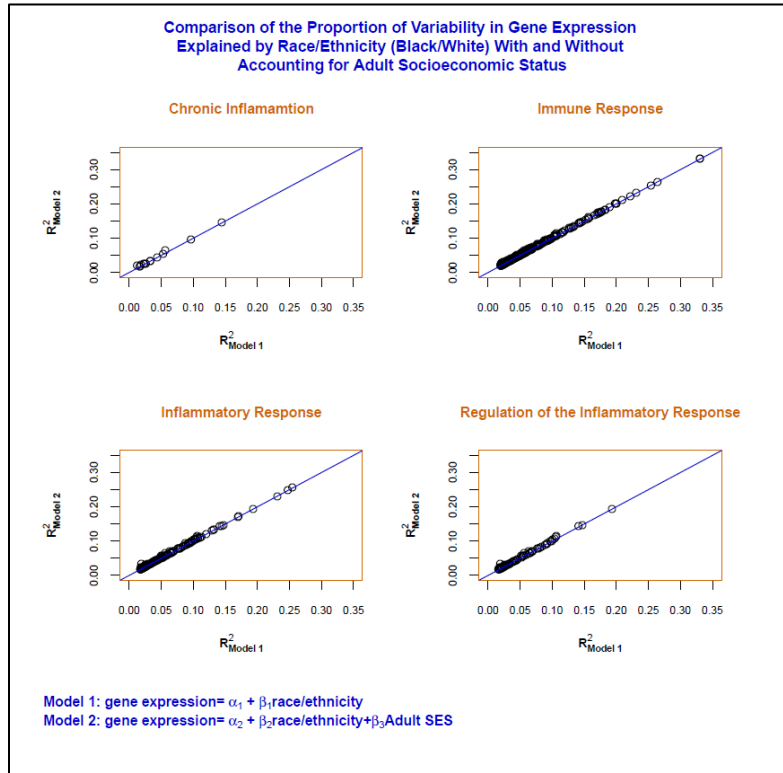
Appendix 35



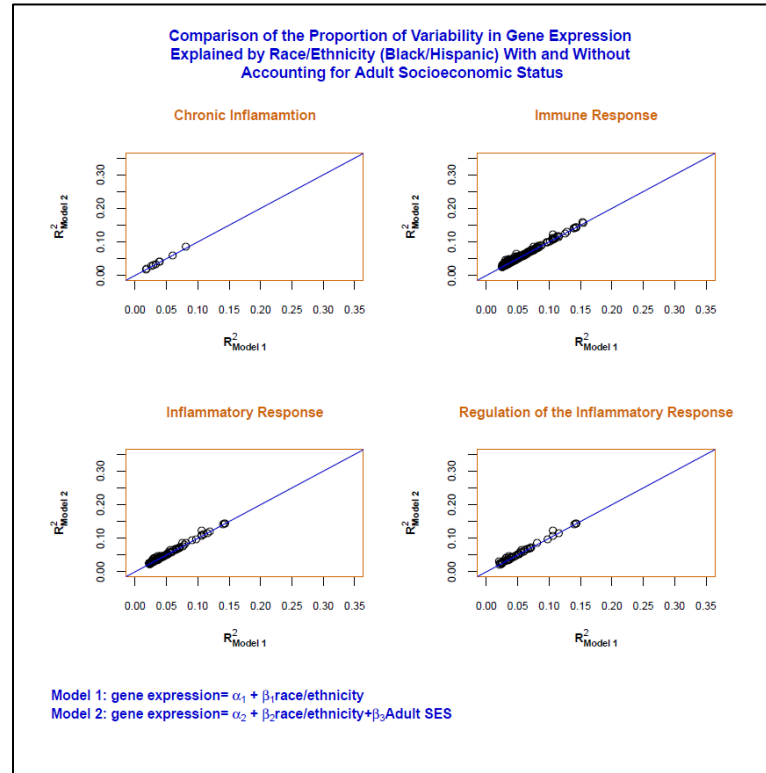
Appendix 36



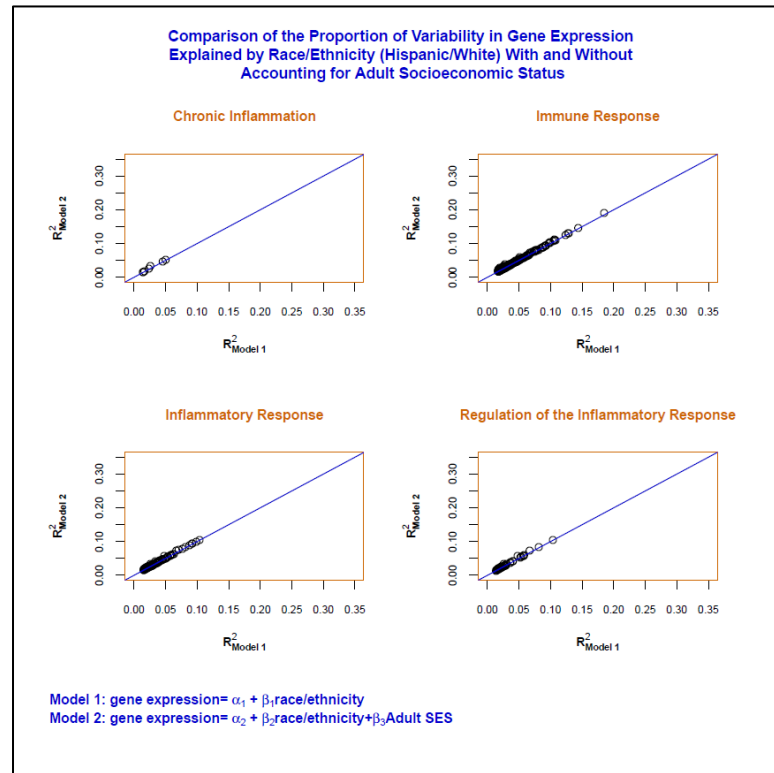
Appendix 37



Appendix 38



Appendix 39



Appendix 40

Overlap between Gene Transcripts Assessed in Aim 2 and 3 with Gene Transcripts Assessed in Aim 1		
Aim 2 & 3 biological processes	# of transcripts in each biological process	# of transcripts also assessed in Aim 1*
Chronic Inflammation	20	17
Immune Response	1251	422
Inflammatory Response	438	148
Regulation of Inflammatory Response	192	60
* 1854 gene transcripts were assessed in Aim 1		