Supplementary Table 1. Differentially methylated regions between rhesus macaques exposed to wildfire smoke in early life and rhesus macaques with no early life exposure to wildfire smoke. Note: betaCoefficient is presented with respect to exposed macaques (*i.e.* a positive betaCoefficient implies hypermethylation in exposed macaques compared to control macaques)

Supplementary Table 2. Canonical pathways from Ingenuity Pathway Analysis that were enriched in all differentially methylated regions.

Supplementary Table 3. Canonical pathways from Ingenuity Pathway Analysis that were enriched in differentially methylated regions hypermethylated in macaques exposed to wildfire smoke in early life.

Supplementary Table 4. Canonical pathways from Ingenuity Pathway Analysis that were enriched in differentially methylated regions hypomethylated in macaques exposed to wildfire smoke in early life.

Supplementary Table 5. Transcription factor binding site motifs that were significantly enriched in differentially methylated regions (from HOMER (1)).

Supplementary Table 6. The differentially expressed gene between rhesus macaques exposed to wildfire smoke in early life and rhesus macaques with no early life exposure to wildfire smoke. Note: log2FoldChange is presented with respect to exposed macaques (*i.e.* a positive

log2FoldChange implies greater expression in exposed macaques compared to control macaques).

Supplementary Table 7. Genes in the purple module (the module most significantly associated with exposure) from the weighted gene coexpression network analysis (WGCNA (2)).

Supplementary Table 8. Canonical pathways from Ingenuity Pathway Analysis that were enriched in genes in the purple module from the WGCNA (2) analysis.

Supplementary Table 9. Genes that showed significant correlation ($p \leq 0.05$) between methylation and expression across all samples.

Supplementary Table 10. Canonical pathways from Ingenuity Pathway Analysis that were enriched in genes that had significantly correlated methylation and expression.

Supplementary Table 11. Comparison between differentially methylated genes from the current study and other studies on respiratory diseases.

Supplementary Table 12. Extended information on the samples in our current study.

Supplementary Figure 1. Enrichment of different CpG features associated with all differentially methylated regions, regions hypermethylated in wildfire-exposed macaques, and regions

hypomethylated in wildfire-exposed macaques. Asterisks indicate a significant deficit or enrichment of the feature in a given set ($p \leq 0.05$).

Supplementary Figure 2. Enrichment of different genic features associated with all differentially methylated regions, regions hypermethylated in wildfire-exposed macaques, and regions hypomethylated in wildfire-exposed macaques. Asterisks indicate a significant deficit or enrichment of the feature in a given set ($p \leq 0.05$).

Supplementary Figure 3. Heatmaps showing sample clustering by A) methylation and B) gene expression, and principal component analysis showing sample clustering by C) methylation and D) gene expression.

Supplementary Figure 4. Module-trait relationship between clusters identified in WGCNA and either exposure or animal weight. The top number in each box is the correlation value (ranging from -1 to 1), while the bottom number in parentheses is the p-value for this correlation.

Supplementary Figure 5. Top enriched biological process, cellular component, and molecular function gene ontology terms identified by GOfuncR (3) associated with differentially methylated regions between wildfire-exposed macaques and control macaques.

Supplementary Figure 6. Heatmap showing all samples clustering by gene expression. Gene expression data from the two leftmost samples were removed from the study as outliers.

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