We thank the reviewers for their time and comments. Below are detailed responses to each comment. The responses are italicized and the modified texts are in red. The main comments have been addressed by including a discussion on the limitations of this study and by clarifying our statistical analysis method. The revisions do not affect the conclusions of the manuscript.

Response to anonymous referee #2 comments:

1) **Limitations of this study: I didn't see any discussion regarding the limitations of this study, and they mainly cited their own DTT papers throughout the discussion. This would be my most major criticism. You have to be careful to say that your acute exposures here will really translate to the in vivo condition. Specifically, why does one need to be careful in extending the results obtained from in vitro exposures to the in vivo condition? What are the potential issues with extracting filters for resuspension into cell culture? Does the chemistry change, and if so, how might that affect the toxicological response?**

   Thank you for your suggestion. We are aware that there are limitations regarding all health studies and have modified the manuscript to include several examples of these limitations. We note that the main objective of this study was to provide perspective on the relative toxicities of different SOA systems. Further studies are required to establish whether results from in vitro assays represent in vivo animal exposures, and from there, whether results from animal exposure studies can be generalized to actual human exposures.

   Line 552: "Additionally, this study confirms…"

   Line 562: "…to fully interpret ROS/RNS measurements. Finally, several limitations must be considered before generalizing results from this study to *in vivo* exposures. For instance, only one cell type was explored in this study, whereas an organism consists of multiple tissues comprised of multiple cell types. Interactions between different cell types and tissue systems were not considered in this study. Furthermore, the doses investigated may not fully represent real world exposures due to differences in exposure routes and potential recovery from doses due to clearance. Nevertheless, this study provides perspective on the relative toxicities of different SOA systems which future studies can build upon."

2) **Rationale for using murine alveolar macrophages: I think the authors should provide the rationale for using murine alveolar macrophages for this study. Would certain phenotype of this cell line differ from human alveolar macrophages? How easily relatable is it to human cells? What are limitations of cell lines versus primary cells and would that matter?**

   Thank you for your comment. We have included rationale for using this cell type in the manuscript. We chose murine alveolar macrophages as they are the first line of defense

against environmental insults, and the particular cell line (MH-S) retains many properties of primary alveolar macrophages (e.g., phagocytosis, cytokine production, ROS/RNS production) (Sankaran and Herscowitz, 1995; Mbawuike and Herscowitz, 1989). Furthermore, we have successfully utilized this cell line to investigate the production of ROS/RNS as a result of exposure to ambient PM samples (Tuet et al., 2016). To our knowledge, immortalized human alveolar macrophages do not exist. Mice have also been widely used as a model organism for studying human responses (Rosenthal and Brown, 2007; Takao and Miyakawa, 2015). As for the choice between cell lines and primary cells, primary cells are harvested from multiple animals, which increases the response variability. Results may therefore be less reproducible compared to cell lines.

Line 137: "Exposures were conducted using immortalized murine alveolar macrophages (MH-S, ATCC®CRL-2019™) as they are the first line of defense against environmental insults (Oberdörster, 1993; Oberdörster et al., 1992). The particular cell line also retains many properties of primary alveolar macrophages, including phagocytosis as well as the production of ROS/RNS and cytokines (Sankaran and Herscowitz, 1995; Mbawuike and Herscowitz, 1989). MH-S cells were cultured…"

3) **I noticed that the authors' cell culture and exposure media contain fetal bovine serum (FBS), which is known to potentially interfere with the ELISA assays. Normally people use serum-free media to avoid such interferences. Do the authors have any control experiments to show that FBS wouldn't interfere with their ELISA measurement?**

We normalized all ELISA responses to a control (cell culture supernatant from cells exposed to stimulant-free media supplemented with FBS) to capture any interferences. For our time point (24 hrs), FBS supplemented media is necessary to prevent serum starvation, which is known to induce oxidative stress (Kuznetsov et al., 2011; Wright et al., 2012). We also disagree that serum-free media is generally used for ELISA measurements, as many previous studies have performed exposures using supplemented media (e.g., Mukherjee et al., 2009; Chen et al., 2007; Sullivan et al., 2000).

4) **They use the cell media to extract filters. Since cell media contain a lot of supplementary materials/nutrients, would this affect the fraction of SOA materials extracted? Also, for the reactive products, would they be hydrolyzed before cell exposure?**

For oxidative potential measurements, it is known that using different extraction methods (e.g., different solvent, filtration, removing the filter) results in different components extracted and hence yields different oxidative potential measurements (Gao et al., 2017). However, there are limitations for each method. For instance, using an organic solvent requires the subsequent removal of the solvent via evaporation, which may result in loss of unstable components (e.g., semi-volatile organics). In this study, we chose to adapt an extraction method best suited for cellular exposure. While media contains species that

would indeed alter the fraction of material extracted, these species are also present in the alveolar fluid and the extract obtained is biologically relevant. We would also like to note that plain media (without FBS) was used for extraction and that FBS was supplemented after filtration of extracts. We did not investigate the hydrolysis of reactive products due to extraction, however this would be a potential issue for all extraction methods used in offline analysis. Further studies comparing offline and online analysis are required to investigate this.

5) **Lines 265-266: I think the redox activity is likely more sensitive to the functionality/electronic configuration of the functional groups, instead of carbon backbone. If it is carbon backbone, it looks to me that DTT is removed by other mechanisms such as absorption, but not through redox mechanisms.**

The referenced section refers to a previous study, where the chemical oxidative potentials as determined by DTT consumption were measured for these SOA systems (Tuet et al., 2017). In this study, we focus on the cellular responses and we find that the precursor identity and formation condition are both important and affect the cellular responses significantly. We note that there may have been some confusion in this section, as we intended "carbon backbone" to include both carbon chain length and functionalities. We have modified the manuscript to clarify our findings.

Line 27: "…which suggests that the chemical structure (carbon chain length and functionalities) of photooxidation products may be important…"

Line 295: "DTT may only be sensitive to larger differences arising from different precursors, whereas cellular assays…"

Line 305: "…for SOA precursors whose products share similar chemical structures (i.e., similar carbon chain length and functionalities)…"

Line 529: "…SOA systems whose products share similar functionalities and carbon chain length are likely to induce…"

6) **How these inflammatory responses relate to each other? Are they involved in the same biological network? They probably need to provide a more detailed biological background for the biomarkers they measured. For example, TNF-alpha induces IL-8 via NF-κB. This is well known in the toxicological literature. In some of the toxicological literature, TNF-alpha is used as positive control to stimulate IL-8 in BEAS-2B cells. I don't see a clear connection between the endpoints they measured in this paper and this needs to be more justified. Without a connection to a specific biological system, it makes it hard (especially for an atmospheric chemist I'm sure) to understand what your results really mean.**

Thank you for your suggestion. We have included justification on our cytokine measurements.

Line 106: "…cytokines indicative of the inflammatory response. TNF-α is a hallmark biomarker involved in triggering a number of cellular signaling cascades. More specifically, TNF-α is involved in the activation of NFκB, which regulates the expression of a variety of genes involved in inflammation and cell death, and the activation of protein kinases, which regulate various signaling cascades (Witkamp and Monshouwer, 2000). IL-6 has both pro- and anti-inflammatory effects, and may directly inhibit TNF-α (Kamimura et al., 2004). Furthermore, both cytokines are produced at relatively high levels in MH-S cells, ensuring a high signal-to-noise ratio and thus reliable measurements (Matsunaga et al., 2001; Chen et al., 2007)."

7) **Lines 288-290: The authors cite Lin et al. (2016, ES&T Letters), but I think this discussion is really unclear. What genes are similar? What pathways do the authors mean? They should make them clear. Note that Lin et al. (2016, ES&T Letters) only measured oxidative stress-associated genes, but not inflammatory genes in that paper. I noted that Lin et al. (2017, ES&T) just had a newly accepted paper where they found most genes are associated with the Nrf2 pathway, but not much inflammatory response from isoprene SOA exposure under non-cytotoxic conditions. Also, in Lin et al. (2017, ES&T) time course experiments, they found that IL-8 expression is time sensitive. The expression maximized at 9 hr and much lowered at 24 hr, which was also shown in Arashiro et al. (2016, ACP). Their cellular materials were collected 24 hr post-exposure, so they might have missed the peak. How do the authors justify the 24 hr post exposure time? Did they conduct a series of time course experiments to see where things might peak in terms of cellular response? The authors and readers need to realize you may only captured 1 slice in time in how the cells responded.**

We have modified the manuscript to clarify this discussion. Specifically, we include an example of a gene whose fold change was similar between the two types of SOA studies in Lin et al. (2016) and discuss how that gene is related to the inflammatory cytokines measured in this study. Oxidative stress plays a crucial role in the inflammatory process, and as such, the oxidative stress related genes measured in Lin et al. (2016) may influence cytokine production. We thank the reviewer for pointing out Lin et al. (2017) and have cited the paper accordingly. We are aware that cytokine production peaks at different time points for different cytokines. In our case, TNF-α peaks around 4 hrs, while IL-6 peaks much later at 24 hrs (Haddad, 2001). We chose to measure both cytokines at the latter time point to allow comparison. Previous studies have shown that the level of TNF-α is sufficiently high at the latter time point for accurate determination (Haddad, 2001; Matsunaga et al., 2001). The manuscript has been modified to include this justification as well.

Line 93: "However, the cellular exposure studies involving SOA focused on SOA formed from a single precursor and included different measures of response (e.g. ROS/RNS,

inflammatory biomarkers, gene expression, etc.) (Arashiro et al., 2016; Lund et al., 2013; McDonald et al., 2010; McDonald et al., 2012; Baltensperger et al., 2008; Lin et al., 2017)."

Line 318: "…the fold change of several genes reported in Lin et al. (2016) are actually similar (e.g., *ALOX12, NQO1)*. Several of these genes directly affect the production of inflammatory cytokines measured in this study. For instance, studies have observed that arachidonate 12-lipoxygenase (*ALOX12*) products induce the production of both TNF-α and IL-6 in macrophages (Wen et al., 2007). As such, a similar response level regardless of SOA formation condition may be observed depending on the biological endpoints measured. Thus, it is possible that the inflammatory cytokines measured in this study are involved in pathways concerning those genes, resulting in a similar response level regardless of SOA formation condition."

Line 231: "following manufacturer's specifications (ThermoFisher). This time point was chosen to enable comparison with ROS/RNS levels (also measured at 24 hrs, optimized in Tuet et al. (2016)) and to ensure a high signal for both cytokines. Previous literature have shown that TNF-α and IL-6 production peak around 4 and 24 hrs, respectively (Haddad, 2001). However, while TNF-α production peaks earlier, the signal at 24 hrs is well above the detection limit of the assay, and previous studies have utilized this time point to measure both cytokines (Haddad, 2001; Matsunaga et al., 2001). Nonetheless, it should be noted that these measurements represent a single time point in the cellular response.…"

8) **Line 305: what kind of chemical structure do they mean here?**

Thank you for the comment. We have modified the manuscript to clarify.

Line 340: "These observations further imply that the chemical structures (e.g., carbon chain lengths and functionalities) of oxidation products…"

9) **Line 322-329: I am not sure about the insertion of pentadecane oxidation products to the membrane. They should at least provide some references to support such a statement. I would expect some cellular response, specifically cytotoxicity, from these products since they are detergent like, which could potentially rupture the cell membrane. Did they see cell death from MTT data for pentadecane oxidation products?**

Thank you for the suggestion. We have included references to support this hypothesis. We did not observe decreases in cellular metabolic activity as measured by the MTT assay (mentioned in lines 282 – 286 in the revised manuscript).

Line 363: "…could potentially insert into the cell membrane (Loza et al., 2014), as previous studies have shown that fatty acids can feasibly insert into the cell membrane bilayer (Khmelinskaia et al., 2014; Cerezo et al., 2011)."

10) **The mechanism of PAH-DNA adduct formation is well known through metabolic activation to diol epoxides. This is not mentioned at all in current discussion.**

We mentioned the formation of DNA adducts briefly in the section on naphthalene SOA (lines 420 – 424). The specific mechanism by which these adducts are formed is beyond the scope of this study, but would be interesting to investigate in future studies.

11) **Statistical Analysis: One more critical comment relates to the authors statistical analysis. Where are their linear regression results and the associated p values? Also, with multiple groups, one-way ANOVA should be used instead of student's t test to get p-values (same idea as the increasing type I error with multiple testing). Lastly, when they talked about the trend, I didn't see any statistical support to differentiate between groups. Are the results really statistically significant?**

Based on the reviewer's comment, we believe the trend referenced refers to Fig. 3. The Pearson's correlation coefficient is given in the original figure. For clarity, we have modified the manuscript and figure caption to reflect that correlations were evaluated using a 95% confidence interval. Since only two variables (cellular response and bulk aerosol composition, e.g., ROS/RNS and $\overline{OS}_c$) were tested, the student's t-test and one-way ANOVA are actually equivalent (Park, 2009).

Line 436: "Nevertheless, a significant correlation ($p < 0.05$) was observed…"

Line 592: "…colored by SOA system. * indicates significance, $p < 0.05$."

12) **I'm curious why the authors didn't gravimetrically weigh the filters before and after sampling to insure actual mass on filter for dose-response purposes? If you use the SMPS, you must make assumptions about density to calculate the mass. How was density accurately determined if you did use that approach? Was the SMPS sheath flow conditioned to the appropriate RH used in the chamber?**

Mass loadings were low for isoprene and pentadecane SOA. To be consistent, we choose to determine mass by integrating the SMPS volume concentrations for all SOA systems. An aerosol density of 1 g cm^{-3} was assumed to facilitate comparison between studies, since SOA density varies with precursor identity and formation condition. We have added this clarification to the manuscript. For all experiments, the SMPS was connected to the chamber for 2-3 hrs before the start of the experiment to condition the recirculating sheath flow.

Line 201: "…multiplying by the total volume of air collected. SMPS volume concentrations were converted to mass concentrations by assuming a density of 1 g cm^{-3} to facilitate comparison between studies…"

13) **Related to #12 above, were extraction efficiencies of aerosol mass from the filters determined by spiking them with representative internal standards? Extracting filters with cell media l may not actually remove a lot of materials (such as oligomers of SOA) from the filters. Why wasn't organic solvents used, then dried, and then the dried extracts reconstituted with cell media for the exposures? Toxicologists might find your dosing completely uncertain as its hard to gauge how well you removed the SOA from the filters without this information. This is a very important point for Figures like Figure 3. The AMS sees most of the SOA mass but filter extractions may not actually remove all of it for the exposure assessment done here.**

Extraction efficiencies were not measured in this study. While different extraction methods are known to result in different constituents being extracted from the PM sample, there are limitations for each method. These are discussed in a recent publication by Gao et al. (2017). For example, using an organic solvent and drying the extract for reconstitution may result in loss of unstable constituents. For this study, we chose an extraction method best suited for cellular exposure.

14) **I have a curiosity question. Did the authors observe brown color on some of their filters (like from naphthalene SOA or isoprene SOA)? If so, did you seen any trends with brown carbon and your toxicological endpoints?**

We only observed brown color on our naphthalene SOA filters. We did not measure brown carbon in this study.

15) **Line 81-84: This seems to be an incomplete sentence or poorly worded sentence. Please revise.**

Thank you for your comment. We have modified the sentence.

Line 91: "However, the cellular exposure studies involving SOA focused on SOA formed from a single precursor and included different measures of response…"

References:

Arashiro, M., Lin, Y. H., Sexton, K. G., Zhang, Z., Jaspers, I., Fry, R. C., Vizuete, W. G., Gold, A., and Surratt, J. D.: In Vitro Exposure to Isoprene-Derived Secondary Organic Aerosol by Direct Deposition and its Effects on COX-2 and IL-8 Gene Expression, Atmos. Chem. Phys. Discuss., 2016, 1-29, 10.5194/acp-2016-371, 2016.

Baltensperger, U., Dommen, J., Alfarra, R., Duplissy, J., Gaeggeler, K., Metzger, A., Facchini, M. C., Decesari, S., Finessi, E., Reinnig, C., Schott, M., Warnke, J., Hoffmann, T., Klatzer, B., Puxbaum, H., Geiser, M., Savi, M., Lang, D., Kalberer, M., and Geiser, T.: Combined determination of the chemical composition and of health effects of secondary organic aerosols: The POLYSOA project, J. Aerosol Med. Pulm. Drug Deliv., 21, 145-154, 10.1089/jamp.2007.0655, 2008.

Cerezo, J., Zúñiga, J., Bastida, A., Requena, A., and Cerón-Carrasco, J. P.: Atomistic Molecular Dynamics Simulations of the Interactions of Oleic and 2-Hydroxyoleic Acids with Phosphatidylcholine Bilayers, The Journal of Physical Chemistry B, 115, 11727-11738, 10.1021/jp203498x, 2011.

Chen, C. Y., Peng, W. H., Tsai, K. D., and Hsu, S. L.: Luteolin suppresses inflammation-associated gene expression by blocking NF-kappa B and AP-1 activation pathway in mouse alveolar macrophages, Life Sci., 81, 1602-1614, 10.1016/j.lfs.2007.09.028, 2007.

Gao, D., Fang, T., Verma, V., Zeng, L., and Weber, R.: A method for measuring total aerosol oxidative potential (OP) with the dithiothreitol (DTT) assay and comparisons between an urban and roadside site of water-soluble and total OP, Atmos. Meas. Tech. Discuss., 2017, 1-25, 10.5194/amt-2017-70, 2017.

Haddad, J. J.: L-buthionine-(S,R)-sulfoximine, an irreversible inhibitor of gamma-glutamylcysteine synthetase, augments LPS-mediated pro-inflammatory cytokine biosynthesis: evidence for the implication of an I kappa B-alpha/NF-kappa B insensitive pathway, Eur. Cytokine Netw., 12, 614-624, 2001.

Kamimura, D., Ishihara, K., and Hirano, T.: IL-6 signal transduction and its physiological roles: the signal orchestration model, in: Reviews of Physiology, Biochemistry and Pharmacology, Springer Berlin Heidelberg, Berlin, Heidelberg, 1-38, 2004.

Khmelinskaia, A., Ibarguren, M., de Almeida, R. F. M., López, D. J., Paixão, V. A., Ahyayauch, H., Goñi, F. M., and Escribá, P. V.: Changes in Membrane Organization upon Spontaneous Insertion of 2-Hydroxylated Unsaturated Fatty Acids in the Lipid Bilayer, Langmuir, 30, 2117-2128, 10.1021/la403977f, 2014.

Kuznetsov, A. V., Kehrer, I., Kozlov, A. V., Haller, M., Redl, H., Hermann, M., Grimm, M., and Troppmair, J.: Mitochondrial ROS production under cellular stress: comparison of different detection methods, Analytical and Bioanalytical Chemistry, 400, 2383-2390, 10.1007/s00216-011-4764-2, 2011.

Lin, Y.-H., Arashiro, M., Martin, E., Chen, Y., Zhang, Z., Sexton, K. G., Gold, A., Jaspers, I., Fry, R. C., and Surratt, J. D.: Isoprene-Derived Secondary Organic Aerosol Induces the Expression of Oxidative Stress Response Genes in Human Lung Cells, Environmental Science & Technology Letters, 3, 250-254, 10.1021/acs.estlett.6b00151, 2016.

Lin, Y.-H., Arashiro, M., Clapp, P. W., Cui, T., Sexton, K. G., Vizuete, W., Gold, A., Jaspers, I., Fry, R. C., and Surratt, J. D.: Gene Expression Profiling in Human Lung Cells Exposed to Isoprene-Derived Secondary Organic Aerosol, Environmental Science & Technology, 10.1021/acs.est.7b01967, 2017.

Lund, A. K., Doyle-Eisele, M., Lin, Y. H., Arashiro, M., Surratt, J. D., Holmes, T., Schilling, K. A., Seinfeld, J. H., Rohr, A. C., Knipping, E. M., and McDonald, J. D.: The effects of alpha-pinene versus toluene-derived secondary organic aerosol exposure on the expression of markers associated with vascular disease, Inhal. Toxicol., 25, 309-324, 10.3109/08958378.2013.782080, 2013.

Matsunaga, K., Klein, T. W., Friedman, H., and Yamamoto, Y.: Involvement of Nicotinic Acetylcholine Receptors in Suppression of Antimicrobial Activity and Cytokine Responses of Alveolar Macrophages to Legionella pneumophila Infection by Nicotine, The Journal of Immunology, 167, 6518-6524, 10.4049/jimmunol.167.11.6518, 2001.

Mbawuike, I. N., and Herscowitz, H. B.: MH-S, a murine alveolar macrophage cell line: morphological, cytochemical, and functional characteristics, Journal of Leukocyte Biology, 46, 119-127, 1989.

McDonald, J. D., Doyle-Eisele, M., Campen, M. J., Seagrave, J., Holmes, T., Lund, A., Surratt, J. D., Seinfeld, J. H., Rohr, A. C., and Knipping, E. M.: Cardiopulmonary response to inhalation of biogenic secondary organic aerosol, Inhal. Toxicol., 22, 253-265, 10.3109/08958370903148114, 2010.

McDonald, J. D., Doyle-Eisele, M., Kracko, D., Lund, A., Surratt, J. D., Hersey, S. P., Seinfeld, J. H., Rohr, A. C., and Knipping, E. M.: Cardiopulmonary response to inhalation of secondary organic aerosol

derived from gas-phase oxidation of toluene, Inhal. Toxicol., 24, 689-697, 10.3109/08958378.2012.712164, 2012.

Mukherjee, S., Chen, L.-Y., Papadimos, T. J., Huang, S., Zuraw, B. L., and Pan, Z. K.: Lipopolysaccharide-driven Th2 Cytokine Production in Macrophages Is Regulated by Both MyD88 and TRAM, J. Biol. Chem., 284, 29391-29398, 10.1074/jbc.M109.005272, 2009.

Oberdörster, G., Ferin, J., Gelein, R., Soderholm, S. C., and Finkelstein, J.: Role of the alveolar macrophage in lung injury: studies with ultrafine particles, Environmental Health Perspectives, 97, 193-199, 1992.

Oberdörster, G.: Lung Dosimetry: Pulmonary Clearance of Inhaled Particles, Aerosol Sci. Technol., 18, 279-289, 10.1080/02786829308959605, 1993.

Park, H. M.: Comparing group means: t-tests and one-way ANOVA using Stata, SAS, R, and SPSS, 2009.

Rosenthal, N., and Brown, S.: The mouse ascending: perspectives for human-disease models, Nat Cell Biol, 9, 993-999, 2007.

Sankaran, K., and Herscowitz, H. B.: Phenotypic and functional heterogeneity of the murine alveolar macrophage-derived cell line MH-S, Journal of Leukocyte Biology, 57, 562-568, 1995.

Sullivan, K. E., Cutilli, J., Piliero, L. M., Ghavimi-Alagha, D., Starr, S. E., Campbell, D. E., and Douglas, S. D.: Measurement of Cytokine Secretion, Intracellular Protein Expression, and mRNA in Resting and Stimulated Peripheral Blood Mononuclear Cells, Clinical and Diagnostic Laboratory Immunology, 7, 920-924, 2000.

Takao, K., and Miyakawa, T.: Genomic responses in mouse models greatly mimic human inflammatory diseases, Proceedings of the National Academy of Sciences, 112, 1167-1172, 10.1073/pnas.1401965111, 2015.

Tuet, W. Y., Fok, S., Verma, V., Tagle Rodriguez, M. S., Grosberg, A., Champion, J. A., and Ng, N. L.: Dose-dependent intracellular reactive oxygen and nitrogen species (ROS/RNS) production from particulate matter exposure: comparison to oxidative potential and chemical composition, Atmos. Environ., 144, 335-344, http://dx.doi.org/10.1016/j.atmosenv.2016.09.005, 2016.

Tuet, W. Y., Chen, Y., Xu, L., Fok, S., Gao, D., Weber, R. J., and Ng, N. L.: Chemical oxidative potential of secondary organic aerosol (SOA) generated from the photooxidation of biogenic and anthropogenic volatile organic compounds, Atmos. Chem. Phys., 17, 839-853, 10.5194/acp-17-839-2017, 2017.

Wen, Y., Gu, J., Chakrabarti, S. K., Aylor, K., Marshall, J., Takahashi, Y., Yoshimoto, T., and Nadler, J. L.: The Role of 12/15-Lipoxygenase in the Expression of Interleukin-6 and Tumor Necrosis Factor-α in Macrophages, Endocrinology, 148, 1313-1322, 10.1210/en.2006-0665, 2007.

Witkamp, R., and Monshouwer, M.: Signal transduction in inflammatory processes, current and future therapeutic targets: A mini review, Veterinary Quarterly, 22, 11-16, 10.1080/01652176.2000.9695016, 2000.

Wright, C. J., Agboke, F., Muthu, M., Michaelis, K. A., Mundy, M. A., La, P., Yang, G., and Dennery, P. A.: Nuclear Factor-κB (NF-κB) Inhibitory Protein IκBβ Determines Apoptotic Cell Death following Exposure to Oxidative Stress, J. Biol. Chem., 287, 6230-6239, 10.1074/jbc.M111.318246, 2012.