In this study we compared a “baseline” condition of uncontrolled diesel engine exhaust (DEE) emissions generated with current (circa 2003) certification fuel to an emissions-reduction (ER) case with low sulfur fuel and a catalyzed particle trap. Lung toxicity assessments (resistance to viral infection, lung inflammation, and oxidative stress) were performed on mice (C57Bl/6) exposed by inhalation (6 hr/day for 7 days). The engine was operated identically (same engine load) in both cases, and the inhalation exposures were conducted at the same exhaust dilution rate. For baseline DEE, this dilution resulted in a particle mass (PM) concentration of approximately 200 μg/m³ PM, whereas the ER reduced the PM and almost every other measured constituent (except nitrogen oxides (NOₓ)) to near background levels in the exposure atmospheres. These measurements included PM, PM size distribution, PM composition (carbon, ions, elements), NOₓ, carbon monoxide, speciated/total volatile hydrocarbons, and several classes of semi-volatile organic compounds. After exposure concluded, one group of mice was immediately sacrificed and assessed for inflammation and oxidative stress in lung homogenate. Another group of mice were intratracheally instilled with respiratory syncytial virus (RSV), and RSV lung clearance and inflammation was assessed 4 days later. Baseline DEE produced statistically significant biological effects for all measured parameters. The use of low sulfur fuel and a catalyzed trap either completely or nearly eliminated the effects. Key words: diesel exhaust, emissions reduction, health effects, metals, organic carbon, particulate matter health effects. Environ Health Perspect 112:1307-1312 (2004). doi:10.1289/ehp.7059 available via http://dx.doi.org/ [Online 7 July 2004]
conditions summarized in Table 1) conducted at the same dilution ratio (620:1). This dilution ratio was determined by the dilution required to obtain 200 µg/m³ PM for the baseline DEE, which is not the minimum concentration for which we have observed effects (for RSV infection), but it is a concentration for which strongly significant effects have been reported to occur (Harrod et al. 2003a, 2003b). We conducted measurements of the biological responses and composition of the exposure atmospheres identically for the two exposures as described below.

**Exhaust generation.** The exhaust generation/exposure system has been described previously (McDonald et al. 2004a). Briefly, DEE was produced by a 5500-watt single cylinder diesel engine generator (Model YDG 5500E; Yanmar, Osaka, Japan) that contains a 406-cc displacement air-cooled engine. Engine oil (15/40-weight, Rotella T, Shell, Houston, TX) was changed immediately prior to each 1-week exposure. The baseline DEE was generated using number 2 diesel certification fuel (15/40-weight, Rotella T, Shell, Houston, TX) was changed immediately prior to each exposure. (15/40-weight, Rotella T, Shell, Houston, TX) was changed immediately prior to each exposure. No. 2 Cert, number 2 diesel certification fuel.

**DEE + ER High load ECD1 Catalyzed trap Same dilution as DEE**

Table 1.

| Engine operation | Fuel                  | After-treatment | Dilution target          |
|------------------|-----------------------|-----------------|--------------------------|
| DEE              | High load             | No. 2 Cert      | Same dilution as DEE     |
| DEE + ER         | High load             | ECD1            | Same dilution as DEE     |

Table 2. Properties of the number 2 diesel certification fuel (No. 2 Cert) and the ECD1 low sulfur fuel.

|                        | No. 2 Cert | ECD1 |
|------------------------|------------|------|
| API gravity            | 35.8       | 35.3 |
| Specific gravity 60/60 | 0.85       | 0.85 |
| Viscosity              | 2.4        | 2.8  |
| Sulfur (ppm)           | 371        | 14   |
| Aromatics (volume %)   | 29         | 32   |
| Cetane index           | 47.6       | 46.1 |
| Cetane number          | 47.3       | 47.7 |

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API gravity is an arbitrary scale representing the gravity of liquid petroleum; cetane number is a measure of ignition quality of diesel fuel; and cetane index is an approximation of cetane number based on the API gravity and mid-boiling point of a fuel.
(reverse transcriptase-polymerase chain reaction). RSV mRNA transcripts were ratioed to amplified β-actin (internal control) mRNA levels to account for intersample variability in mRNA isolation and amplification. RSV was thus compared in each treatment group as the average of the RSV/β-actin responses for each animal.

Lung cross-sections for histopathology of the RSV infected mice were obtained approximately 500 µm caudal to the junction of the mainstream bronchus, stained with hematoxylin and eosin, and analyzed by a pathologist under light microscopy. The pathologist scored (0–4 scale) the levels of inflammation in the airways and vessels without knowledge of the origin of the sample.

Inflammation. We measured inflammatory signaling proteins (cytokines) in homogenates of the right caudal and middle lung lobes (six per group). Immediately after sacrifice, lungs were frozen in 1 mL Dulbecco's phosphate-buffered saline (PBS) with a cocktail of proteinase inhibitors. Before analysis, lungs were removed from the freezer and brought to room temperature. Lungs were homogenized for 1 min at full speed in a TissueMizer (Tekmar, Mason, OH) and centrifuged for 5 min at 14,000 × g. The supernatant was transferred to clean microfuge tubes and kept on ice. Cytokines [tumor necrosis factor-α (TNF-α), interferon-γ (IFN-γ), interleukin-6 (IL-6)] were determined (two measurements for each cytokine for each sample) by ELISA using commercially available mouse analysis kits (Biosource International, Camarillo, CA). To normalize the cytokine measurements to total protein, supernatants were diluted to 2 mg/mL in PBS and total protein was assayed by the Coomassie-dye binding assay (Pierce, Rockford, IL) with bovine serum albumin as the standard.

HO-1. We measured HO-1 induction in lung homogenate by Western blotting using 30 µg of a sample of lung homogenate supernatant (prepared as described above for inflammatory indicators) in 1x Laemmli sample buffer containing 25 mM dithiothreitol. Samples were heated for 5 min at 95°C and resolved on a 15% polyacrylamide gel. Proteins were electroblotted to polyvinylidene difluoride membranes. The blots were blocked with 5% nonfat dry milk in Tris-buffered saline with 0.1% Tween-20, and incubated with 1 µg/mL polyclonal anti-HO-1 (Calbiochem, San Diego, CA) followed by 1 µg/mL horseradish peroxidase-labeled goat anti-rabbit IgG. HO-1 was detected by chemiluminescent reagents (ECL, Amersham, Piscatway, NJ) exposure of BioMax Film (Kodak, Rochester, NY) and quantified by densitometry as described by Li et al. (2003).

Statistical analysis. We used analysis of variance (ANOVA) to evaluate DEE and DEE + ER responses relative to values from concurrent control groups. Levene's test (Levene 1960) was first performed to evaluate the appropriateness of the standard ANOVA assumption of equality of variances among experimental group responses. These tests showed that for all end points except lung histopathology, there was significant evidence of inequality of variances (p < 0.05). To address this problem, we used a weighted least-squares analysis (Neter et al. 1996) using the reciprocals of the variances in experimental groups as weights. F-test contrasts (Searle 1971) were used to compare DEE and DEE + ER responses with baseline values in concurrent control groups. Because the baseline values for the two control groups differed substantially for some end points, reported means and SEs were scaled by the mean values from concurrent control groups. Statistical significance was assessed at p = 0.05 and p = 0.01; however, several treatment groups showed p-values much lower than this. Calculations were performed using SAS software (SAS Institute, Cary, NC).

Table 3. Summary of measurements, measurement conditions, and analytical techniques used to characterize exposure atmosphere composition.

| Analysis                  | Collection device          | Collection media    | Sample flow rate (L/min) | Analytical instrument |
|---------------------------|----------------------------|---------------------|--------------------------|-----------------------|
| Particle mass             |                            |                     |                          |                       |
| NO₃                      | Aluminum in-line filter holder | TIGF filter       | 10                       | MB                    |
|                           |                            |                     |                          | NA                    |
| CO                        |                            | Photocoustic analyzer | NA                      | NA                    |
| Organic/elemental carbon  | Aluminum in-line filter holder | Quartz filter      | 20                       | TOR                   |
| (sulfate/nitrate/ammonium) | Aluminum in-line filter holder | Quartz filter      | 20                       | IC                    |
| Metals and other elements | Teflon in-line filter holder         | Teflon filter      | 20                       | ICPMS                 |
| Speciated organic compounds | Volatile organic sampler | Electropolished canister | 0.1                    | GCFD                  |
| Volatile hydrocarbons (C₄-C₇) | Volatile organic sampler | DNPH cartridge      | 0.3                      | LC/UV                 |
| Semivolatile/aromatic/alkane | Filter/PUF sampler | TIGF filter/PUF/XAD/PUF | 60                      | GCMS                  |
| Size distribution         |                            |                     |                          |                       |
| 0.05–10 µm particle mass distribution | MOUDI impactor | Aluminum             | 30                       | MB                    |
| 0.02–0.7 µm particle number distribution | SMPS | NA                  | 0.3                      | NA                    |

Abbreviations: DNPH, dinitrophenylhydrazine; GCFD, gas chromatography flame ionization detection; GCMS, gas chromatography/mass spectrometry; IC, ion chromatography; ICPMS, inductively coupled plasma mass spectrometry; LC/UV, liquid chromatography/ultraviolet detection; MB, microbalance; NA, not applicable; PUF, polyurethane foam; SMPS, scanning mobility particle sizer; TIGF, Teflon impregnated glass fiber; TOR, thermal/optical reflectance; XAD, XAD resin.

*Analyses conducted at the Desert Research Institute, Reno, NV. *Analyses conducted at the Carlsbad Environmental Monitoring and Research Center, Carlsbad, NM. *Source: MSP Corp, St. Paul, MN.
elements, especially calcium and zinc, which are lube oil and fuel additives (Docekal et al. 1992), were observed in DEE and decreased substantially with ER.

The ER atmosphere had low quantities of both gases and PM, many of which were in the same range as the concentrations observed in the control atmosphere. However, several individual organic compounds were present in DEE and DEE + ER at concentrations significantly above background. Acetylene, a compound that has been used in ambient source apportionment studies as an indicator of mobile source emissions, was enriched in DEE but reduced in DEE + ER to background levels, as was also true for most of the aromatic [including polycyclic aromatic hydrocarbons (PAHs)] and alkene (including 1,3-butadiene) compounds. However, removal of the carbonyl compounds, especially formaldehyde and acetaldehyde, was much less efficient (~ 17–45%).

**Lung toxicity.** The results of the cytokine/ HO-1 up-regulation in noninfected animals and the lung viral burden/histopathology scores after exposure/RSV infection are shown in Figures 2–4. The DEE exposure resulted in statistically significant differences from control exposed animals for all measured lung responses, but the DEE + ER exposure resulted in no significant differences from control for any biological measurement. Figure 2 summarizes the lung viral burden and lung histopathology of virus-infected mice. As expected from previous studies (Harrod et al. 2003a, 2003b), DEE exposure significantly (p = 0.002) decreased the clearance of virus from the lungs and significantly increased (p = 0.003) the histopathology scores. Figure 3 shows the increase in cytokines. All cytokines were significantly elevated above control values in the DEE group, but not in the ER group. HO-1, the oxidative stress response indicator, also significantly increased after DEE exposure but not after DEE + ER exposure (Figure 4).

**Discussion**

The present study showed that implementation of a low sulfur fuel/catalyzed trap combination decreased the concentration of most components of emissions and diminished the biological effects of DEE on viral clearance, inflammation, and oxidative stress. These findings suggest that this type of ER technology may have substantial health benefits. Of course, ER technologies may vary considerably, and it is not known how broadly these results might apply to other technologies.

The ER case significantly decreased nearly every measured exposure constituent except NOx to background levels. Except for a few volatile organic compounds and elements, the ER and control exposure atmosphere had similar low concentrations of both gases and PM. These similarities suggest that a portion of the constituents observed in the DEE exposure atmosphere downstream of the trap was contributed by background in the dilution air or by the rodents themselves. Although the dilution air was pretreated by filtration

### Table 4. Comparative composition of DEE, DEE + ER, and control (clean air) exposure chambers.

| Analyte or chemical class | Control | DEE | DEE + ER | DEE vs. DEE + ER (percent decrease) |
|---------------------------|---------|-----|---------|-------------------------------------|
| NOx (ppm)                 | <0.04   | 2.1 | 1.9     | 10                                  |
| Nonmethane volatile organic (µg/m³) | 54.4 | 162.3 | 63.2 | 61                                  |
| CO (ppm)                  | 0.3     | 2.0 | 0.2     | 90                                  |
| Particle mass (µg/m³)     | 5.1     | 235.7 | 7.0 | 99                                  |
| Particle composition      |         |     |         |                                     |
| Black (elemental) carbon (µg/m³) | 0.0 | 200.3 | 0.0 | 100                                 |
| Organic carbon (µg/m³)    | 4.5     | 39.9 | 4.2     | 90                                  |
| Nitrate (µg/m³)           | 0.5     | 0.2 | 0.0     | 100                                 |
| Sulfate (µg/m³)           | 0.2     | 0.0 | 0.0     | 100                                 |
| Ammonium (µg/m³)          | 0.0     | 0.1 | 0.1     | 100                                 |
| Sum of elements (µg/m³)   | 0.0     | 2.1 | 0.7     | 67                                  |
| Speciated organic classes |         |     |         |                                     |
| Sum carbon (µg/m³)        | 5.3     | 37.7 | 21.9 | 42                                  |
| Acetylene (alkyne) (µg/m³) | 0.5 | 16.7 | 0.4 | 98                                  |
| Sum of C₂-C₄ alkanes (µg/m³) | 26.6 | 27.7 | 21.1 | 24                                  |
| Sum of C₅-C₉ alkanes (µg/m³) | 3.0 | 31.7 | 1.6 | 95                                  |
| Sum of volatile aromatics (µg/m³) | 8.5 | 25.2 | 13.2 | 48                                  |
| Sum of C₁₀-C₂₀ alkanes (µg/m³) | 6.8 | 26.7 | 9.6 | 64                                  |
| Sum of naphthalenes (µg/m³) | 1.0 | 4.7 | 1.0 | 80                                  |
| Sum of phenanthrenes (µg/m³) | 0.5 | 6.2 | 0.4 | 93                                  |
| Sum of other SVOC PAHs (µg/m³) | 0.4 | 1.7 | 0.6 | 65                                  |
| Sum of particulate PAHs (ng/m³) | 0.0 | 23.0 | 0.0 | 100                                 |
| Sum of Oxy-PAHs (µg/m³)   | 0.05    | 1.29 | 0.08 | 94                                  |
| Select speciated organics |         |     |         |                                     |
| Formaldehyde (µg/m³)      | 1.8     | 14.1 | 11.6 | 17                                  |
| Acetaldehyde (µg/m³)      | 1.5     | 17.0 | 9.4 | 45                                  |
| Benzenaldehyde (µg/m³)    | 0.5     | 1.9 | 0.3 | 84                                  |
| Ethene (µg/m³)            | 0.5     | 25.9 | 0.5 | 98                                  |
| 1,3-Butadiene (µg/m³)     | 0.0     | 2.2 | 0.0 | 100                                 |
| Benzene (µg/m³)           | 0.4     | 4.5 | 0.2 | 95                                  |
| Pyrene (µg/m³)            | 0.03    | 0.34 | 0.02 | 93                                  |
| Benz[a]pyrene (ng/m³)     | 0.00    | 0.08 | 0.00 | 100                                 |
| Dibenzothiophene (µg/m³)  | 0.06    | 0.10 | 0.05 | 43                                  |
| 9-Fluorenone (µg/m³)      | 0.05    | 1.07 | 0.05 | 95                                  |
| Xanthone (µg/m³)          | 0.00    | 0.12 | 0.00 | 100                                 |
| Select elements           |         |     |         |                                     |
| Zinc (µg/m³)              | −0.01   | 0.71 | 0.07 | 90                                  |
| Calcium (µg/m³)           | −0.03   | 0.41 | 0.22 | 47                                  |
| Iron (µg/m³)              | −0.02   | 0.24 | 0.07 | 71                                  |
| Potassium (µg/m³)         | −0.01   | 0.16 | 0.04 | 73                                  |
| Silicon (µg/m³)           | −0.05   | 0.26 | 0.07 | 73                                  |
| Magnesium (µg/m³)         | 0.00    | 0.08 | 0.03 | 58                                  |
| Copper (µg/m³)            | 0.01    | 0.06 | 0.05 | 11                                  |
| Lead (µg/m³)              | 0.01    | 0.07 | 0.02 | 74                                  |

Abbreviations: PAHs, polycyclic aromatic hydrocarbons; SVOC, semivolatile organic compound.

*Concentrations not obtained during exposures due to analyzer failure; data was obtained from an identical fuel and engine operation exposure study.
through HEPA and charcoal filters, these filters do not efficiently remove CO or methane. The contribution of rodent respiration and excretion to the composition whole-body exposure atmospheres has been discussed previously (McDonald et al. 2004b). Among the compounds that are contributed by respiration and background are the C2-C12 alkanes, for which there were similar concentrations among all of the exposure atmospheres (including DEE). Similar to the control chamber, the small PM component of the DEE + ER exposure atmosphere was nearly 100% organic carbon, which was likely contributed by the rodents (dander, exhaled organics, etc.).

Figure 3. Inflammatory signaling proteins measured in lung homogenates of mice exposed to clean air (DEE control, DEE + ER control), DEE, or DEE + ER. Error bars indicate SE. DEE and DEE + ER exposures were conducted at equivalent dilutions. 

* $p = 0.003$, ** $p = 0.036$, and *** $p = 0.001$, compared to control.

Despite the contribution of rodents and dilution air to the exposure atmospheres, several individual organic compounds were present in DEE and DEE + ER at concentrations significantly above background, indicating a variable efficiency of removal. Acetylene, a compound that has been used in ambient source apportionment studies as an indicator of mobile source emissions, was enriched in DEE but reduced in the DEE + ER to background levels. This also occurred for most of the aromatic (including PAH) and alkene (including 1,3-butadiene) compounds. However, removal of the carbonyl compounds, especially formaldehyde and acetaldehyde, was much less efficient (~17–45%). These findings agree with previous reports comparing a baseline DEE to DEE with low sulfur fuel and a trap (Durbin et al. 2003), where the ER was most efficient at removing acetylene, moderately efficient at removing alkenes/aromatics, and poor at removing volatile carbonyls.

Although it provided an important first look at the effects of ER, this study had several limitations. First, the exhaust was not produced by an engine that would be used on-road. We previously demonstrated the usefulness of this model system by showing both similar composition (McDonald et al. 2004a) and similar biological responses (Harrod et al. 2003a) at selected operating conditions compared to DEE produced from a multicylinder diesel engine operated on a heavy-duty engine cycle. This model system was therefore considered adequate to show “proof of concept” or to develop testing protocols. However, the applicability of the present results to emissions generated from larger on-road and off-road engine systems needs to be confirmed. In addition, it may be important to assess the performance of a wider range of ER technologies operating under a variety of engine operation conditions. The high constant workload and new particle trap (emissions may change after trap “ages”) used in this study allowed the optimal performance of the ER. Under this condition, the emissions were substantially decreased. Rudell et al. (1996) reported that humans exposed to DEE from an idling vehicle both with and without a ceramic particle trap (no catalyst) had inflammation (as assessed by increase in neutrophils and infiltration of alveolar macrophages into their airways) in both cases. In that study the ceramic trap removed only half of the particle count.

Although the results of the present study clearly demonstrated a near total mitigation of the effects of DEE exposure on retardation of viral clearance and pathology, inflammation, and oxidative stress, the results must be extrapolated to humans with caution. There is no direct evidence for the effect of DEE on human resistance to RSV infection, although RSV is certainly a pervasive human pathogen (Collins et al. 2001); proximity to heavy traffic has been associated with increased categories of respiratory illnesses that encompass viral infection (e.g., Romieu et al. 2002). The correspondence between responses of mice and humans can be questioned, but the use of mice as models for the pathophysiology of human RSV infection is widely accepted (Graham et al. 1988). The use of only one exposure concentration for DEE was another limitation of this study; however, we previously demonstrated that the effect of DEE inhalation on RSV clearance was concentration related (Harrod et al. 2003b). We believe that the single concentration, which is in the range of occupational exposures to DEE (e.g., McDonald et al. 2002) was adequate to explore the effects of the ER strategy.

The induction of respiratory inflammation by exposure to whole, diluted DEE and its partial mitigation by a particle trap (also at single concentrations) has been demonstrated in humans (Rudell et al. 1996). Although evidence for the role of oxidative stress in responses to DEE is derived largely from animal and in vitro studies, the induction of HO-1 stress response protein has been well documented in humans as an indirect indicator of oxidative stress (Morse and Choi 2002). In this study we did not attempt to fully characterize the nature and magnitude of DEE-induced oxidative damage.

The approach used in this study had the advantage of being a) by inhalation, b) short-term, and c) relevant to known public health hazards. It provided data using contemporary chemical and physical characterization techniques coupled to three biological response categories that are relevant to human health endpoints observed by laboratory (e.g., Rudell et al. 1996; Sydborn 2001) and epidemiology studies (e.g., Nicolai et al. 2003; Romieu et al. 2002; Samet et al. 2000; Van Vliet et al. 1997). The present study illustrates one approach to the challenge posed to the scientific and regulatory community to develop appropriate testing protocols aimed at placing changing DEE health hazards in context. We did not assess several classes of health effects that may be of importance (e.g., tumor formation, cardiovascular toxicity, exacerbation of asthma/inflammation), including effects that are commonly studied after long-term exposure periods. The study included only a few of the biological responses that have been reported in response to DEE, but these are...
among the most sensitive (e.g., RSV end points respond to DEE diluted as low as 30 µg/m³).

The concordance in response among the biological end points lends confidence in the overall conclusions of decreased health hazard.

**Conclusions**

ER (low sulfur fuel/catalyzed trap) technology decreased or diminished the emissions and the toxicity of DEE. With ER in place there was no detectable black (elemental) carbon, particle organic carbon in the range of background air, and decreased (relative to uncontrolled emissions) concentrations of the elements. Nearly all-gaseous components (except NOₓ, which was only slightly reduced, and select carbonyls) were in the range of background air. Baseline DEE exposures (no emission controls) produced significant biological responses in all measured end points. These responses, including lung inflammation (response to lung injury), resistance to a viral infection, and induction of a lung oxidative stress indicator, were not observed with ER in place. These results indicate that the use of low sulfur fuel and a catalyzed trap markedly reduce the DEE health hazard associated with resistance to infection, inflammation, and oxidative stress.

**References**

Abdul-Khalek I, Kittelson DB, Brear F. 1998. Diesel Trap Performance: Particle Size Measurements and Trends. SAE Technical Paper 982599. Warrendale, PA: SAE International. Bagley ST, Baumgard KJ, Gratz LD, Johnson JH, Leddy DG. 1996. Applied Linear Statistical Models. 4th ed. Chicago:Irwin. Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner O. 2003. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. Eur Respir J 21:956–963.

Romieu I, Samet JM, Smith KR, Bruce N. 2002. Outdoor air pollution and acute respiratory infections among children in developing countries. J Occup Environ Med 44(7):640–649.

Levene H. 1960. Robust tests of equality of variances. In: Contributions to Probability and Statistics (Olkin I, ed). Stanford, CA:Stanford University Press, 278–292.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002a. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002b. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002c. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002d. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002e. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002f. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002g. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002h. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002i. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002j. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002k. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002l. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002m. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002n. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002o. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002p. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002q. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002r. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002s. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.

Sawicki G, Nicklisch M, Sioutas C, Nel A. 2002t. Gasoline and diesel engine emissions. J Toxicol Environ Health 66:1113–1132.