Chapter 6: Environmental Benefit

This chapter presents the methodology used by EPA to quantify the emission reduction benefits that would be realized through the proposed Phase2 HC+ NOx in-use emission standards for small SI engines. Benefits, in terms of HC+NOx emission reductions, are presented in the form of aggregate benefits by engine class. These benefits are estimated in terms of future nationwide emission reductions not including California from affected small SI engines used in a variety of equipment types. Estimated benefits illustrate the potential future effect of the proposed standard on the emission inventory. Air quality benefits are discussed qualitatively for all pollutants.

Many of the detailed results discussed below are presented in separate tables included in Appendix F - Supplementary Tables and Chapter 9 (in-use emission factors). In addition a detailed description of the Nonroad Small Engine Emissions Model (NSEEM) is available in the form of a memo to Docket A96-55.

6.1.   Estimated Emissions Reductions

To estimate the average annual emissions at baseline (Phase 1), EPA calculated the tons per year estimates based on revised Phase 1 Emission Factors. The in-use factors have now been determined as a multiplicative rather than an additive (as was the case for the Phase 1 rule-making) function of new engine emission factors and a deterioration factor which is a function of engine hours of use. As before, total emissions are calculated for each type of equipment using the equation :

$$MASS_{i,j} = N_{i,j} \times HP_{i,j} \times LOAD_i \times HOURS_i \times EF_{i,j}$$

In the above equation,

$N_{i,j}$ -   nationwide population of ith equipment type using engine j

$HP_{i,j}$ -   average rated horsepower of engine j used in equipment type i

$LOAD_i$ -   ratio (%) between average operational power output and rated power for the ith equipment type

$HOURS_i$ -   average annual hours of usage for the ith equipment type

$EF_{i,j}$ -   brake specific in-use emission rate (kilowatts/hr) for engine type j used in equipment i

$MASS_{i,j}$ -   annual nationwide emissions (grams) for the j th engine type used in equipment i

For the benefits analysis described here, EPA performed separate calculations for each of 12 major equipment categories, each one of which could be equipped with one or more of 14 different engine types with average power ratings as displayed in Table F-01. The 'All Other Equipment Category' is a major catch-all category which includes Shredders, Pressure Washers, etc. and Loose Distributed Engines. Population and activity information used to construct the Inventories relied predominately on data available in a commercially available marketing research data base that includes most types of nonroad equipment (1).

6.1.1. Aggregate HC+ NOx Reductions

The calculation of aggregate HC+ NOx reductions is described in this section. The source of data and/or methodology used for determining U.S. sales of small SI engine/equipment types, hours of use, average power rating and related equipment survival rates is described below. Along with estimated values for Phase I in-use emission rates and proposed Phase II in-use engine emissions standards, EPA has determined nationwide annual emissions under the baseline and controlled scenarios through calendar year 2026.

6.1.1.1. Sales--In order to estimate future emission totals, some projections of future populations of Phase1 and Phase2 controlled engines are needed. Because engines are introduced into the field through sales, sales figures for all engine/equipment types are needed both for the period prior to and after the standards go

into effect. For years between 1973 and 1995, sales of major categories of nonroad equipment have been reported by leading industry sources. The methodology used by EPA for future sales projections through year 2026 is described in Appendix B. These sales have been adjusted to exclude engines that are covered by California's lawn, garden, and utility engine rule.

The results of the analyses are summarized in Table F-02 in Appendix F, which represents both figures reported by PSR (1) as well as projected estimates by type of use i.e. Residential and Commercial. However, it should be recognized that, while national growth is measured at the level of the economy as a whole, growth in specific areas of the country is likely to vary from area to area in response to the specific demographic and commercial trends in those areas. These effects should be taken into account in estimating growth at the local level.

To account for the implementation of the proposed standard that would begin to take effect in year 2001, EPA distinguished between sales of pre- and post- control engines. Tables F-03 and F-04 present sales fractions used to determine sales before and after the implementation of the new standards.

6.1.1.2. **Annual Hours of Use**-- this number is simply the average yearly hours of use for a particular nonroad small engine application. The numbers used in NSEEM are taken from the 1992 version of the PSR PartsLink Data base(1) and as indicated in the PSR PartsLink Reference Guide are "typical annual hours of operation" for each application (Table F-05). The annual hours of use is a critical factor for developing the level of activity.

6.1.1.3. **Average Power Rating**-- the rated power of each engine is represented in kilowatts. The PSR PartsLink database designates a rated power for each engine for which the population number is provided. Engine rated power is also a critical input in the development of level of activity. As presented in Table F-01, the power ratings vary both by engine technology and the equipment type the engine is used into.

6.1.1.4. Survival Probabilities--In calculating emission reductions that are expected to occur during the life of an equipment, the emissions of whose engine are

controlled in response to the proposed standard, EPA relied on the estimates of survival rates presented by EEA to CARB (2). Table F-06 presents the parameter values *theta* and *b* that define the Weibull Cumulative distribution or scrappage function. These parameters were determined based on data given in Table 3-3 of the Jack Faucett Report (3) submitted to the EPA.

6.1.1.5. In-Service Population--By coupling the sales estimates and projections given in Table F-02 with the engine survival rate function described in F-06 and the pre- and post control sales fractions in Tables F-03 and F-04, EPA estimated the in-use populations from 1973 to 2026 for all equipment types and thereby the engines they are equipped with. In doing so, EPA distinguished between controlled and uncontrolled engines, so that the effect of the proposed Phase2 standards could be ascertained.

6.1.1.6. Emission Factors--The in-use emission factors for the pre-control (Phase 1) scenario were recalculated based on 1) revised new engine values obtained from EPA and CARB certification data and 2) a multiplicative deterioration factor determined to be a linear a function of SQRT(Hours of equipment usage). This constitutes a major departure from the methodology used during the Phase 1 rule-making. For the current (Phase 2) scenario, the new engine emission factor values were back-calculated using 1) the proposed in-use emission factors (Phase 2 standards) and 2) a multiplicative deterioration factor as defined for Phase 1 above. The pre-Phase I (original baseline) in-use emission factors remain unchanged and have been used as such for emissions calculations where applicable. The exhaust emission factors for HC, NOx and CO along with those for Fuel Consumption are displayed in Table F-07. The rows titled df constants that appear in the above Table are the values of the slope of the deterioration factor equation, which takes the form:

$$DF = 1 + constant*SQRT(Hours\ of\ use)$$

The determination of this function was based on analysis done on data provided by the engine manufacturers of NHH engines. As for HH engines, with the exception of Class IV 4-stroke engines, the deterioration factor function was assumed to be linear, of the form :

DF = 1 + constant*(Hours of use)

6.1.1.7. Emissions reductions -- EPA calculated baseline per-equipment emissions using revised pre-control emission factors obtained from Phase1 engine certification data. The in-use population estimates were generated by taking into account the pre-control sales mix (table F-03) and a survival function defined by constants as displayed in Table F-06. To obtain average annual per-equipment emissions for engines controlled to the levels required to comply with EPA's proposed emission standards , emissions were recalculated using post-control sales mix (Table F-04) and in-use emission factors (Table F-07).

Table F-08 presents total annual nationwide emissions from engines addressed in this proposal under both the baseline (Phase1) and the controlled (Phase2) scenario. These are shown graphically in Figure 4-01 below.

Figure 4-01

Total Exhaust HC+NOx (tpy)
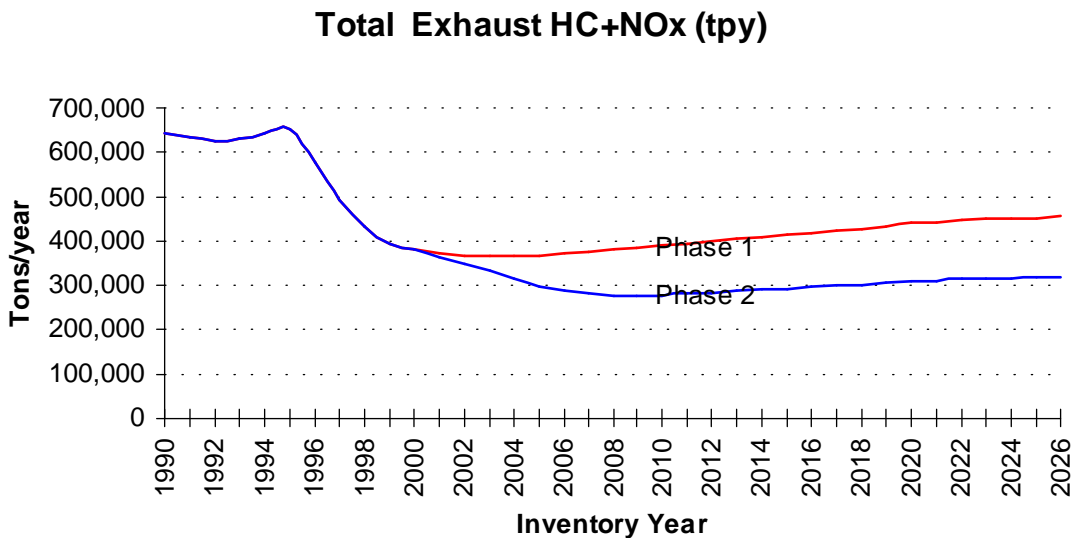
In Figure 4-01, the annual benefit of the proposed regulation from reduction in Total exhaust HC+NOx is indicated by the difference between the upper and lower

curves. The area between the curves represents the net benefit of the proposed regulation during the time required for the nonroad small SI engine and equipment fleet to completely turn over. The averaged results indicate that the proposed standard represents on average a 30% reduction in annual HC+NOx emissions from engines to which the standards apply.

6.1.1.8. Fuel Consumption Benefits-- in addition, the proposed rule is expected to reduce Fuel Consumption by an additional 9% from Phase1 by year 2025 over and above the 16% reduction the Phase1 rule is expected to achieve. Refuelling losses were recalculated accounting for the percentage decrease in fuel consumption between uncontrolled and Phase1 and between Phase1 and Phase2 scenarios. These adjusted numbers were then used in calculating the percentage reduction in refuelling losses between 1996 to 2026. EPA expects to obtain a 9% reduction in HC refuelling losses by year 2025. The tabulations are presented in Table F-09.

6.2 Air Quality Benefits

Air quality benefits associated with reduction in VOC emissions are discussed in this section. Health and welfare effects of the pollutants as they impact on ozone formation are described.

6.2.1. VOC

EPA expects that reducing VOC emissions from small nonroad spark ignition engines will help to mitigate the health and welfare impacts of ambient HC on urban and regional tropospheric ozone formation and transport.

6.2.1.1. Health and Welfare Effects of VOC Emissions--VOC is the general term used to denote volatile organic compounds, a broad class of pollutants encompassing hundreds of specific toxic compounds, primarily Benzene and 1,3 Butadiene as well as aldehydes and gasoline vapors. As stated previously, VOC is a criteria pollutant for which the EPA has established a NAAQS. Measures to control VOC emissions should reduce emissions of hazardous air pollutants (HAPs). However, the

magnitude of reduction will depend on whether the control technology reduces the individual HAPs in the same proportion that total VOCs are reduced. Since nonroad engines have significant VOC impacts , thay are expected to have significant impacts on HAPs as well.

At elevated concentrations, VOC, a precursor to ozone, can adversely affect human health, agricultural production and environmental welfare. EPA is examining new directions and long-term efforts toward VOC reductions as well as approaches that are largely untried.One such step is the establishment of the new national ambient air quality standards (NAAQS), promulgated on July 17, 1997 for ground-level ozone. EPA phased out and replaced the previous 1-hour primary ozone standard (health-based) with a new 8-hour standard in order to protect against longer exposure periods. The new 8-hour standard is set at 0.08 parts per million(ppm) and is defined as a "concentration-based" form. EPA also replaced the previous secondary standard (to protect the environment, including agricultural crops, national parks, and forests) with a standard identical to the new primary standard.

Nonroad sources contribute substantially to summertime VOC and NOx emissions and winter CO emissions. The median contribution of total nonroad emissions to VOC and NOx inventories in summer, and CO inventories in winter, ranges from 7.4-12.6% VOC, 14.5-17.3% NOx, and 5.2-9.4% winter CO, depending on the area [4]. The lawn and garden equipment category is a major contributor to summertime VOC emissions, accounting for a median ranging from 2.4% to 4.7% of the total VOC inventory in tons per summer day, depending on the area.

6.2.2. Benzene

Benzene is a clear, colorless, aromatic hydrocarbon which has a characteristic odor. It is both volatile and flammable. Benzene contains 92.3% carbon and 7.7% hydrogen with the resulting chemical formula C_6H_6. Benzene is present in both exhaust and evaporative emissions. Data show the benzene level of gasoline to be about 1.5%. Some exhaust benzene is unburned fuel benzene. Some benzene also forms from engine

combustion of non-aromatic fuel hydrocarbons. The fraction of benzene in the exhaust varies depending on control technology and fuel composition and is generally about 3 to 5%. The fraction of benzene in the evaporative emissions also depends on control technology and fuel composition and is generally about 1%.

Mobile sources account for approximately 65% of the total benzene emissions, of which 30% can be attributed to nonroad mobile sources (5). For nonroad engines, benzene was estimated to be about 3.0% of VOC emissions and 1.7% of evaporative VOC emissions. The split between exhaust and evaporative benzene emissions was assumed to be 80% exhaust to 20% evaporative. Thus, the overall benzene fraction of nonroad VOC emissions was estimated to be 2.7%.

6.2.2.1. Projected Benzene Emission Reductions--Nonroad

engines account for approximately 20% of the total benzene emissions with 45% attributed to highway motor vehicles and 35% to stationary sources. Many of the stationary sources attributed to benzene emissions are industries producing benzene as a by-product or use benzene to produce other chemicals.

Since benzene levels generally decrease proportionally to overall HC emissions, once newer emission control technology is applied, the amount of benzene produced by new small SI engines should be reduced further from Phase 1 after this new rule becomes effective.

6.2.2.2. Health Effects of Benzene Emissions--Health effects

caused by benzene emission differ based on concentration and duration of exposure. EPA's Total Exposure Assessment Methodology (TEAM) Study identified the major sources of exposure to benzene for much of the U.S. population. These sources turn out to be quite different from what had previously been considerd as important sources. The study results indicate that the main sources of human exposure are associated with personal activities, not with the so-called "major point sources". The results imply that personal activities or sources in the home far outweigh the contribution of outdoor air to human exposure to benzene. Since most of the traditional sources exert their effect

through outdoor air, some of the nonroad small SI engine sources could explain the increased personal exposures observed. The TEAM Study is described in detail in a four-volume EPA publication (6) and in several journal articles (7-8) .

The average ambient level of benzene ranges from 4.13 to 7.18 µg/m^3, based on urban air monitoring data. A crude estimate of ambient benzene contributed by < 19kW SI engine sources can be calculated by multiplying the total ambient concentration by the percentage of nonroad engine-produced benzene. This figure must be adjusted then to reflect time spent indoors and in other microenvironments by using the factor developed in the Motor-Vehicle-Related Air Toxics Study. Applying the nonroad adjustment factor of .25 and integrated adjustment factor of .622 to reflect only nonroad exposure to benzene, the range becomes .642 to 1.12 µg/m^3.

Based on data from EPA's NEVES(4), the exhaust and crankcase emissions from a 2.9 kW (3.9 hp) lawnmower with a 4-stroke engine contain 3.5 grams of benzene. A 2.9 kW (3.9 hp), 2-stroke lawnmower exhaust has 17 grams of benzene. A small, 2.2 kW (3 hp) chainsaw emits 28.2 grams of benzene per hour, compared to a large, 4.5 kW (6 hp) chainsaw that emits 40.8 grams per hour. No study as yet has been conducted on the health effects of benzene emissions specifically from small SI engines.

A separate study conducted at Southwest Research Institute (SWRI) reported a 2-stroke, 4.5 kW(6hp) moped engine fueled with industry average unleaded gasoline emitted 2,260 mg/hph of benzene. A 4-stroke walk-behind mower powered by an overhead valve, 2.6 kW (3.5 hp) engine emitted 690 mg/hph of benzene when fueled with average unleaded gasoline.

Concentration and duration of exposure to benzene are especially important to consider in the case of small SI engine applications, since the operator is typically in the direct path of the exhaust given out by the engine. Rate of dilution of the exhaust by the air surrounding the engine depends on local weather conditions.

6.2.2.3. Carcinogenicity of Benzene and Unit Risk Estimates--

The International Agency for Research on Cancer (IARC), classified benzene as a Group I carcinogen . A Group I carcinogen is defined as an agent that is carcinogenic to

humans. IARC (1987) based this conclusion on the fact that numerous case reports and follow-up studies have suggested a relationship between exposure to benzene and the occurrence of various types of leukemia. The leukemogenic (i.e., the ability to induce leukemia) effects of benzene exposure were studied in 748 white males employed from 1940-1949 in the manufacturing of rubber products in a retrospective cohort mortality study (9). Statistics were obtained through 1975. A statistically significant increase in the incidence of leukemia was found by comparison to the general U.S. population. The worker exposures to benzene were between 100 ppm and 10 ppm during the years 1941-1945. There was no evidence of solvent exposure other than benzene. In addition, numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene (IARC 1982).

Exposure to benzene has also been linked with genetic changes in humans and animals. EPA has concluded that benzene is a Group A, known human carcinogen based on sufficient human epidemiologic evidence demonstrating an increased incidence of nonlymphocytic leukemia from occupational inhalation exposure. The supporting animal evidence showed an increased incidence of neoplasia in rats and mice exposed by inhalation and gavage. EPA (10) calculated a cancer unit risk factor for benzene of 8.3×10^{-6} $(\mu g/m^3)^{-1}$ based on the results of the above human epidemiological studies in benzene-exposed workers in which an increase of death due to nonlymphocytic leukemia was observed. EPA's National Center for Environmental Assessment (NCEA) of the office of Research and Development (ORD) has recently announced a Notice of Peer-Review Workshop and Public Comment Period to review an external review draft document titled, *Carcinogenic Effects of Benzene: An update (EPA/600/P-97/001A).* EPA will consider comments and recommendations from the workshop and the public comment period in document revisions.

The California Department of Health Services (DHS, 1984), which provides technical support to CARB, has also determined that there is sufficient evidence to consider benzene a human carcinogen. CARB performed a risk assessment of benzene

that was very similar to EPA's risk assessment. The CARB risk estimate is actually a range, with the number calculated by EPA serving as the lower bound of cancer risk and a more conservative (ie., higher) number, based on animal data , serving as the upper bound of cancer risk. The CARB potency estimate for benzene ranges from $8.3x10^{-6}$ to $5.2x10^{-5}$ µg/m^3.

A number of adverse noncancer health effects have also been associated with exposure to benzene. People with long-term exposure to benzene at levels that generally exceed 50 ppm (162,500 µg/m^3) may experience harmful effects on the blood-forming tissues, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components, such as red blood cells and blood platelets, leading to anemia and a reduced ability to clot. Exposure to benzene at comparable or even lower levels can be harmful to the immune system, increasing the chance for infection and perhaps lowering the body's defense against tumors by altering the number and function of the body's white blood cells. In studies using pregnant animals, inhalation exposure to benzene in the range of 10-300 ppm (32,500-975,000 µg/m^3) indicates adverse effects on the developing fetus, including low birth weight, delayed bone formation, and bone marrow damage.

6.2.3. 1,3- Butadiene

1,3-Butadiene is a colorless, flammable gas at room temperature with a pungent, aromatic odor, and a chemical formula C_4H_3. 1,3-Butadiene is insoluble in water and because of its reactivity , is estimated to have a short atmospheric lifetime. The actual lifetime depends upon the conditions at the time of release, such as the time of day, intensity of sunlight, temperature etc. 1,3-Butadiene is formed in vehicle exhaust by the incomplete combustion of the fuel and is assumed not to be present in vehicle evaporative and refueling emissions. The percentage of 1,3-butadiene in unregulated small SI engines is estimated to be approximately 1.3 percent. The contribution of 1,3 -butadiene from Nonroad Sources to Nationwide Toxic Emissions Inventory is 21.2% (5).

6.2.3.1. Projected 1,3-Butadiene Emission Reductions--Current

EPA estimates (5) indicate that mobile sources account for approximately 68% of the total 1,3-butadiene emissions, out of which 31% can be attributed to nonroad mobile sources. The remaining 1,3-butadiene emissions come from stationary sources mainly related to industries producing 1,3-butadiene and those industries that use 1,3-butadiene to produce other compounds. 1,3-Butadiene emissions appear to increase roughly in proportion to exhaust hydrocarbon emissions. Since hydrocarbons are decreased by the use of a catalyst on a motor vehicle, 1,3-butadiene emissions are expected to decrease proportionally with the use of any emission control technology that decreases total hydrocarbon emission.

6.2.3.2. Health Effects of 1,3 - Butadiene Exposure--The annual

average ambient level of 1,3-butadiene ranges from 0.12 to 0.56 $\mu g/m^3$. According to data from EPA's NEVES, 1,3-Butadiene content in exhaust and crankcase from a 2.9 kW (3.9 hp), 4-stroke lawnmower is approximately 1.5 gms/hr of usage. For a 2.9 kW (3.9 hp), 2-stroke lawnmower, 1,3-butadiene content in exhaust is 7.0 grams per hour. Butadiene emitted from small, 2.2 kW (3hp) chainsaw is approximately 12.2 grams per hour from a large 4.5 kW (6 hp) chainsaw.

A separate study conducted at SwRI revealed a 2-stroke, 4.5 kW (6 hp) moped engine emitted 207 mg/kW-hr (154 mg/hp-hr) when fueled with industry average unleaded gasoline. A 2.6 kW (3.5 hp) overhead valve, walk-behind mower emitted 209 mg/kW-hr (156 mg/hp-hr) of 1,3-butadiene when fueled with industry average unleaded gasoline. Since 1,3-butadiene levels normally decrease proportional to overall hydrocarbons once emission control technology is applied, 1,3-butadiene levels are expected to be less from new small SI engines after this rule becomes effective . This, in turn, will reduce risk of exposure to 1,3-butadiene produced by these sources.

Since the operator of a small SI engine- equipped application is typically near the equipment while it is in use, the concentration of toxic pollutants in the exhaust and their health effects need to be investigated. Although the air around the engine quickly dilutes the exhaust, the rate of dilution depends on the weather conditions.

6.2.3.3. **Carcinogenicity of 1,3-Butadiene**--Long-term inhalation

exposure to 1,3-butadiene has been shown to cause tumors in several organs in experimental animals. Epidemiologic studies of occupationally exposed workers were inconclusive with respect to the carcinogenicity of 1,3-butadiene in humans. Based on the inadequate human evidence and sufficient animal evidence, EPA has concluded that 1,3-butadiene is a Group B2, probable human carcinogen. IARC has classified 1,3-butadiene as a Group 2A, probable human carcinogen. EPA calculated a cancer uinit risk factor of $2.8X10^{-4}$ $(\mu g/m^3)^{-1}$ for 1,3-butadiene based on the results of a study in mice in which an increase in the incidence of tumors in the lung and blood vessels of the heart, as well as lymphomas were observed. EPA's Office of Research and Development is currently in the process of releasing an updated 1,3-butadiene risk assessment factor.

Exposure to 1,3-butadiene is also associated with adverse noncancer health effects. Exposure to high levels (on the order of hundreds of thousands ppm) of this chemical for short periods of time can cause irritation of the eyes, nose, and throat, and exposure to very high levels can cause effects on the brain leading to respiratory paralysis and death Studies of rubber industry workers who are chronically exposed to 1,3-butadiene suggest other possible harmful effects including heart disease, blood disease, and lung disease. Studies in animals indicate that 1,3-butadiene at exposure levels of greater than 1,000 ppm ($2.2X106$ $\mu g/m^3$) may adversely affect the blood-forming organs. Reproductive and developmental toxicity has also been demonstrated in experimental animals exposed to 1,3-butadiene at levels greater than 1,000 ppm.

6.2.4 CO

The Clean Air Act directs the Administrator of the EPA to establish National Ambient Air Quality Standards (NAAQS) for several widespread air pollutants, based on scientific criteria and allowing for an adequate margin of safety to protect public health. The current primary and secondary NAAQS for CO are 35ppm for a 1-hour average and 9ppm for an 8-hour average.

According to the Nonroad Study, a 4-stroke, 2.9 kW (3.9 hp) lawnmower engine

emits 1051.1 g/hr CO while a 2-stroke, 2.9 kW (3.9 hp) engine meets 1188.4 g/hr CO. A separate study conducted at SwRI revealed that a 2-stroke moped engine fueled with typical unleaded gasoline emits 184 g/Kw-hr (137 g/hp-hr) of CO. A 4-stroke, 2.6 kW overhead valve, walk-behind mower fueled with typical unleaded gasoline emits 480 g/kW hr (358 g/hp-hr) of CO.

Although the proposed Phase 2 emission standards for small SI engines does not include CO, reductions in CO beyond Phase 1 levels, due to improved technology, is also to be expected by year 2025.

6.2.4.1. Health and Welfare Effects of CO--The EPA has documented the detrimental health effects that CO can have on populations(11). Carbon monoxide is a colorless, odorless, tasteless and nonirritating gas and gives no signs of its presence. It is readily absorbed from the lungs into the bloodstream, there forming a slowly reversible complex with hemoglobin (Hb) known as carboxyhemoglobin (COHb).

Blood COHb levels do not often exceed 0.5 to 0.7% in normal individuals unless exogenous CO is breathed. Some individuals with high endogenous CO production can have COHb levels of 1.0 to 1.5% (e.g. anemics). The presence of COHb in the blood reduces the amount of oxygen available to vital tissues, affecting primarily the cardiovascular and nervous systems. Although the formation of COHb is reversible, the elimination half-time is quite long because of the right binding between CO and Hb. This can lead to accumulation of COHb, and extended exposures to even relatively low concentrations of CO may produce substantially increased blood levels of COHb.

Health effects associated with exposure to CO include cardiovascular system, central nervous system (CNS), and developmental toxicity effects, as well as effects of combined exposure to CO and other pollutants, drugs, and environmental factors. Concerns about the potential health effects of exposure to CO have been addressed in extensive studies with various animal species as subjects. Under varied experimental protocals, considerable information has been obtained on the toxicity of CO, its direct effects on the blood and other tissues, and the manifestations of these effects in the form of changes in organ function. Many of these studies, however have been conducted at

extremely high levels of CO (i.e., levels not found in ambient air). Although severe effects from exposure to these high levels of CO are not directly germane to the problems from exposure to current ambient levels of CO, they can provide valuable information about potential effects of accidental exposure to CO, particularly those exposures occurring indoors.

All gasoline-powerd engines produce carbon monoxide. According to the National Institute for Occupational Safety and Health (NIOSH), Americans who use gasoline-powered pressure washer indoors are risking their lives. This gas can rapidly build up in any indoor area, and individuals can be overcome without even realizing that they are being exposed. Confusion, headache, dizziness, fatigue, and weakness may set in too quickly for victims to save themselves. According to NIOSH director, Dr. J. Donald Millar, " Carbon monoxide strikes quickly, and it strikes without warning. Workers must be aware of the hazard and prevent exposure to this potentially fatal gas." Each of the victims interviewed by NIOSH expressed shock at how quickly they were overcome. Carbon monoxide poisoning can cause permanent brain damage , including changes in personality and memory. Once inhaled, carbon monoxide decreases the ability of the blood to carry oxygen to the brain and other vital organs. Even low levels of carbon monoxide can set off chest pains and heart attacks in people with coronary artery disease.

Although no studies measuring the human health effects of CO emanating from small SI engine exhaust have been conducted, ample research results are available concerning general health effects of exposure to CO . The effects of exposure to low concentrations-such as the levels found in ambient air - are far more subtle and considerably less threatening than those occurring in direct poisoning from high CO levels. Maximal exercise performance in healthy individuals has been shown to be affected at COHb levels of 2.3% and greater. Central nervous sytem effects, observed at peak COHb levels of 5% and greater, include reduction in visual perception, manual dexterity, learning, driving performance, and attention level. Of most concern, however, are adverse effects observed in individuals with chronic heart disease at COHb levels of 3 to 6%. At these levels, such individuals are likely to have reduced capacity for physical

activity because they experience chest pain (angina) sooner. Exercise-related cardiac arrhythmias have also been observed in some people with chronic heart disease at COHb levels of 6% or higher and may result in an increased risk of sudden death from a heart attack .

The NAAQS set by EPA are intended to keep COHb levels below 2.1% in order to protect the most sensitive members of the general population (i.e., individuals with chronic heart disease). However, elderly people, pregnant women (due to possible fetal effects), small children, and people with anemia or with diagnosed or undiagnosed pulmonary or cardiovascular disease are also likely to be at increased risk for CO effects.

Since small SI engines are typically used in applications that require the operator to be near, and perhaps in the direct path of the exhaust, the effects of exhaust CO on the operator of the engine is a matter of concern. Although no studies measuring the human health effects of CO emanating from small SI engine exhaust have been conducted, laboratory animal studies reveal that CO can adversely affect the cardiovascullar system, depending on the --- conditions utilized in these studies.

6.2.4.2. Developmental Toxicity and Other Systemic Effects of Carbon monoxide--Studies in laboratory animals of several species provide strong evidence that maternal CO exposures of 150 to 220 ppm, leading to approximately 15 to 25% COHb, produce reductions in birth weight, cardiomegaly, delays in behavioral development, and disruption in cognitive function (12). The current data (13) from human children suggesting a link between environmental CO exposures and sudden infant death syndrome are weak. Human data from cases of accidental high CO exposures (14) are difficult to use in identifying a low observed-effect level for CO because of the small numbers of cases reviewed and problems in documenting levels of exposure.

Behaviors that require sustained attention or sustained performance are most sensitive to disruption by COHb. The group of human studies (15) on hand-eye coordination (compensatory tracking), detection of infrequent events (vigilance), and continuous performance offer the most consistent and defensible evidence of COHb

effects on behavior at levels as low as 5%. These effects at low CO-exposure concentrations, however, have been very small and somewhat controversial. Nevertheless, the potential consequences of a lapse of coordination, vigilance, and the continuous performance of critical tasks by operators of machinery could be serious.

At higher levels of exposure, where COHb concentrations exceed 15 to 20%, there may be direct inhibitory effects of CO resulting in decreases in xenobiotic metabolism , which might be important to individuals receiving treatment with drugs. Inhalation of high levels of CO, leading to COHb concentrations greater than 10 to 15%, have been reported to cause a number of other systemic effects in laboratory animals as well as humans suffering from acute CO poisoning. There are reports in the literature of effects on liver, kidney, bone, and immune capacity in the lung and spleen (16). It generally is agreed that these effects are caused by severe tissue damage occurring during acute CO poisoning.

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