INVESTIGATION OF THE LEVELS OF PARTICULATE MATTER CONCENTRATIONS IN MINNA, NIGER STATE

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Abstract

Over the last couple of decades interest has been centred on environmental pollution and climate change issues all over the world. This study was designed and executed such that an appropriate town-gown synergy advocacy could evolve. Thus, the objectives of this study are to help prepare the framework for a particulate matter pollution database and to help build the nucleus for an environmental awareness advocacy programme to be funded and executed by the Niger State Government. Co-ordinate identification for this study was facilitated by the use of hand-held Global Positioning System (GPS) units. The core field equipment employed for this study is the particulate level meter and the main component of the device is the particulate sensor that detects the presence of particulate matter when they fall on it. Stations of interest that were identified were appropriately geo-referenced and marked in the conventional way. The stations were re-visited with the particulate level equipment whence information about the sources of particulate matter and their corresponding values were logged progressively from one point to the next. For each type of source of particulate matter pollution two values were taken independent of one another. All the stations occupied for this survey (about 11,408 domestic and commercial locations) indicate particulate matter emission levels greater than the internationally-recommended safe threshold of 0.100mg/m³. The Geographic Information System (GIS) tool was employed in the analysis of the full body of the dataset acquired for this study with the core objective of producing a particulate matter pollution layer for Minna. Thus at the click of a mouse, information about the particulate matter pollution signature for Minna could readily be assessed. From the particulate matter pollution map, it is observed that the Barkin-Sale, Minna Central, Soje and Tunga neighbourhoods have high red dot densities, whilst the dot densities for Sabon Gari and Sauka Kahuta neighbourhoods are sparsely distributed.

Keywords: Particulate-matter, pollution, GIS, advocacy

Introduction

From well before recorded history, man has been working towards improving his standard of living, and in the process evolving means by which he could effectively exploit the natural resources around him. In his quest for a better living, he invented stoves, power generators, cars, motor-cycles, trains, ships, aeroplanes, and so on. All of these contraptions are powered by fossil fuels, with attendant environmental pollution. As population grows and society becomes complex, industrial and motorised activities also grow in direct proportion. Consequently the quantity of waste products released into the atmosphere increases in such huge amounts that at some point they begin to manifest negatively in the ecosystem. Over the last couple of decades interest has been centred on environmental pollution and climate change issues all over the world. Thus it is fitting and proper that, in fulfilment of one of its founding charters, the Federal University of Technology, Minna, should be able to make its contribution in the field of environmental pollution, especially as it affects the local communities. To this end,

this study of the levels of particulate matter concentrations in Minna was designed and executed such that the results would assist in policy-making concerning pollution.

Particulate matter (PM) is a complex mixture of solid and liquid particles that are suspended in air. These particles typically consist of a mixture of inorganic and organic chemicals, including carbon, sulphates, nitrates, metals, acids, and semi-volatile compounds. The size of particulate matter in air ranges from approximately 0.005 to 100 micrometers (μ m) in diameter (that is, the size of just a few atoms to about the thickness of a human hair). Researchers have defined size categories for these particles differently. Particulate matter is defined by three general categories commonly used by the U.S. Environmental Protection Agency (U.S. EPA): coarse (10 to 2.5 μ m), fine (2.5 μ m or smaller), and ultrafine (0.1 μ m or smaller). Research suggests that particle size is an important factor that influences how particles deposit in the respiratory tract and affect human health. Coarse particles are deposited almost exclusively in the nose and throat, whereas fine and ultrafine particles generally are able to penetrate to deep areas of the lung; fine and ultrafine particles are present in greater numbers and have greater surface area-to-volume ratio than larger particles of the same mass, and they are generally considered to be more toxic (Richard *et al.*, 2001; Jokin *et al.*, 2007).

The effects of inhaling particulate matter have been widely studied in humans and animals and include asthma, lung cancer, cardiovascular issues, and premature death. The size of the particle is a main determinant of where in the respiratory tract the particle will come to rest when inhaled. Depending of the size of the particle, they can penetrate the deepest part of the lungs. Larger particles are generally filtered in the nose and throat and do not cause problems, but particulate matter smaller than about 10 micrometers, referred to as PM₁₀, can settle in the bronchi and lungs and cause health problems. The 10 micrometer size does not represent a strict boundary between respirable and non-respirable particles, but has been agreed upon for monitoring of airborne particulate matter by most regulatory agencies. Similarly, particles smaller than 2.5 micrometers, PM_{2.5}, tend to penetrate into the gas exchange regions of the lung, and very small particles (< 100 nanometers) may pass through the lungs to affect other organs. In particular, PM_{2.5} leads to high plaque deposits in arteries, causing vascular inflammation and atherosclerosis, a hardening of the arteries that reduces elasticity, which can lead to heart attacks and other cardiovascular problems. Researchers suggest that even short-term exposure at elevated concentrations could significantly contribute to heart disease.

The principal study objectives of this work are as follows: (i) to help prepare the framework for a particulate matter pollution database for Minna; this study will be the substratum upon which subsequent studies would be laid, (ii) to help build the nucleus for an environmental awareness advocacy programme to be funded and executed by the Niger State Government; such an awareness programme could take the form of the production of specific posters detailing the prevalence or otherwise of environmental pollution in any particular locality of Minna, and (iii) to fulfil one of the founding charters of the Federal University of Technology, Minna, that is, the deployment of academic resources to solve pressing community needs (the town-gown synergy concept).

Mutlu *et al.* (2007) published an article in which they posited that the mechanisms by which exposure to particulate matter increases the risk of cardiovascular events were not known. They pointed out further that human and animal data available to them suggested that particulate matter may induce alterations in hemostatic factors. In their study they determined the

mechanisms by which particulate matter might accelerate thrombosis; they found that mice treated with a dose of well characterized particulate matter of less than 10 µm in diameter exhibited a shortened bleeding time, decreased prothrombin and partial thromboplastin times (decreased plasma clotting times), increased levels of fibrinogen, and increased activity of factor II, VIII, and X. This prothrombotic tendency was associated with increased generation of intravascular thrombin, an acceleration of arterial thrombosis, and an increase in bronchoalveolar fluid concentration of the prothrombotic cytokine IL-6. Knockout mice lacking IL-6 were protected against particulate matter—induced intravascular thrombin formation and the acceleration of arterial thrombosis. Depletion of macrophages by the intratracheal administration of liposomal clodronate attenuated particulate matter—induced IL-6 production and the resultant prothrombotic tendency. Their findings suggest that exposure to particulate matter triggers IL-6 production by alveolar macrophages, resulting in reduced clotting times, intravascular thrombin formation, and accelerated arterial thrombosis. These results provided a potential mechanism linking ambient particulate matter exposure and thrombotic events.

Roux et al. (2008) reported that exposure to airborne particulate matter had been linked to cardiovascular events. Using data from the Multi-Ethnic Study of Atherosclerosis collected at baseline (2000–2002), the authors investigated associations of 20-year exposures to particulate matter with measures of subclinical disease (coronary calcium, common carotid intimal-medial thickness, and ankle-brachial index) in 5,172 US adults without clinical cardiovascular disease. Particulate matter exposures for the 20 years prior to assessment of subclinical disease were obtained from a space-time model of Environmental Protection Agency monitor data linked to residential history data for each participant. Intimal-medial thickness was weakly, positively associated with exposures to particulate matter <10 µm in aerodynamic diameter and <2.5 µm in aerodynamic diameter after controlling for age, sex, race/ethnicity, socioeconomic factors, diet, smoking, physical activity, blood lipids, diabetes, hypertension, and body mass index (1-4% increase per 21-ug/m³ increase in particulate matter <10 µm in aerodynamic diameter or a 12.5-µg/m³ increase in particulate matter <2.5 µm in aerodynamic diameter). No consistent associations with other measures of atherosclerosis were observed. There was no evidence of effect modification by sociodemographic factors, lipid status, smoking, diabetes, body mass index, or site. Results are compatible with some effect of particulate matter exposures on development of carotid atherosclerosis.

McCreanor *et al.* (2007) studied a panel of people in London with either mild or moderate asthma who took 2-hour walks on Oxford Street and, on separate occasions, through the western side of Hyde Park. The only vehicular traffic permitted on Oxford Street was diesel-powered buses and taxis, which presented a unique opportunity to contrast the exposures of the subjects to fresh effluents from diesel engines during their distance-matched walks, which were separated by at least 3 weeks and occurred between November and March to avoid exposure to pollen. For each walk, peak flow measurements were made before the walk, at 1 hour into the walk, and after the walk, and data on symptoms were collected at the end. During each session, the authors measured nitrogen dioxide and the ultrafine-particle count and collected filter samples that were analyzed for elemental carbon and $PM_{2.5}$ (particulate matter that was <2.5 µm in aerodynamic diameter) mass. Baseline respiratory function, the fraction of nitric oxide in exhaled breath (FE_{NO}), and the pH of exhaled breath condensate were measured in a hospital setting 10 minutes before and at various times after each walk; these measurements were thought to reflect the state of the inflammatory response within the airway, with a higher FE_{NO} value and a lower pH indicating more inflammatory activity. Methacholine

responsiveness and symptoms were recorded after the walk, and sputum samples were collected the next morning for measurements of cell counts, interleukin-8, myeloperoxidase, and eosinophil cationic protein. As expected, subjects had higher median exposures on Oxford Street than in Hyde Park (PM_{2.5}, 28 vs. 12 µg per cubic meter; nitrogen dioxide, 142 vs. 22 µg per cubic meter; elemental carbon, 7.5 vs. 1.3 µg per cubic meter; and ultrafine-particle count, 64,000 vs. 18,000 per cubic centimeter). The subjects with mild asthma had a higher baseline FEV₁ than those with moderate asthma (93% vs. 84% of their predicted normal values). Significant declines in FEV₁ and FVC occurred within 1 hour after the walk at both locations but were significantly greater after the walks on Oxford Street and were greater for subjects with moderate as compared with mild asthma. Although the decline in FEF₂₅₋₇₅ was not significantly associated with the site, it was greater and more significantly associated with the concentrations of PM_{2.5}, nitrogen dioxide, elemental carbon, and ultrafine particles than was either FEV₁ or FVC. An increase in FE_{NO} was significantly associated with increases in elemental carbon from 3 to 22 hours after exposure but not with the other pollutants at any time. A decrease in the pH of exhaled condensate was associated with nitrogen dioxide, but only at 3 hours after exposure. Sputum myeloperoxidase was significantly related to ultrafine particles, but there were no associations between pollutant and cell counts or interleukin-8. In multipollutant models, elemental carbon and ultrafine-particle counts tended to be stronger predictors of responses than did PM_{2.5} or nitrogen dioxide. Elemental carbon and ultrafine-particle counts are hypothesized to be useful markers of diesel exhaust, and the results reported by the authors provided support for this hypothesis. Furthermore, the extent and statistical significance of their reported effects were greater than the effects reported by other investigators that were based on laboratory exposures to diluted diesel exhaust in chambers and of those that were based on studies of populations exposed to more complex mixtures that included elemental carbon and ultrafine particles from gasoline-engine exhaust and particles resuspended from roadways. Also, many of these other studies involved subjects with mild asthma. In any case, this highly structured and well-executed study, with realistic, street-level exposures, clearly demonstrates that diesel exhaust, as indexed by elemental carbon and ultrafine particles, at levels seen in a modern, first-world city, does produce acute small-airway effects in people with mild or moderate asthma and that greater, clinically significant effects are likely in people with more severe asthma. Both groups of researchers were able to produce statistically significant grouplevel effects at particulate-matter concentrations below current WHO guidelines and EPA standards because of their ability to limit exposure error by assigning personal exposure levels to each subject. The magnitude of the effects was relatively small and of limited clinical relevance to individual patients, but it does have public health relevance. Standards and guidelines were needed to protect sensitive subpopulations such as those with preexisting lung disease. For nominally healthy people, there was a wide degree of susceptibility to reduced longevity from exposure to particulate matter (for reasons not yet established), and a small increase in the annual mortality rate related to particulate matter would correspond to thousands of excess deaths. These studies provide additional biologic data indicating that relatively low levels of airborne particles have adverse effects on human health.

Ladental *et al.* (2000) reported that fine particle mass (particulate matter (less than and equal to) 2.5 microm; PM(2.5), which is primarily from combustion sources, but not coarse particle mass, which is primarily from crustal sources, was associated with daily mortality in six eastern U.S. cities. In their study, they used the elemental composition of size-fractionated particles to identify several distinct source-related fractions of fine particles and examined the association of these fractions with daily mortality in each of the six cities. Using specific rotation factor

analysis for each city, they identified a silicon factor classified as soil and crustal material, a lead factor classified as motor vehicle exhaust, a selenium factor representing coal combustion, and up to two additional factors. They extracted daily counts of deaths from National Center for Health Statistics records and estimated city-specific associations of mortality with each source factor by Poisson regression, adjusting for time trends, weather, and the other source factors. Combined effect estimates were calculated as the inverse variance weighted mean of the city-specific estimates. In the combined analysis, a 10 microg/m increase in PM(2.5) from mobile sources accounted for a 3.4% increase in daily mortality [95% confidence interval (CI), 1.7-5.2%], and the equivalent increase in fine particles from coal combustion sources accounted for a 1.1% increase (CI, 0.3-2.0%). PM(2.5) crustal particles were not associated with daily mortality. These results indicate that combustion particles in the fine fraction from mobile and coal combustion sources, but not fine crustal particles, are associated with increased mortality.

According to Riley (2001) adverse human health effects have been observed to correlate with levels of outdoor particulate matter (PM), even though most human exposure to PM of outdoor origin occurs indoors. In their study, they applied a model and empirical data to explore the indoor PM levels of outdoor origin for two major building types: offices and residences. Typical ventilation rates for each building type are obtained from the literature. Published data are combined with theoretical analyses to develop representative particle penetration coefficients, deposition loss rates, and ventilation-system filter efficiencies for a broad particle size range (i.e., 0.001–10 μm). They applied archetypal outdoor number, surface area, and mass PM size distributions for both urban and rural airsheds. They also used data on mass-weighted size distributions for specific chemical constituents of PM: sulfate and elemental carbon. Predictions of the size-resolved indoor proportion of outdoor particles (IPOP) for various conditions and ambient particle distributions are then computed. The IPOP depends strongly on the ambient particle size distribution, building type and operational parameters, and PM metric. They concluded that an accurate determination of exposure to particles of ambient origin requires explicit consideration of how removal processes in buildings vary with particle size. Elevated levels of ambient particulate matter (PM₁₀) are associated with increased cardiopulmonary morbidity and mortality. They previously showed that the deposition of particles in the lung induces a systemic inflammatory response that includes stimulation of the bone marrow. This marrow response is related to mediators released by alveolar macrophages (AM) and in their study they measured cytokines produced by human AM exposed to ambient particles of different composition and size. Identified cytokines were also measured in the circulation of healthy young subjects exposed to air pollutants during the 1997 Southeast Asian forest fires. Human AM were incubated with particle suspensions of residual oil fly ash (ROFA), ambient urban particles (EHC 93), inert carbon particles, and latex particles of different sizes (0.1, 1, and 10 µm) and concentrations for 24 h. Tumor necrosis factor-alpha (TNF-x) increases in a dosedependent manner when AM were exposed to EHC 93 particles (p < 0.02). The TNF response of AM exposed to different sizes of latex particles was similar. The latex (158 \pm 31%), inert carbon $(179 \pm 32\%)$, and ROFA $(216 \pm 34\%)$ particles all show a similar maximum TNF response (percent change from baseline) whereas EHC 93 (1,020 \pm 212%, p < 0.05) showed a greater maximum response that was similar to lipopolysaccharide (LPS) $1 \mu g/ml$ (812 ± 320%). Macrophages incubated with an optimal dose of EHC 93 particles (0.1 mg/ml) also produce a broad spectrum of other proinflammatory cytokines, particularly interleukin (IL)-6 (p < 0.01), IL-13 (p < 0.05), macrophage inflammatory protein- 1α (MIP- 1α) (p < 0.05), and granulocyte macrophage colony-stimulating factor (GM-CSF) (p < 0.01) with no difference in concentrations of the anti-inflammatory cytokine IL-10 (p = NS). Circulating levels of IL-1 β , IL-6, and GM-CSF

were elevated in subjects exposed to high levels of PM_{10} during an episode of acute air pollution. These results show that a range of different particles stimulate AM to produce proinflammatory cytokines and these cytokines are also present in the blood of subjects during an episode of acute atmospheric air pollution. They postulated that these cytokines induced a systemic response that has an important role in the pathogenesis of the cardiopulmonary adverse health effects associated with atmospheric pollution.

Tamagaya et al. (2007) commented that epidemiologic and animal studies have shown that whether PM-induced lung and systemic inflammation is involved in this process is not clear. They hypothesized that PM exposure causes lung and systemic inflammation, which in turn leads to vascular endothelial dysfunction, a key step in the initiation and progression of atherosclerosis. New Zealand White rabbits were exposed for 5 days (acute, total dose 8 mg) and 4 wk (chronic, total dose 16 mg) to leither PM smaller than 10 µm (PM₁₀) or saline intratracheally. Lung inflammation was quantified by morphometry; systemic inflammation was assessed by white blood cell and platelet counts and serum interleukin (IL)-6, nitric oxide, and endothelin levels. Endothelial dysfunction was assessed by vascular response to acetylcholine (ACh) and sodium nitroprusside (SNP). PM_{10} exposure increased lung macrophages (P < 0.02), macrophages containing particles (P < 0.001), and activated macrophages (P < 0.006). PM₁₀ increased serum IL-6 levels in the first 2 wk of exposure (P < 0.05) but not in weeks 3 or 4. PM₁₀ exposure reduced ACh-related relaxation of the carotid artery with both acute and chronic exposure, with no effect on SNP-induced vasodilatation. Serum IL-6 levels correlated with macrophages containing particles (P = 0.043) and ACh-induced vasodilatation (P = 0.014 at week 1, P = 0.021 at week 2). Exposure to PM₁₀ caused lung and systemic inflammation that were both associated with vascular endothelial dysfunction. This suggests that PM-induced lung and systemic inflammatory responses contribute to the adverse vascular events associated with exposure to air pollution.

According to Binkowski (1995) the Regional Acid Deposition Model has been modified to create the Regional Particulate Model, a three-dimensional Eulerian model that simulates the chemistry, transport, and dynamics of sulfuric acid aerosol resulting from primary emissions and the gas phase oxidation of sulfur dioxide. The new model uses a bimodal lognormal distribution to represent particles in the submicrometer size range. In addition to including the horizontal and vertical advection and vertical diffusion of the aerosol number concentration and sulfate mass concentration fields, the model now explicitly treats the response of the distribution parameters to particle coagulation within and between the modes, condensation of sulfate vapor onto existing particles, formation of new particles, evaporation and condensation of ambient water vapor in the presence of ammonia, and particle-size-dependent dry deposition. The model has been used to study how the degree of sulfuric acid neutralization by ambient ammonia affects the total aerosol concentrations and particle size distributions over eastern North America. Preliminary results for three representative locations, rural, near-source, and nominal downwind of source, show that the effect is greatest for the rural and smallest for the near-source regions, which corresponds with the largest and smallest values, respectively, of ammonium-to-sulfate molar ratios. The results indicate that the model could provide a tool for investigating the effects of various pollution control strategies, as well as new or alternative formulations of important aerosol processes.

Pope et al. (2009) concluded that background exposure to fine-particulate air pollution has been associated with increased morbidity and mortality, suggesting that sustained reductions in

pollution exposure should result in improved life expectancy. Their study directly evaluated the changes in life expectancy associated with differential changes in fine particulate air pollution that occurred in the United States during the 1980s and 1990s. They compiled data on life expectancy, socioeconomic status, an demographic characteristics for 211 county units in the 51 U.S. metropolitan areas with matching data on fine-particulate air pollution for the late 1970s and early 1980s and the late 1990s and early 2000s. Regression models were used to estimate the association between reductions in pollution and changes in life expectancy, with adjustment for changes in socioeconomic and demographic variables and in proxy indicators for the prevalence of cigarette smoking.

Methodology

Co-ordinate Identification. Co-ordinate identification for this study was facilitated by the use of hand-held Global Positioning System (GPS) units. A GPS unit measures the geographical location (and elevation) of a place in terms of its longitude and latitude in units of degrees, minutes and seconds as well as the geographical location on the Universal Traverse Mercator (UTM) protocol. The operation of this device is done in open spaces, away from trees, tall buildings, and high tension cables which could be sources of interference of the signals transmitted to satellites in space. As soon as the device is switched on, signals are sent from the device to a special network of geostationary satellites. When at least three or four of these satellites are located, the location or elevation of any point on the surface of the earth could be fixed within an acceptable margin of error. A typical GPS device is shown in Fig.1.



Fig.1: Typical GPS device

Field Equipment. The core field equipment employed for this study is the particulate level meter. The main component of the device is the particulate sensor that detects the presence of particulate

matter when they fall on it. Such a device is shown in Fig.2.



Fig.2: Particulate level meter

Data Collection Procedure: Stations of interest that were identified were appropriately georeferenced and marked in the conventional way. The stations were re-visited with the particulate level equipment whence information about the sources of particulate matter and their corresponding values were logged progressively from one point to the next. For each type of source of particulate matter pollution, two values were taken independent of one another.

Concept of GIS

A Geographic Information System (GIS) is a computer-based tool for mapping and analysing geographic phenomenon that exist, and events that occur, on Earth. GIS technology integrates common database operations such as query and statistical analysis with the unique visualisation and geographic analysis offered by the maps. These abilities distinguish GIS from other information systems and make it valuable to a wide range of public and private enterprises for explaining events, predicting outcomes, and planning strategies. Map making and geographic analysis are not new, but a GIS performs these tasks faster and with more sophistication than

do traditional manual methods. We commonly think of a GIS as a single, well-defined, integrated computer system. However, this is not always the case. A GIS can be made up of a variety of software and hardware tools. The important factor is the level of integration of these tools to provide a smoothly operating, fully functional geographic data processing environment. In general, a GIS provides facilities for data capture, data management, data manipulation and analysis, and the presentation of results in both graphic and report form, with a particular emphasis upon preserving and utilising inherent characteristics of spatial data. The ability to incorporate spatial data, manage it, analyse it, and answer spatial questions is the distinctive characteristic of geographic information systems.

The Area of Study: The area covered for this study is Minna, the capital of the state of Niger, Nigeria. Minna town is where the Federal University of Technology is located and, for a novel study of this kind, this town provides the natural first choice location of study. This study was designed to cover all of the developed areal extent of Minna town, more of a house-to-house coverage scheme. The map of the areal extent of this study location, in a geo-referenced grid, is shown as Fig.3.

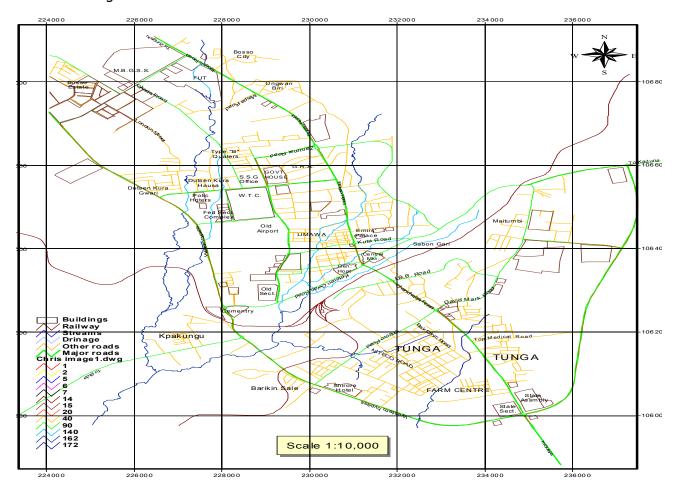


Fig.3: Map of area of study

Result Presentation and Creation of a Unique GIS Layer of Pollution Index Digitisation of Analogue Map of Study Area. The map of the study area shown in Fig.3 was digitised in the ArcView3.3 environment and the result is shown in Fig.4.

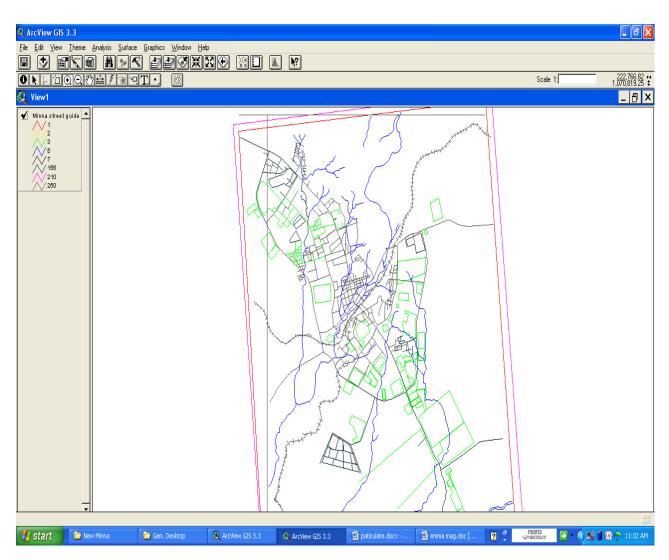


Fig.4: Digitised map of study area

Naming of Location on Digitized Maps: The attributes were named using the text tools on the ArcView menu. From the theme and edit icons, the text mode was enabled in order that locations on the map could be named. Shown in Fig.5 is the ArcView 3.3 map corresponding to the naming of locations.

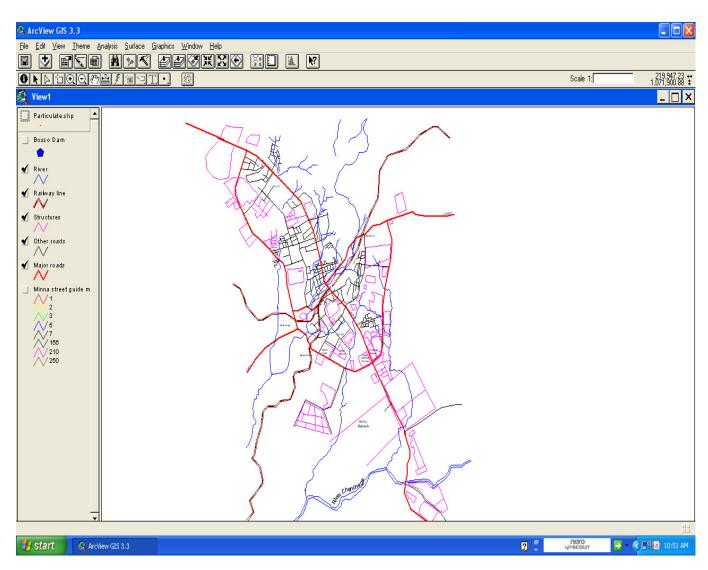


Fig. 5: Map corresponding to the naming of locations

Creation of Pollution Layer on Arcview3.3

The database was inputted and hot-linked to the spatial data (map and coordinate). The stations with high levels of particulate matter are identified on the map as red dots. High levels in this case means, having values greater than the safe threshold of 0.1mg/m³. The particulate matter pollution status map on ArcView3.3 is shown in Fig.6.

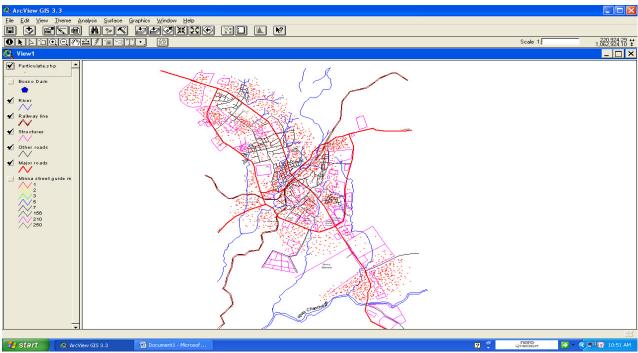


Fig.6: Particulate matter pollution status map

Particulate Matter Pollution Map on Interactive GIS

The particulate matter pollution map for Minna on interactive GIS is shown as Fig.7.

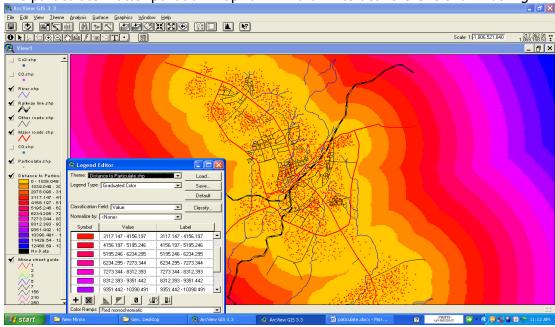


Fig.7. Particulate matter pollution for Minna

Discussion and Conclusion

All the stations occupied for this survey (about 11,408 domestic and commercial locations) indicate particulate matter emission levels greater than the internationally-recommended safe threshold of 0.100mg/m³. The predominant sources of particulate matter are common household generators, firewood and charcoal hearths. Others are grain milling machines, metal milling machines, and furniture workshops. The high dot densities observed at the Barkin-Sale, Minna Central, Soje, and Tunga neighbourhoods are pointers to the crowded human-habitation of these guarters where grain-milling activities easily combine with other sources to produce real environmental nuisance; wood-milling activities are commonplace at Tunga, metal-milling activities are commonplace at Minna Central, whilst at the Barkin-Sale and Soje area grainmilling plus firewood and charcoal hearths are heavy PM emitters. The dot densities that are sparsely distributed for the Sabon Gari and Sauka Kahuta neighbourhoods are pointers to the comparatively lower population densities of these quarters; metal-working activities are quite commonplace at the Sabon Gari area in addition to plumes from household generators whilst at the Sauka Kahuta area the culprits are exactly like those for the Barkin-Sale neighbourhood. The health of the population of these neighbouhoods are constantly put at risk by these almost continuous effluents, especially given the fact that efficient town-planning considerations were not taken into accounts when these veritable slum-dwellings sprung up at their present locations.

The GIS tool has been employed in the analysis of the full body of the dataset acquired for this study with the core objective of producing a particulate matter pollution layer for Minna. This unique GIS layer is the first of its kind anywhere in Nigeria. Thus at the click of a mouse, information about the particulate matter pollution signature for Minna can readily be assessed.

It can be deduced from the pollution map produced for this study that, overall, the particulate matter level regime over Barkin-Sale, Minna Central, Soie and Tunga neighbourhoods are guite high because a significant number of domestic and commercial locations use alternative power sources when the central power supply is cut. The more developed a neighbourhood is, the greater the particulate matter dot density. This GIS pollution layer map could now serve as a veritable urban development tool that would assist public health officials and town planners recommend appropriate action to be taken to safeguard the health of the citizenry. Based on the observation that particulate matter emissions can make our environment very unhealthy thus causing health and environmental issues, the following recommendations are suggested, viz: (i) increased use of alternative sources of energy by adopting the use of solar panels in place of wood and charcoal (fossil fuels), (ii) imposition of environment levies on users of fossil fuels or fossil fuel powered machines by the Niger State government, (iii) funding of research programmes by the government to bring out ways by which particulate matter emissions can be managed, (iv) an advocacy programme should be encouraged by the government to enlighten the individual on the health and environmental hazards of excessive use of particulate matter emission sources, (v) to prevent prolonged exposure to coarse particles, an appropriate supervision and quick completion of road construction projects by the ministry of works and housing should be emphasised, (vi) the Niger State Ministry of Environment should discourage open-discharge incineration around populated built-up areas, and (vii) finally, this study should be replicated in all the major towns and cities in Nigeria.

References

- Binkowski, F. S., & Shankar, U. (2001). The regional particulate matter model 1: Model description and preliminary results. 26, 191-26, 209.
- Jokin, J., Brunekreef, B., Janssen, N. A., Hartog, J., & Vanviet, R. (2007). Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology*, 3, 298-303.
- Ladental, J., Jemeft, M. I., Burnett, W. E., Krewski, R. A., Thurston, G., Finkelstien, N., & Thun, M. J. (2000). Spartial analysis of air pollution and mortallity in Los Angeles, *Epidemiol*, 16, 723-736.
- Mutlu, G. W., David, G., A., Bakar, C. M., Zach, B., Rajamannan, N, Christman, J. W., Foiles, N., Kamp, D. W., Gluo, A. J., Chandels, N. D. S., Scott-Budinge, G. R., Clin, J. I. (2007). Ambient particulates alter vascular function through induction of reactive oxygen and nitrogen species. 117 (10), 2952-2961.
- Pope C. A., Ezzati M, Douglas W., & Dockery S. C. D. (2009). Fine particulate air pollution and mortality extended follow-up of the Harvard Six Cities study. *Am. J. Resp. Care Med.*
- Richard, H. A., Anderson, R., Sunyer. J., Ayres, J., Baccini, M., Judith, M., Boumghar, A., Francesco, F. & Forsberz, B. (2001). Acute effects of particulate air pollution on respiratory admissions, 164, 1860-1866.
- Riley, W. J., Kone, M. C., Alvin, R., & William, W. N. (2001). Indoor particulate matter of outdoor origin: Importance of size-dependent removal mechanisms. 36 (2), 200-207.
- Roux, A. V., Amy, H. A., Franklin, T. G., Raghunathen, T., Graham, R. B., Kaufman, J., Astor B., & Keeler, J. (2008). Long-term exposure to ambient particulate matter and prevalence of sub-clinical atherosclerosis in the multi-ethnic study of atherosclerosis. 167(6), 697-675.
- Tamagawa, E., Morimoto K., Gray, C., Mill, T., Yatera, K., Zhang, X., Lixing, J., Yuexinli, S., & Laher, I. (2007). Particulate matter exposure induces persistent lung inflamation and endothelial dysfunction. 295 (1), 79.