(morbidity code 9440/3, mean annual incidence 453.4 per 1000 CNS malignancies) ($r_s = -0.87$). The trend for glioblastoma multiforme increased with time ($r_s = 0.93$), whereas the trend for anaplastic and low-grade astrocytomas declined with time ($r_s = -0.87$). Mixed and unspecified malignant gliomas (morbidity code 9380/3) had a mean annual incidence of 57.8 per 1000 CNS malignancies, without substantial correlations with time or with either of the other groups of gliomas.

The interchangeable dynamics in the site- and tumor-specific incidence rates of malignant gliomas over time could be derived, for example, from higher diagnostic sensitivity, accompanied by lower specificity and higher false-positive rate, of the imaging and tumor-classification tools used over time to diagnose high-grade gliomas in the temporal and frontal lobes. Such phenomena might introduce into epidemiologic studies a nondifferential misclassification bias of site- and tumor-specific outcomes that could distort the measured association and thus should be accounted for.  


**FIGURE.** Annual site-specific incidences per 1000 central nervous system malignancies during 2000–2008 in 17 registries of the United States Surveillance, Epidemiology and End Results (SEER).

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**REFERENCES**

**Effects of Local and Saharan Particles on Cardiovascular Disease Mortality**

To the Editor:

Recent studies suggest that outbreaks of Saharan dust over southern European populations may be harmful, after comparing exposure to particulate matter (PM) fractions during Saharan and non-Saharan dust days. Studies conducted in Barcelona1,2 and Rome3 found that the effects of short-term exposure to coarse fraction during dust days were associated with increased total mortality and, more specifically, cardiovascular mortality. However, the specific contribution of Saharan dust versus other local dust remains to be clarified. If Saharan dust is indeed a risk agent, there is a need to develop a source-specific risk function to guide European legislation on setting standards and acceptable risks.

In this study, we estimated the association between daily cardiovascular mortality and PM $<10\mu m$ in aerodynamic diameter (PM$_{10}$) differentiating the effects from Saharan dust and other human-made particulates, during Saharan dust days, in the city of Barcelona between 2003 and 2007. Identification of Saharan dust days was done as in our previous studies.1,2 Additionally, daily PM$_{10}$ Saharan contributions versus local contributions were calculated from data collected at a regional background site, using a monthly moving 40th percentile to the PM$_{10}$ time-series after omitting all days with Saharan air mass influence.4 These values were subtracted from the measured daily PM$_{10}$ concentrations at the regional background site to obtain the
amount of African dust brought on any given day under Saharan air-mass influence. Finally, the calculated daily African dust was used to distinguish between the urban background PM$_{10}$ data in Barcelona and daily PM$_{10}$ Saharan and non-Saharan contributions. We explored the association between daily cardiovascular mortality and PM$_{10}$ using a time-stratified case-crossover design. We used Poisson regression adjusting for temperature, humidity, public holidays, and influenza epidemics, as well as for the 3-way interaction between day of the week, month, and year, to control for seasonality and time trends. This was done to replicate the adjustment made by the case-crossover design with the time-stratified approach for the selection of control days.

Applying this source apportionment method, 50% of PM$_{10}$ concentrations on Saharan dust days were due to human-made or local sources (Table). During non-Saharan dust days, the percentage increase of risk (%IR) per 10 µg/m$^3$ at lag 1 due to PM$_{10}$ was 2.8 (95% confidence interval = 1.6 to 4.1). During Saharan dust days, we found larger estimates, for this same lag, from PM$_{10}$ attributed to the Saharan contributions (4.0 [−0.4 to 8.7]) and to the local contributions (9.7 [4.3% to 15.3%]) than on non-Saharan dust days.

Using a novel method to differentiate the Saharan dust contribution from our daily total PM$_{10}$, our results confirm the role of Saharan dust in triggering negative short-term cardiovascular effects. Intriguingly, however, our results also suggest that human-made particulates on those days have stronger effects than on other days, and that the effects observed in past studies may be driven mostly by the increase in effects of these specific contributions. We have no way of knowing the distribution of particle size of the local distribution, and therefore, we cannot determine whether this increase in effects is driven by the very fine traffic-related particles. However, it may be possible that the local particles become more toxic on Saharan dust days. Saharan dust contains Ca-Mg carbonates and other components that may react with pollutant gases (such as SO$_2$ or NO$_x$), thus forming new particles. Similarly, there could be chemical reactions with other gases or additional condensation of organic compounds on the particles, enhancing toxicity. While the toxicity of particles remains to be understood, these results reinforce the need to re-evaluate the current exemption during Saharan dust days that the European Union has legislated, because this allows for tolerating an average higher exposure to PM$_{10}$ in population, on the specific days when there are increased effects of both local and exogenous particles.

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REFERENCES

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