We would like to thank the reviewers for the helpful comments. Please find below our replies.

Anonymous referee #2:
Reply to general comments:
The concentration response functions (CRF) applied in the health impact function to estimate the excess mortality due to desert dust ($DU_{2.5}$) have been derived from relative risks (RR) based on the American Cancer Society (ACS), Cancer Prevention Study – II cohort study analyzed by Krewski et al., 2009. Relative risks for $PM_{2.5}$ were derived from the analysis performed for the years 1999–2000, using a standard Cox model adjusted for 44 individual-level and seven ecologic covariates. We state clearly and discuss that this is a critical assumption in our work. The ACS study used total $PM_{2.5}$ and did not provide information on the composition and toxicity of the individual particles. Although it is very likely that a part of the total $PM_{2.5}$ comes from airborne dust, the properties and toxicity of these particles, and the exposure conditions, are different from that in the regions where desert dust dominates (like North Africa, Middle East, Eastern Asia). The lack of long-term epidemiological studies in regions outside the United States and Europe, like the dust belt, and the little work that has been done on the RR due to individual anthropogenic and natural (mainly dust) components of fine particulate matter impose this assumption. Despite these limitations, the ACS study is one of the longest and most comprehensive cohort studies, it has been used and analyzed from leading groups in this research area, and is considered as one of the best available sources of data on the risk of mortality associated with long-term exposure to $PM_{2.5}$ (Cohen et al., 2004, Krewski et al., 2009, Evans et al., 2012). We acknowledge that the use of RRs from the ACS study strengthens our assessment, rather than the use of RRs based on short-term studies. Short-term mortality impacts, that are not addressed in RR estimates from the cohort studies, represent a very small percentage of the total mortality impact (Roman et al., 2008). We make this clearer in the revised manuscript.
In the revised manuscript we add the following part in the discussion (P24034, second paragraph, L16):
“A critical assumption in this work is, therefore, that desert dust $DU_{2.5}$ has the same health impact as $PM_{2.5}$ in the ACS epidemiological study. Despite this limitation, our estimates are based on this study because it is the most comprehensive one, and is considered as one of the best available sources of data on the risk of mortality associated with long-term exposure to $PM_{2.5}$ and is also considered representative for other regions (Cohen et al., 2004, COMEAP, 2009, Krewski et al., 2009, Evans et al., 2012).”

In this work we did not take into account climate conditions like temperature. Other factors such as active and passive smoking, occupational hazards, and personal characteristics are not expected to be confounding factors, since they do not vary with air pollution on a daily basis and are accounted for in the epidemiological studies (Ostro, 2004, Roman et al., 2008, Krewski et al., 2009). Personal and general characteristics vary among populations in different parts of the world, but this is already included in the RR assumptions.

Reply to specific comments (reviewer comments in italic):

- INTRODUCTION: - the authors state that "most of the studies that relate air quality and human health have focused on the impact of anthropogenic particulate matter". This is not true. Most of the studies were set in urban areas, however they used total PM2.5 as main exposure, without being able to distinguish the fraction originating from traffic and other anthropogenic sources, from the fraction originating from natural events such as desert dust advections. Only recently, studies have been focusing on specific components of PM, but results are still controversial. - Concentration-response functions (CRF) used for health impact assessment are from studies focusing on PM2.5. This should be clearly stated in the Introduction.

Reply: We correct this and revise the text in the introduction (P24025, line 7):
"Most of the studies that relate air quality and human health were performed in urban areas, mainly in United States and Europe, where the particulate pollution is dominated by anthropogenic particles (such as PM by combustion engines)."

- METHODS: - there are two main limitations, already acknowledged by the authors. First, CRF used in the analyses come from PM2.5, not desert dust. This implicated that size matters more than composition, which is questionable. Second, CRFs are from US studies, which do not represent the areas most affected by desert dust. Unfortunately, the authors did not provide sensitivity analyses on either assumption. I would suggest them to provide sensitivity analyses as, for example, using different CRFs from European or Asiatic studies, most affected by desert dust and representative of the interested areas. - All estimates of impact assessments should be complemented with estimates of statistical errors (standard errors, confidence intervals, etc.), and the corresponding methods should be reported in the Methods section

Reply: In addition to our reply in the general comments, particles from combustion processes are likely to be more toxic than total ambient $PM_{2.5}$ on average, while crustal material like dust may be less toxic (Tuomisto et al., 2008). On the other hand dust particles rapidly mix with acids and organic compounds, therefore in large urban and

industrial centers (e.g., Cairo, Beijing, Tehran etc.) where both dust and anthropogenic pollution concentrations are high it is likely that the toxicity of dust does not deviate from the average ambient $PM_{2.5}$. Please see also our reply to the reviewer #4.

Statistical errors: In the revised manuscript we add the following in the methodology section (P24029, right before the last paragraph):
"We carried out a statistical uncertainty analysis assuming random errors; by propagating the quantified errors of all terms in Eq. (1), estimated from the 95% confidence intervals (CI95) reported in the ACS studies. A detailed analysis of our uncertainty methodology is presented in Lelieveld et al. (2013)."
We present the statistical errors in the results and remove the corresponding paragraph in the discussion section (P24036, L11). The following paragraph has been added (P24031, right after the first paragraph):
"The quantified errors of all terms in Eq. (1), estimated from the 95% confidence intervals (CI95) reported in the ACS studies, shows that the global mortality estimates are quite robust with an uncertainty up to about ±5% for annual global dust induced mortality. At the country level the uncertainties are much higher (exceeding ±100%) for countries in North Africa and Middle East. Note that this analysis only addresses statistical uncertainty, while non-representativeness of the applied concentration response factors outside the USA and the toxicity issue mentioned above add to the uncertainty."

- RESULTS: - It is surprising (and against previous publications) that areas such as Europe are not affected by desert dust. Europe is very close to Sahara, and northern winds bring large amounts of dust to areas like Sicily, Greece and Cyprus (see Pey et al. 2013 ACP). Similarly, other areas in the far east, like Korea and Japan, are interested by desert dust advections, and some studies reported short-term health effects. - If Table 1 refers to premature mortality due to desert dust, this should be explicitly stated in the Table. Since countries in the dust belt are those largely affected by desert dust (Figure 1), it is obvious that they rank in the top positions of table 1, so the corresponding sentence in the Results section should be deleted. - Results in Table 2 seems to be inflated. They come from the ratio of the CPD from dust to the total CPD deaths. However, people exposed to desert dust are likely to be exposed to other environmental stressors as well, so I am not sure that percentages should add up. Maybe authors should be more explicit on what kind of assumptions they are making when computing the percentages in Table 2.

Reply: In this work we assess the impact in mortality of the long-term exposure to desert dust. We refer to the discussion part about short-term studies showing the adverse health impacts in regions like the Mediterranean and Eastern Asia, where dust

outbreaks frequently occur. Table 1 shows the top 20 countries with highest premature mortality due to desert dust. We made this clearer in the table description. As we mention in section 4 (sensitivity calculations), the number of countries with significant numbers of deaths expands by reducing the background from 7.5 μgm^{-3} to 0 dust concentrations, including countries from other regions like Turkey, USA, Russia, Ukraine, Japan, Mexico, Italy, Greece, Brazil, Spain and others. Table 2 refers to the countries with the highest percentage of dust induced CPD and LC mortality relative to the total mortality from CPD and LC. Mauritania and Niger with the higher percentage are two countries with high baseline mortality rates, small fraction of population above 30 years (30% and 27% respectively), and high median dust concentrations in 2005. Poverty in these countries (and in other in the dust belt) is problematic and cardiovascular diseases are more likely to be caused from indoor and outdoor air pollution rather other factors like bad dietary habits. In countries like Egypt, Iraq, and Saudi Arabia, with large urban and industrial centers it is likely that desert dust pollution is mixed with other anthropogenic pollutants thus the impact of dust in the cardiorespiratory system is more severe. These factors can explain the estimated percentages in table 2.

Anonymous referee #4:
For the main assumption of this work regarding the choice of CRF from the ACS-CPS-II cohort epidemiological study, please see replies to general and specific comments to the anonymous referee 2. The short-term impact studies are indeed helpful, because they can give indications about the health impacts of dust outbreaks. On the other hand the relative risks from short-term studies are not appropriate to apply in a long-term study.
We add this sentence in the discussion section (P24036, L7) of the revised manuscript:
‘‘Short-term exposure impacts in mortality, that are not included in relative risk estimates from cohort studies, represent a very small percentage of the total mortality impact (Roman et al., 2008).’’

Lung cancer: We agree that for the impact of $PM_{2.5}$ to lung cancer mortality, the presence of some carcinogenic substance processes are required. It is not known if $DU_{2.5}$ has the same impact in the lungs as other $PM_{2.5}$ components (e.g. substances from combustion processes). However, in countries with large urban and industrial centers (like China, Egypt, Pakistan, Iran), where both dust and anthropogenic pollution concentrations are high, and can rapidly mix, it is likely that the toxicity of dust does not deviate from other particulate constituents, and may acts in the lungs similarly to other $PM_{2.5}$ compounds. In the revised manuscript we emphasize this issue and play down the lung cancer mortality (e.g. removing the results from the abstract). We add the following lines to section 2 (P24028) and section 3 (P24031) respectively:

‘‘For regions that are strongly affected by desert dust particulates and also have different living conditions compared to the USA, like in many African, Middle East and Asian regions (with high baseline mortality rates), the ACS results are likely to be less representative (Cohen et al., 2005). In addition, although ACS cohort and other epidemiological studies associate exposure to ambient $PM_{2.5}$ with lung cancer mortality, there is no clear evidence between desert dust pollution and lung cancer risks. Similarly as the GBD assessment (Lim et al., 2012), we assume that $DU_{2.5}$ affects human health the same way as $PM_{2.5}$ in the ACS cohort study and therefore we implicitly assume that particle size and mass concentration matters more than their composition.’’
‘‘Also unsurprisingly, almost all lung cancer mortality is found to occur in the dust belt. We emphasize that these numbers are particularly uncertain as it is not known if dust has the same impact through lung cancer mortality as ambient $PM_{2.5}$. However, in countries with large urban and industrial centers (like China, Egypt, Pakistan, Iran), where both dust and anthropogenic pollution concentrations are high, and can rapidly mix, it is likely that the toxicity of dust does not deviate from other particulate constituents, and may acts in the lungs similarly to other $PM_{2.5}$ compounds.’’

Shape of the concentration response function: Regarding the shape of the CRF other studies use linear and other log-linear functions (Pope et al., 2002, Cohen et al., 2004, Ostro et al., 2004, Krewski et al., 2009, Pope et al., 2009, 2011). At higher concentrations than 30 $\mu g/m^3$ $PM_{2.5}$ there is no clear evidence which is the optimum shape of CRF for CPD and lung cancer mortality attributable to $PM_{2.5}$. We refer for the influence of the shape of the exposure response function associated with $PM_{2.5}$, to the sensitivity analysis by Cohen et al. (2004, 2005) who used the same CRF. They considered three alternatives: (case 1) no increase in excess mortality above 30 $\mu g/m^3$ $PM_{2.5}$, (case 2) linear increase of excess mortality above 30 $\mu g/m^3$ $PM_{2.5}$, and (case 3) increase with the log of the $PM_{2.5}$ concentration across the entire range. The 30 $\mu g/m^3$ level represents the annual average $PM_{2.5}$ concentration that is typically not exceeded in most cities in Europe and North America, while in developing countries concentrations in cities are frequently much higher (Cohen et al., 2004). It is also the level up to which linearity of the CRF has been confirmed (Krewski et al., 2009, Pope et al., 2011). It was found that in case 1, excess mortality estimates decrease by 27-29%, largely in developing countries, while in case 2, mortality increases in Eastern and Southeastern Asia and in some parts of North Africa and Middle East. In case 3, the RR increases more steeply below 30 $\mu g/m^3$ $PM_{2.5}$. As a consequence, mean excess mortality is calculated to be about 63% higher in regions with relatively low $PM_{2.5}$ concentrations (eg USA, Europe) while the mortality in regions with high concentrations is relatively insensitive to the assumption of a log-linear CRF (Cohen et al., 2004). Roman et al (2008) also state that the relatively largest uncertainty in estimated effects on mortality is expected at lower $PM_{2.5}$ concentrations.

We performed a sensitivity calculation applying a log-linear CRF as suggested by the reviewer. Our results show about 14% lower CPD mortality and about 6% lower lung cancer mortality. This outcome agrees with other studies indicating that the assumption of the CRF shape is more critical for relatively low $PM_{2.5}$ concentrations. We included this sensitivity case to the section 4 (sensitivity calculations) and further discuss it in section 5 of the revised manuscript.

We added the following text to the discussion section:
"The assumption about the relationship between mortality and the associated exposure to $PM_{2.5}$ contributes to the uncertainties. Several studies use both linear and log-linear functions for the shape of the CRF (Pope et al., 2002, Cohen et al., 2004; Ostro et al., 2004; Krewski et al., 2009; Pope et al., 2009, 2011). For higher $PM_{2.5}$ concentrations than 30μgm^{-3} $PM_{2.5}$ there is no clear evidence which is the optimum shape of CRF for CPD and lung cancer mortality attributable to $PM_{2.5}$. Pope et al. (2011) conclude in their discussion that for cardiovascular mortality the exposure response function is non-linear with a steep increase in risk at low exposures and flattening out at higher exposures. For lung cancer they found that a nearly linear response function can be appropriate. Cohen et al. (2004) presented sensitivity analyses to compare to their base-case estimates, in which the burden of disease was estimated by applying the ACS CRF over the range of 7.5 to 50μgm^{-3}. In the sensitivity analysis they considered three alternatives: (case 1) no increase in excess mortality above 30 μgm^{-3} $PM_{2.5}$, (case 2) excess mortality increases linearly above 30 μgm^{-3} $PM_{2.5}$, and (case 3) excess mortality increases with the log of concentration of $PM_{2.5}$ across the entire range. They found that in case 1, excess mortality estimates decrease by 27-29%, worldwide. The extrapolation of the ACS coefficients to the higher $PM_{2.5}$ concentrations on the linear and logarithmic scales (cases 3 and 4 respectively), results in increases of 10% and 12% in the estimated CPD mortality, and 8–24% in the estimated lung cancer mortality, relative to the base-case estimates. In case 2, mortality increases in Eastern and South/Southeastern Asia and in regions in North Africa and Middle East. In case 3, mean excess mortality is calculated to be about 63% higher in regions with relatively low $PM_{2.5}$ concentrations (e.g. USA, Europe), while the mortality in regions with high concentrations remained unchanged or slightly reduced (Cohen et al., 2004). Evans et al (2012) obtained similar results. In their study mortality was substantially greater under the log-linear versus the linear case for regions with relatively low concentrations of $PM_{2.5}$. However global mortality estimates were only slightly higher with the log-linear compared to linear case, since the regions with large populations and high pollution levels, which contribute most to the global mortality, had lower mortality estimates under the log-linear case (Evans et al., 2012). Our analysis shows that in regions with high $PM_{2.5}$ exposure like the dust belt (where dust dominates total particulate pollution) the excess mortality due to $DU_{2.5}$ is slightly lower (about 14% and 6% for CPD and LC

respectively) with the log-linear function compared to the linear CRFs. As indicated in previous studies, the shape of the CRF is more critical at lower $PM_{2.5}$ concentrations where largest uncertainty in estimated effects on mortality is expected (Cohen et al., 2004, Roman et al., 2008, Evans et al., 2012)."

In the discussion part (P24036, comparison to previous work) we included a reference and comparison to Evans et al., 2012.
"In a recent study, Evans et al. (2012) estimated the expected number of deaths from all causes, cardiopulmonary diseases, lung cancer and ischemic heart disease due to chronic $PM_{2.5}$ exposure. They used risk coefficients based on the ACS cohort study and $PM_{2.5}$ concentrations from satellite retrievals. In their assessment they accounted for $PM_{2.5\_total}$ and $PM_{2.5\_no\ dust}$. They found that the $PM_{2.5}$ mortality in the Mediterranean region (several countries in North Africa and Middle East) was mainly related to the non-anthropogenic component of total $PM_{2.5}$. In their base case scenario (linear CRF, reference exposure 5.8μgm^{-3}), they estimated about 2.48 million deaths for CPD and 222 thousand for lung cancer attributed to total $PM_{2.5}$, and about 1.65 million and 170 thousand CPD and lung cancer deaths respectively after the removal of the natural dust component in 2004. The difference of about 830 thousand for CPD and 52 thousand for lung cancer in mortality indicates the impact of natural dust. These estimates show significant higher dust induced global mortality compared to our central assessment, and compared to the sensitivity case where we used annual mean dust concentrations. The use of 5.8μgm^{-3} as background $PM_{2.5}$ concentration (we used 7.5 μgm^{-3}), and annual mean $PM_{2.5\_total}$ and $PM_{2.5\_no\ dust}$ satellite concentrations (we used modeled median or mean $DU_{2.5}$ concentrations) may explain to some extent the discrepancies."

Anonymous referee #5
Reply to general comments:
We believe that the title, the structure and the context of the manuscript make sense and describe clearly the objective of this work. The methodology we follow, to assess the impact of long-term exposure to desert dust, is well addressed in previous studies. In the revised manuscript we changed the first lines of the last paragraph of the introduction as:
"The aim if this work is to investigate the impact of natural dust to human health and specifically to premature mortality by cardiopulmonary diseases. We assess the effect of the long-term exposure to airborne desert dust particles with an aerodynamic diameter smaller than 2.5 μm ($DU_{2.5}$) on human mortality for the year 2005 in the 231 countries distinguished by the United Nations."
In the revised manuscript we state clearer the objective of this study. We investigate the impacts of long-term exposure to natural desert dust $DU_{2.5}$ on human health and

specifically on premature mortality from cardiorespiratory diseases. Airborne desert dust is the main natural particulate pollutant.

Reply to specific comments (reviewer comments in italic):
- For the calculation of the concentration - response function (CRF) the authors use those estimated by Krewski et al (2009) calculated for cities with a range of PM2.5 between 5.8 to 22.2 µg/m3 and the linearity of the relationship having been tested only to a level of 30. Beyond this limit it cannot be assumed what kind of relationship exists between exposure (DU2.5) and response (mortality). If we consider that precisely in the most exposed areas 30 µg/m3 are exceed by far, it is questionable to think that the results of the study may be valid from an epidemiological point of view. Additionally Aneberg et al (2010) found that mortality estimates were highly sensitive to the PM2.5 thresholds and to different CRFs. o Moreover, it can not be assumed that the associations between PM2.5 and mortality found in the United States are valid in all analyzed regions since the composition and toxicity of these PM2.5, the patterns of exposure in their populations etc. are very, very different. So is quite questionable to apply the same CRF to mineral PM in other regions. o Our experience is that CRF in short term effect in the Canary Islands, with PM levels highly influenced by mineral dust, are quite different from other urban regions (López-Villarrubia,E.,etal.,Characterizing mortality effects of particulate matter size fractions in the two capital cities of the Canary Islands. Environ. Res. (2011), doi:10.1016/j.envres. 2011.10.005).

Reply: For the assumptions of the use of CRFs from the ACS-CPS study in other parts of the world, the toxicity issue of the different $PM_{2.5}$ components and the shape of CRF please see our replies to the anonymous referees #2 and #4.
The short-term effect studies are very important indicators to motivate and support long-term cohort studies. However, for the impacts of the long-term exposure to natural dust, we cannot use data from short-term studies. Short-term mortality impacts, that are not included in RR estimates from the cohort studies, represent a very small percentage of the total mortality impact (Roman et al., 2008).
We performed a sensitivity analysis assuming different threshold concentrations, i.e. 0, 5, and 10 μgm^{-3} and compared to our central results (with 7.5 μgm^{-3}).

- According to GBD project the main problems associated with air pollution are respiratory infections in children under 5 years and mortality from lung cancer and cardiorespiratory disease in people over the age of 30. On the other hand in the Krewski cohort the study population was restricted to persons who were at least 30 years of age and who where members of households with at least one individual 45 years of age or older. The authors refer to premature mortality, but it is not really so, (this would be caused among people from 1 to 65 or 70 years depending on the life expectancy of

each country). Personally it gives me the impression that the authors do not leave clear in the manuscript which is the criterion for selecting the older than 30 years in the mortality indicators.

Reply: We state clearly in the manuscript that we address the population of 30 years and older, to be consistent with the ACS CPS-II cohort epidemiological research, addressing same age group. (Please see methodology section: "…Pop is the total population with an age of 30 yr and older exposed to the pollutant. This age category coincides with the epidemiological studies in which the CRFs for different causes of mortality have been derived"

- *There is another important issue: the validity of mortality data for specific causes in certain countries. Probably is in the most exposed areas to desert dust, where mortality and morbidity information systems have to improve. Much more if this information has to be compared with that of other countries. Uncertainty ranges (WHO) "is generally larger for deaths from specific diseases than for all-cause mortality. For example, the relative uncertainty for deaths from IHD ranged from ±12% for high-income countries to ± 25–35% for countries in Sub-Saharan Africa". Limitations in these highâˇARˇmortality regions reinforce the need for caution when interpreting global comparative cause of death assessments*.

Reply: We agree that mortality and morbidity data has to be improved worldwide and especially in countries in Asia and Africa being most affected by desert dust and particulate pollution in general. The accessibility to national mortality and morbidity data is very difficult or sometimes times impossible. We believe that the WHO does a great effort to evaluate and record this kind of data. Please note that other studies (including GBD) work with the same data sets. We hope that our study helps motivate the appropriate epidemiological studies.

- Authors do not present the 95% confidence intervals for mortality indicators.

Reply: Please see our reply above (reviewer #2).

- In some sections it is not specified if they are using, number of deaths, rates.

Reply: We will check the manuscript for more consistency.

- The article lacks some tables that are necessary for knowing health information on used data: baseline mortality rates, population, YLL0 etc.

Reply: We believe that in the methodology section we adequately describe the data we used for our analysis.

- Finally, and perhaps most striking, is that despite the computational effort that has been made: The objective is not well defined and the methodology used to achieve the objective (that could be deducted after the reading of the manuscript) requires a number of very questionable assumptions: ï´C ˇg linearity of the CRF curve, ï´C ˇg that the urban PM25 has the same toxicity than those of desert origin and on populations with quite different social and demographic characteristics. ï´C ˇg The validity of health outcomes information in some regions

Reply: Please see replies above and to reviewers #2 and #4.
In the revised manuscript we discuss, as suggested, a sensitivity case where a log-linear CRF was used.

Anonymous referee #3:
In the revised manuscript we state clearer in the introduction the aim of our study, and introduce better the available epidemiological studies that relate dust pollution to human mortality.
For the use of epidemiological parameters (RR, CRF) from the ACS-CPS-II to other parts of the world like the dust-belt, please see our replies above to the reviewers #2, #4, #5 and the inclusions to the revised manuscript. For the association of airborne desert dust to lung cancer mortality, please see the reply to reviewer #4. We address this issue in the methods section and in the results, and have presented these results less prominently and more carefully.