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Deliverable

D 2.5 Inflammatory risk regional maps

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Changes with respect to the DoW

Issue	Comments
Delay of deliverable	<p>The PO was informed timely about this delay and accepted it on 16 November 2018 via e-mail.</p> <p>The simulations necessary for this deliverable were planned to be calculated on the new established computer cluster in Abidjan (Université Félix Houphouët-Boigny). This computer cluster is installed and functional since end of 2017 but due to some internal problems the official opening is postponed since then monthly.</p> <p>Due to this fact the simulations now had to be calculated in Toulouse but this could only start with a delay.</p>

Dissemination and uptake

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Table of content

Introduction	5
1 Methodology	5
1.1 Description of parameters used in the modelling chain issued from DACCIWA experimental program.....	8
1.1.1 Emissions	8
1.1.2 Relative risk values (or dose response function)	8
1.1.3 Concentration inflammatory response function	9
2 Results	10
2.1 Aerosol concentrations	10
2.2 Health Impact assessments	11
2.2.1 Inflammatory risk regional maps	11
2.2.2 Estimation of premature deaths	12
3 Conclusion	14
4 References	15

Introduction

The main objective of the final task (2.5) was to produce regional distributions of inflammation risks and mortality for Africa from results obtained in the different previous tasks (2.1 to 2.4). A number of simulations with RegCM4 new developments (Task 2.2) were conducted with this modelling chain using regional emission inventories (Task 2.1, D2.1) for different scenarios (all sources, anthropogenic sources only, domestic fire source only and traffic source only). Regional distributions of concentrations of the different aerosol components were then obtained for each simulated scenarios. PM_{2.5} concentrations were calculated from these data following the relationship described in Task 2.2 (D2.2). Then, dose/response functions determined for the different studied sites (and sources) in Task 2.3 (D2.3) were used in the epidemiological model developed in Task 2.2 to calculate regional distributions of HIA (health impact assessments) in terms of mortality. In this deliverable, results of health impact for anthropogenic sources were estimated. Also, a tentative calculation of regional distributions of aerosol inflammatory impact through IL6 biomarker plotting, was performed from modelled OC (and EC) concentrations and the ratio between OC (and EC) and IL6 inflammatory biomarkers obtained in the Task 2.4, source by source (D2.4). Note that these impacts are here presented for January 2015, by considering a scenario with anthropogenic sources only (traffic, domestic fire, waste burning and industrial sources).

1 Methodology

Figure 1 presents the methodology used in WP2 to determine regional distribution of inflammatory risks, diseases and mortality. As we may notice, such result is obtained at the end of a modelling chain.

The model used for this modelling chain in WP2 is RegCM4. RegCM4 from International Centre for Theoretical Physics is a hydrostatic model with compressible sigma-p vertical coordinates that uses the dynamic core of Mesoscale Meteorological Model version 5 (MM5) from the National Centre of Atmospheric/Pennsylvania state University (NCAR/PSU's MM5; Grell et al., 1994). The Regional Climate Model (RegCM) has been widely used in Africa for several studies on climate variability, aerosols and their impact with good performance (Sylla et al., 2009; Konare et al., 2008; Solmon et al., 2008; 2012; Zakey et al., 2006; Tummon et al., 2010; Malavelle et al., 2011). Its accessibility and large community support contribute to its wide usage in several research teams on the African continent and overseas. These works are generally performed at quite coarse spatial resolution of 30 – 50 km with focus on dust, carbonaceous and sulphate aerosols using simple to complex chemistry (Solmon et al., 2006; Konare et al., 2008; Touré et al., 2012; Shalaby et al., 2012) options.

During DACCIWA task 2.2, RegCM4 model was configured to be consistent with WP2 aims (see D2.2). Note that all the tests were done for the year 2005 and 2015. (1) RegCM4 was configured at the resolution of 15 x15 km over Africa. (2) Secondary organic particle determination was improved: up to now, modelled primary OC was multiplied by 1.4 to obtain total organic carbon by including

SOA formation. According to different recent experiments on primary and secondary organic carbon, an exhaustive literature review was conducted to assess $OC_{\text{primary}}/OC_{\text{total}}$ ratios directly obtained at the source. From this compilation, the coefficients were applied on primary OC emission sources to finally transport total OC particles. (3) the two aerosol modules included in RegCM4 model (AERO simple model and DDCB explicit and complex model using ISORROPIA model, Nenes et al., 1998a, b; Fountoukis and Nenes, 2007) were tested to correctly handle water-soluble aerosol estimation and to limit simulation costs. AERO module given SO_4 particles from SO_2 gas was selected; (4) Finally, $PM_{2.5}$ calculations were fixed from RegCM4 outputs with the following relationship:

$$[PM_{2.5}] = [BC] + 75\% [OC] \times 1.7 \times 1.4 + 90\% [SO_4] \times (1/0.43) + [PM_{2.5}|_{\text{dust}}] + [PM_{2.5}|_{\text{seasalts}}] \quad (1)$$

where BC is directly given by the model.

Fine water soluble is obtained by multiplying respectively sulphate given by the model in the AERO option by 0.90 (to consider only fine WS particles) and by 1/0.43 (to convert sulphate into water soluble particles).

Fine POM is obtained by multiplying total OC given by the model by 0.75 and 1.7 (to respectively consider fine particle and convert total OC into POM).

Both dust and sea salt particles are emitted on-line with the parametrizations given in Zaakey et al. (2006) and Zaakey et al. (2008), respectively.

All these new configurations summarized in the table 1 were validated with a simulation for the year 2015 including the recent anthropogenic inventory developed in DACCIIWA task 2.1. As shown in the figure 1, comparisons with BC aerosol concentration and ground AOD measurements performed in the task 2.2 and 2.3 associated to satellite AOD were conducted for such a purpose.

Then, a health module to estimate different health impacts has been developed, based on RegCM4 outputs. Excess mortalities due to different emission scenario of $PM_{2.5}$ have been calculated with the following relationships (Anenberg et al., 2010; Lelieveld et al., 2013)

$$\Delta\text{death} = y_0 \times (1 - \exp(-\beta \Delta PM_{2.5})) \times \text{POP} \quad (2)$$

where $\Delta PM_{2.5}$ values are given by the model.

y_0 is the baseline mortality rate for different mortalities given by World Bank database and WHO Department of Health Statistics and Informatics, and POP is the exposed population (here >30yrs) given by Global Population of the World version 3 (GPWv3). β is the concentration response factor obtained from RR, which is the relative risk (or dose/response functions as shown in figure 1) for a $10\mu\text{g}/\text{m}^3$ $PM_{2.5}$ increase ($RR = \exp(\beta \times \Delta PM_{2.5})$). Choices of RR will be later detailed.

Regional distributions of Inflammatory risk were calculated from regional modelled aerosol concentrations multiplied by a Concentration inflammatory response function obtained from experimental relationships between aerosol concentrations and biomarkers (also called dose response function in figure 1). Values for such a ratio will be also below detailed.

Finally, following this methodology and parameters (table 1), simulations have been performed for the year 2015 for different emission scenarios which will be below detailed.

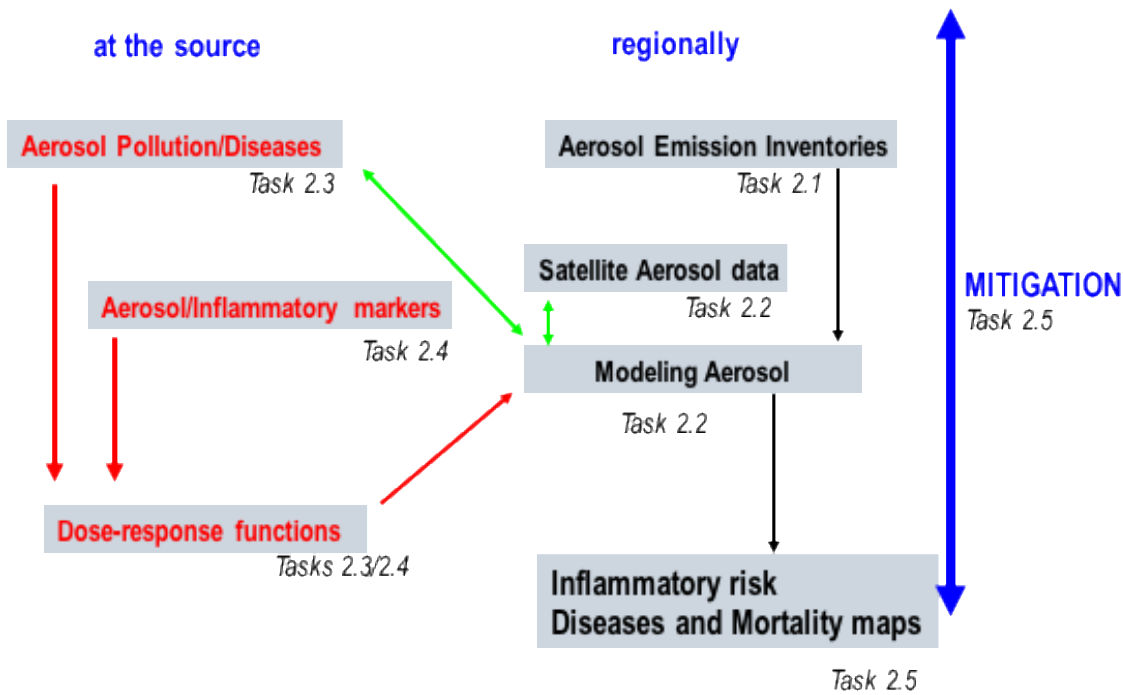


Figure 1: Methodology used in WP2 to determine regional distribution of inflammatory risks, diseases and mortality.

Table 1: Configurations of RegCM4 simulations

Year	2015
Spatial resolution	15 km x 15 km
Land surface	Biosphere-Atmosphere Transfer Scheme (BATS, Dickinson et al., 1993).
Convection scheme	Tiedtke, (1989)
Vertical levels	23 levels
Large scale precipitation	scheme from Sundqvist et al. (1989)
Emissions	Biomass burning: corrected GFASv1.2 (multiplied by 3.4 as suggested by Kaiser et al., 2012) Anthropogenic emissions including traffic, cooking, charcoal making, waste burning, industries, power plant, flaring (see the deliverable D2.1 on Emission inventories of WP2 (Keita et al.))
Chemistry option	AERO (Aerosol)
Dust module	4 bins dust module (Zakey et al., 2006).
Sea Salt module	2 bins sea salt (Zakey et al., 2008)
SST	Weekly Optimum Interpolation SST (OIWK)
Meteorological field forcing	NNRP2 (not available 2015 EIN15 data)

1.1 Description of parameters used in the modelling chain issued from DACCIWA experimental program

Here we detail the parameters retrieved from our experimental tasks for the simulations described above.

1.1.1 Emissions

Data are obtained from the task 2.1. Different scenarios were studied. A first scenario called TOT include all the sources described in the table 1. Dust and sea salt on line emissions are activated. Biomass burning inventories are given by GFASv1.2 multiplied by 3.4 as suggested by Kaiser et al., 2012 whereas anthropogenic emissions including traffic, cooking, charcoal making, waste burning, industries, power plant, flaring are given by the DACCIWA inventory developed in task 1 (see task 2.1, Keita et al., in preparation). A second scenario called ANTH is for anthropogenic emissions. It only includes the DACCIWA anthropogenic inventory including traffic, cooking, charcoal making, waste burning, industries, power plant, flaring. Note that 2 other scenarios (not presented here) have been also studied: one including only domestic fires and the other dealing with traffic only.

1.1.2 Relative risk values (or dose response function)

As shown in figure 1, such data were obtained from the task 2.3 (see D2.3). Indeed, the main objective of this task was to establish links between health and air pollution (PM_{2.5}, NO₂ and O₃) for the different studied sources in order to obtain statistical dose-response functions for respiratory diseases and mortalities. Note that as the specific sample size for both NO₂ and O₃ were limited, focus was put only on PM_{2.5} for Côte d'Ivoire. Weekly characterization of PM_{2.5} mass was performed from December 2014 to March 2017 in our three sites of measurements (domestic fires, traffic and waste burning sites). Daily census of hospital admissions for respiratory diseases, cardiovascular, dermatological problems and mortality with African medical doctors were performed with the same methodology in 5 medical centres located nearby our measurement sites, in June-August (2015 and 2016) and in December-February (2016 and 2017). Moreover, thanks to Cocody hospital, data on emergency room visits were also collected for the year 2016. Relative Risks (RR) or dose response function of the relationship between both datasets were then estimated using a Poisson Regression model. Analyses were further separated by district (Cocody (waste burning), Adjamé (traffic), Yopougon (domestic fire) as well as by season (rainy, dry) and year. Table 2 presents the cases with a significant relative risk above 1.0, which indicates the existence of a risk. In most cases, risks were only observed during the rainy season. The exception is that the risk of non-urgent respiratory symptoms is present throughout the year in Yopougon, which had the highest levels of PM_{2.5}. These RR are valid for long term (several months) exposure to PM_{2.5}. No significant results were observed for the dry season only.

Table 2: Relative risk values obtained in task 2.3.

	Health Outcome	RR/10 μ g/m ³
YOPOUGON	Respiratory Outpatient Visit (annual)	1.001
	Respiratory Outpatient Visit (rainy)	1.003
	Cardiac Outpatient Visit	1.004
	Dermatology Outpatient Visit	1.002
ADJAME	Respiratory ER Visit	1.029
	Cardiac ER Visit	1.030
COCODY	Respiratory ER Visit	1.033
	Cardiac ER Visit	1.032
	Mortality ER Visit	1.042

These first measurements are encouraging since there are the first RR measured in West Africa. It may be observed that values are surprisingly lower than those measured in Northern countries and used by Lelieveld et al. (2013) or Anenberg et al. (2010) (table 3). In our view, it underlined the need to go further in the crossed measurements, e.g. to better harmonize chemical and health sampling in terms of time step since, as recalled, PM_{2.5} mass and health outcome census are weekly and daily studied respectively. It is important to note that thanks to DACCIWA, this work is now on-going over Abidjan with the regional PASMU program. By expecting these new values, RR usually given by the literature (table 3) was first applied in the modelling chain.

Table 3: Relative risk values given by literature.

Health Outcome	RR/10 μ g/m ³
All causes (annual)	1.06
Lung cancer	1.06
Cardiopulmonary	1.11
Respiratory	1.06

1.1.3 Concentration inflammatory response function

As shown in figure 1, concentration inflammatory response function data are obtained from the task 2.4 (see D2.4). To our knowledge, this is a new initiative to quantitatively determine such data. For this purpose, a cross-statistical analysis between physico-chemical and toxicological results was realized for January and July 2016. For each studied sites (domestic fire, traffic and waste burning sites), physicochemical results include mass, carbonaceous aerosol, organic and non-organic water-soluble particles and trace elements for each aerosol size classes (ultra-fine, fine and coarse particles) whereas toxicological results include biological reactivity of aerosols through in-vitro measurements of inflammation markers. Correlations and slopes giving values of concentration inflammatory response ratios (CIR) have been scrutinized between size/chemical compounds and biomarkers dataset for each sites and campaigns. Table 4 presents CIR ratios obtained with significant correlations occurring for EC, OC and WSOC, all size pooled (since the modelling outputs do not include size information). Maximum CIR ratio values are observed for domestic fire site in the dry season whatever the chemical compounds. This difference (but less marked) is also observed

in both traffic sites. In waste burning site (not shown here), it may be observed weaker correlations and ratios due to the higher distance of the source than in other sites. Globally for all the sites, correlations between inflammation markers and EC, OC and WSOC particles are better in dry season than in wet season whatever the sizes, even if there are higher for coarse and ultrafine particles.

Table 4: Concentration inflammatory response ratios obtained from task 2.4 results.

	Aerosols	Concentration/Inflammatory response ratio	
		Dry	Wet
Domestic fire site	EC	386	65
	OC	82	10
	WSOC	130	27
Traffic site	EC	166	45
	OC	82	10
	WSOC	130	27
All sites pooled	EC	221	74
	OC	64	11
	WSOC	136	30

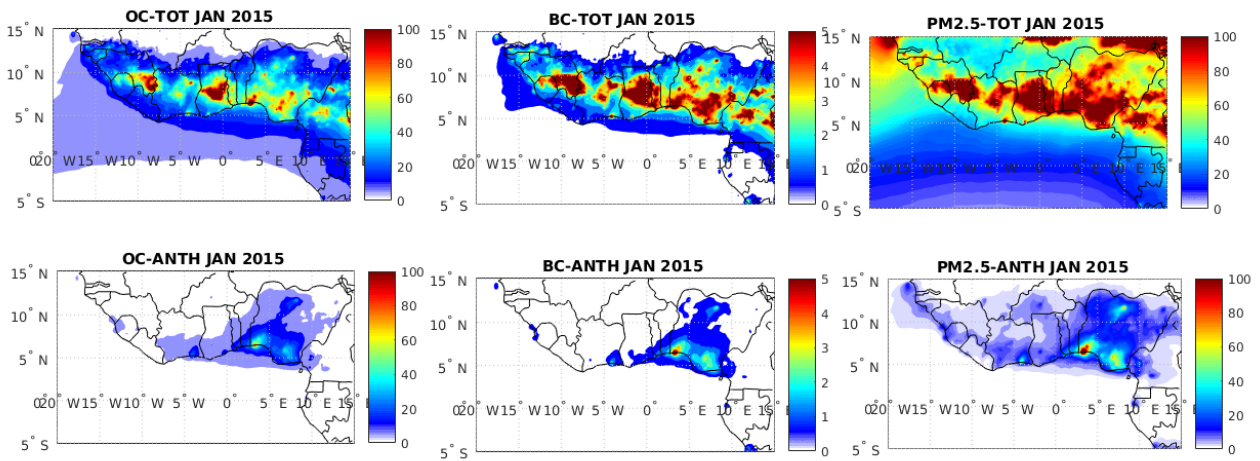
2 Results

Results of our modelling chain, associated to the parameters needed to estimate health impacts are now presented. Focus is put on simulations for January 2015. The scenario which is studied is the scenario including all the anthropogenic sources (traffic, domestic fires, industries, power plants, flaring and waste burning sources) with the parameters in red-underlined in paragraph 1.1.3.

2.1 Aerosol concentrations

Figure 2 presents spatial distributions of OC, BC and PM_{2.5} (in $\mu\text{g}/\text{m}^3$) given by the modelling chain for January 2015, for anthropogenic scenario (ANTH) and for all sources (TOT). As described in D2.2 when all the sources are included, BC and OC concentrations peak above savanna areas due to biomass burning sources which are high at this period of the year with a few spots over the coast due to anthropogenic sources. The important dust aerosol source is also stressed in PM_{2.5}-TOT figure in the north of the window. Let us note that the comparisons performed between simulation results and DACCIWA measurements have been shown to be suitable. Considering the anthropogenic scenario simulation (ANTH), the relative importance of anthropogenic source predominance over the coasts is revealed.

Figure 2 shows important concentrations of BC, OC and PM_{2.5} above all West-African cities: Dakar, Conakry, Abidjan, Accra, Lomé, Cotonou, Lagos, Abuja, Port-Harcourt and Yaoundé with maximum values in Nigeria and Côte d'Ivoire. It is interesting to mention (figure 3) that the relative contribution of anthropogenic sources in aerosol is important, representing in average, 45% of the total BC, OC and PM_{2.5} at Abidjan whereas 40% at Cotonou. The other important sources being the biomass burning.



Figures 2: Spatial distributions of OC, BC and PM2.5 (in $\mu\text{g}/\text{m}^3$) given by RegCM4 modelling in January 2015, for all sources (TOT) and for anthropogenic sources (ANTH).

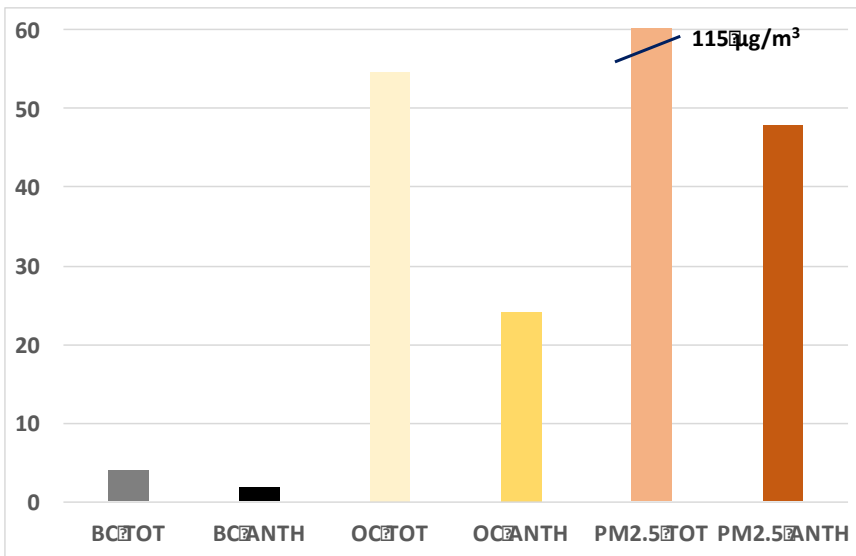


Figure 3: Mean BC, OC and PM2.5 modelled concentrations in Abidjan in January 2015 for all sources (TOT) and for anthropogenic sources (ANTH).

2.2 Health Impact assessments

2.2.1 Inflammatory risk regional maps

Inflammatory risk regional maps are obtained at the end of the modelling chain from aerosol concentration and concentration inflammatory response (CIR) ratios. Figure 4 presents inflammatory risks due to OC particles (left graph) and to EC particles (right) in January 2015 due to anthropogenic sources only.

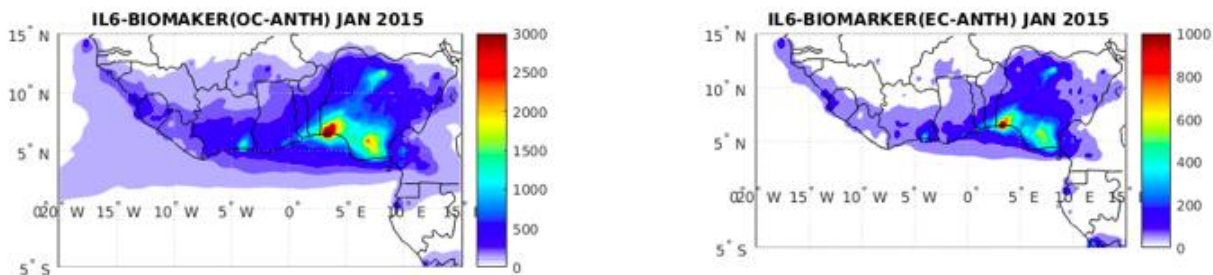


Figure 4: Inflammatory risk regional maps due to OC particles (left) and to EC particles (right) for January 2015 and for anthropogenic sources over West Africa.

As expected, regional distribution of inflammatory risks is comparable with regional distribution of pollutants. In terms of intensity, inflammatory risks are roughly three times higher for OC than for EC. Figure 5 proposes a zoom on Cote d'Ivoire, detailing the areas where inflammatory risks are important.

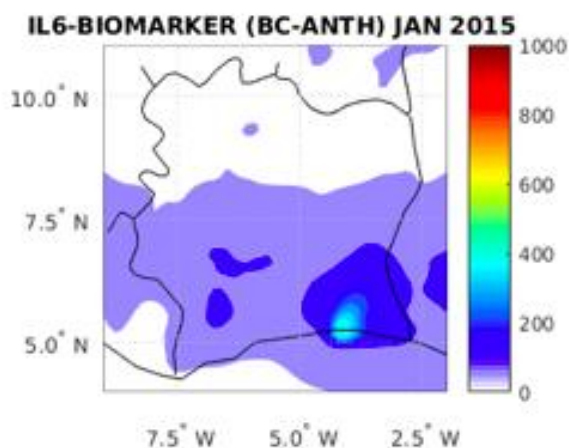


Figure 5: Inflammatory risk regional map due to EC particles for January 2015 and for anthropogenic sources over Cote d'Ivoire.

Such representation will be interesting to be obtained for the on-going simulations (domestic fire only and traffic only scenarios) in proposing to policy makers, a proxy able to quantify the exposure risk of populations to inflammatory problems due to different choices of sources.

2.2.2 Estimation of premature deaths

The second health indicator obtained at the end of our modelling chain deals with the estimation of mortality (all causes, cardiovascular, lung cancer and respiratory diseases causes) due to anthropogenic PM2.5 aerosol. This is obtained from Equation (2) with parameters given in paragraph 1.1.2.

Figures 6 and 7 present premature death estimations for all causes and respiratory diseases respectively, for each African country in January 2015. As it may be observed, highest premature

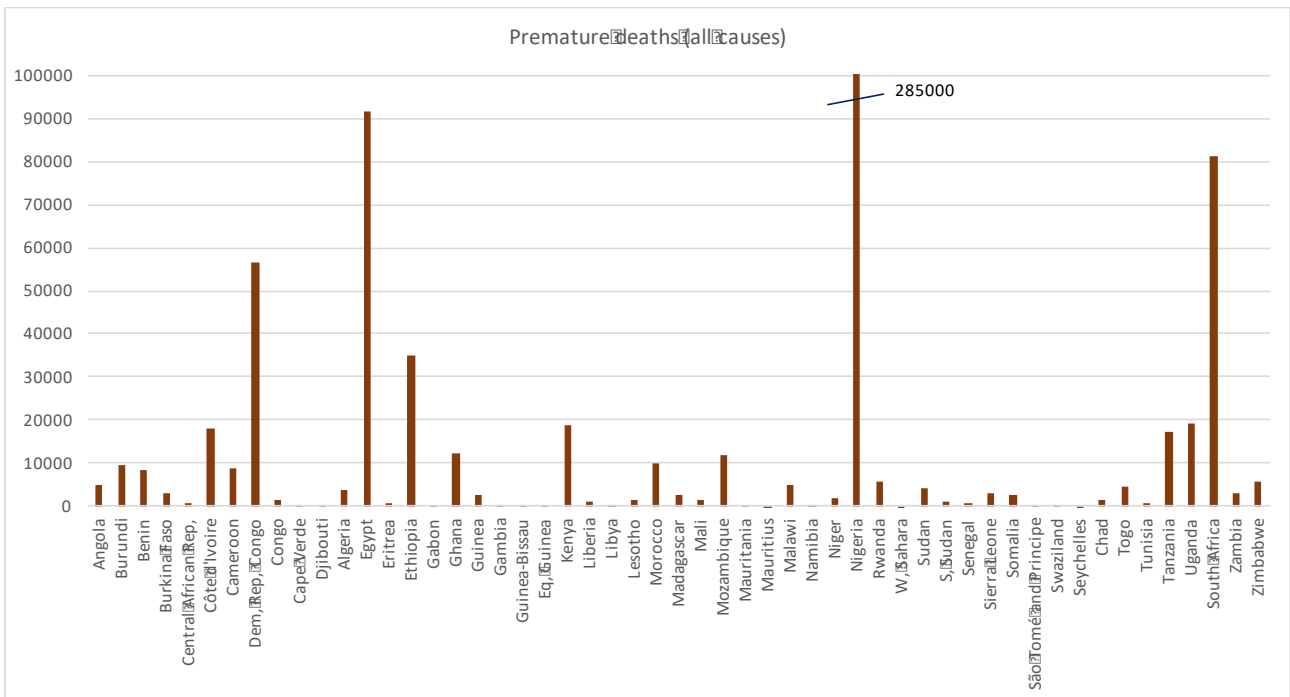


Figure 6: All causes premature death modelled estimations due to anthropogenic PM2.5 for January 2015.

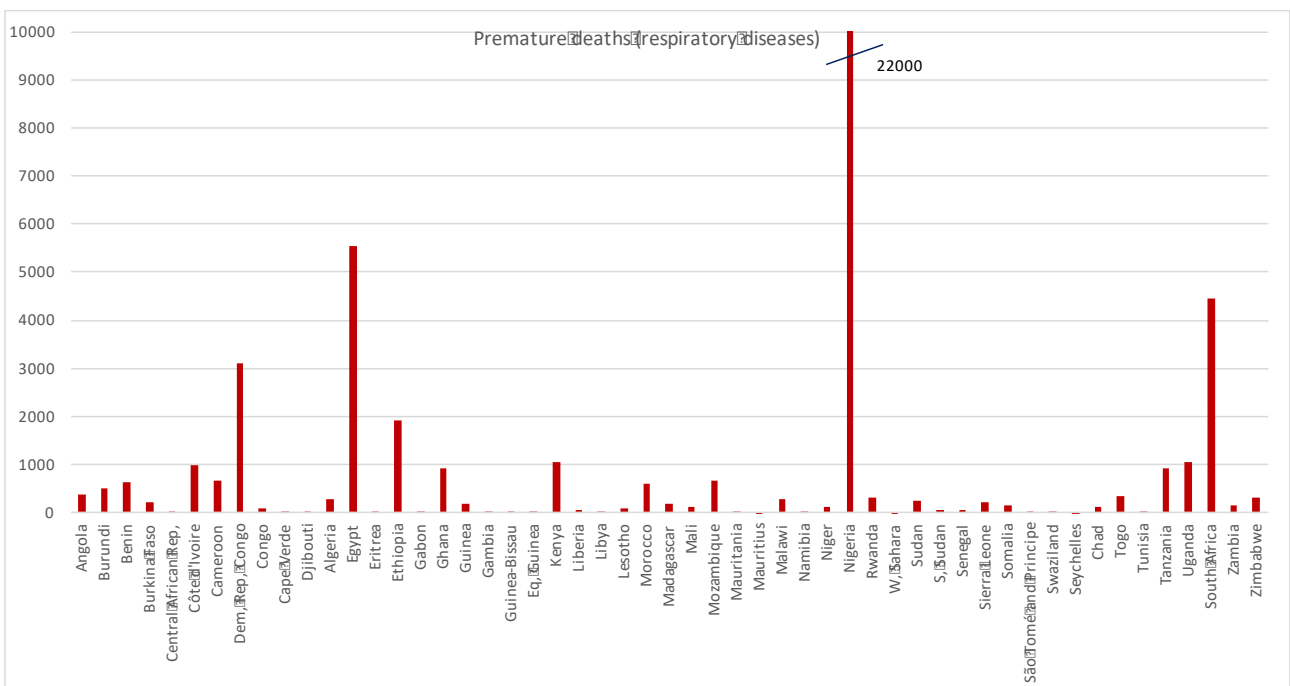


Figure 7: Respiratory disease causes premature death modelled estimations due to anthropogenic PM2.5 for January 2015.

death numbers for adults older than 30 years are found in Nigeria, South Africa, Egypt and DRC Congo. Cote d'Ivoire and Benin are in the second rank of importance with respectively 18223 and 8479 premature deaths of all causes. It is interesting to underline that 6% and 8% of these death numbers may be attributed to respiratory diseases whereas 42% and 56% to cardiovascular diseases. Regional distribution of all causes premature deaths is presented in figure 8 for Cote d'Ivoire, stressing high values above biggest cities such as Abidjan, Bouaké, Khorogo. Abidjan

district relative contribution is about 40% of Cote d'Ivoire premature deaths (table 5). All these numbers are attributed to PM2.5 emitted by all the anthropogenic sources.

It is also interesting to note that regional distributions of inflammatory risk (figure 5) and premature deaths (figure 8) are comparable.

With the on-going simulations taking into account domestic fire sources or traffic sources only, we will be able to estimate how much premature death values are due to each source.

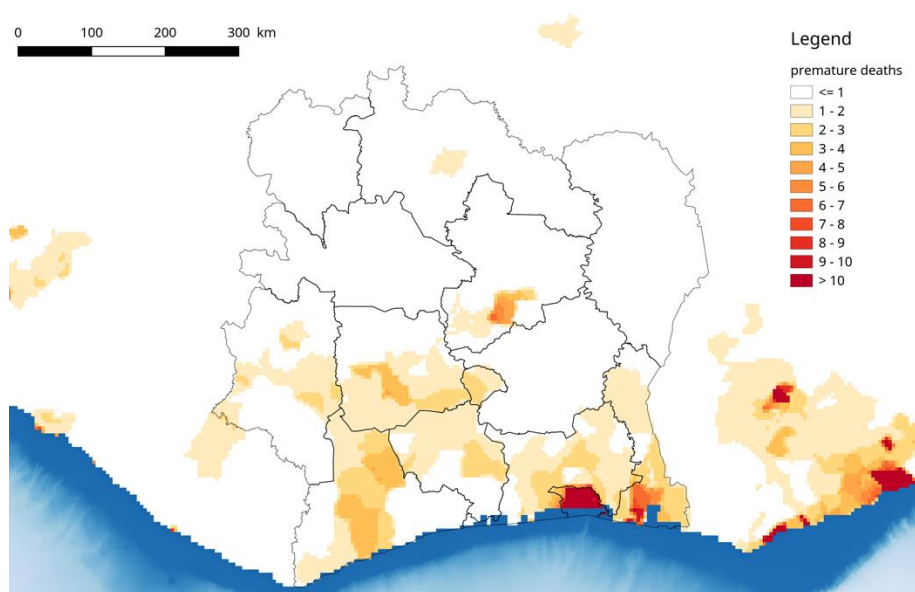


Figure 8: Regional distribution of all causes premature deaths in Cote d'Ivoire in January 2015 due to anthropogenic PM2.5.

Table 5: Premature deaths in Abidjan district in January 2015 due to anthropogenic PM2.5.

Premature deaths	All causes	Cardiovascular	Lung cancer	Respiratory diseases
Abidjan District	7315	3070	27	402

3 Conclusion

The modelling chain developed in DACCIWA WP 2 framework associated to DACCIWA experimental results has allowed us to estimate inflammatory risk and mortality maps at the resolution of 15 x15 km over Africa due to anthropogenic sources. As mentioned earlier, simulations with different emission scenarios are on-going to assess relative importance of domestic fire and traffic sources on health outcomes. Such values will be important for further mitigation. Note that pioneer studies have been launched in 2016 and 2017 in Abidjan, on populations leaving close to our sites (taxi drivers, students close to the waste burning site and fish and meat smokers) with personal exposure measurements (Xu et al., 2019, in review) and also with sociological census, in order to quantitatively calculate risk culture indexes to further improve health impact assessments (Becerra et al., 2019, in review).

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