Risk assessment for redevelopment of contaminated land at an old industrial site

D.F. VENDAS (a,1), C.N. COSTA (a, c, 2), M.G. BRITO (a, b, 3)
a – CIGA - Applied Geosciences Innovation Centre, Faculty of Sciences and Technology, New University of Lisbon, Quinta da Torre, 2829-516 Caparica, PORTUGAL.
b – Earth Sciences Department, Faculty of Sciences and Technology, New University of Lisbon, Quinta da Torre, 2829-516 Caparica, PORTUGAL.
c – Civil Engineering Department, Faculty of Sciences and Technology, New University of Lisbon, Quinta da Torre, 2829-516 Caparica, PORTUGAL.
1 – daniel@ciga.fct.unl.pt; 2 – cnc@fct.unl.pt; 3 – mgb@fct.unl.pt

Abstract: - This paper presents an integrated two phased methodology for the evaluation and mapping of potential human health risk areas at contaminated sites. In a first step, a human health risk index is calculated for each sample location based on exposure pathways and land use scenarios. In a second step, human health risk maps are obtained by the estimation of local index values using geostatistical models. Spatial estimation of human health risk allowed, on one hand, the identification of most dangerous areas inside the contaminated site and, on the other hand, the quantification of respective polluted media, subject to further remediation.
The methodology was applied to an old industrial site, located near Lisbon, on the left margin of the Tagus River (Barreiro - Portugal). The priority area, with an extension of about 30 ha, has been scenario of several chemical industries over the last one hundred years. Nowadays, the area is almost deactivated and designated for urban redevelopment.

Key-Words: Ground contamination, risk assessment, remediation, geostatistics, indicator kriging, heavy metals

1 Introduction

Human Health Risk Assessment is a methodology used to describe and estimate adverse effects on human health due to exposure to certain chemical substances, for a certain period of time [1].
Commonly, contaminated sites are classified based on an index risk value calculated for the most polluted collected sample. This approach gives rise to the overestimation of risk areas, especially in cases where sites enclose non contaminated sub-areas. To avoid this situation and obtain spatial mapping of human health risk grade, geostatistical modelling is used as a tool for estimating distribution of risk indexes based on values calculated for the whole set of sampled locals. Spatial zoning of site risk index contributes for optimization of future remediation actions and, additionally helps planning extra site investigation works, when necessary.

2 Methodology

The methodology consists on the integration of geostatistical models with Human Health Risk Assessment procedures [1] to estimate the spatial distribution of human health risk grade of a site, based on local contamination. Considering that the generalization of the risk calculated based only on the highest value of a sample cannot identify its spatial variability, in this study it was decided to adopt a specific methodology for risk assessment, composed of two sequential stages described as following:

(i) Stage 1 – Risk Assessment - Calculates, on each sample location, the carcinogenic effects (cancer risk) on human health and non-carcinogenic effects (hazard quotient) of chemical pollutants taking into account different exposure pathways and scenarios;
(ii) Stage 2 – Risk Mapping - Spatial mapping of risk areas using indicator kriging geostatistical techniques.

2.1 Stage 1 - Risk assessment

Developed by the United States Environmental Protection Agency, the first stage of risk assessment approach consists on the following sequential procedures:

(i) data compilation and evaluation;
(ii) exposure assessment;
(iii) toxicity assessment;
(iv) risk characterization and;
(v) risk monitoring [1].

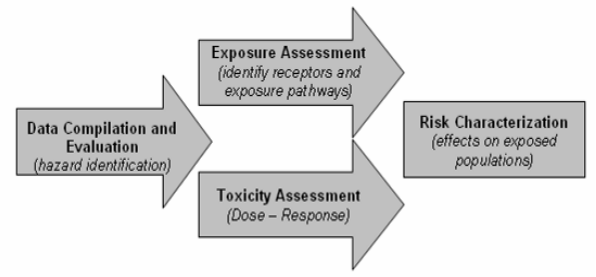

Fig.1 – Human health risk assessment steps [1]

Data compilation and evaluation – starts in the preliminary evaluation and continues in the following evaluations and consists on the acquisition of relevant site data for the human health risk evaluation. The identification and concentration of contaminants, the spatial distributions are important in the selection of contaminants for the risk evaluation process [1].

Exposure assessment (*I*) – consists in the estimation of the type, frequency, duration, magnitude and the routes of exposure *n* to the chemical substance of concern *i*. This evaluation is based in the following steps:

1. Characterization of exposure setting, with respect to the general physical characteristics of the site and the characteristics of the populations on and near the site. Important site characteristics include: temperature, precipitation, wind speed and direction, geologic setting, vegetation, soil type, groundwater hydrology;

2. Identification of exposure pathways by which the previously identified populations may be exposed. Each exposure pathway describes a unique mechanism by which a population may be exposed to the chemicals at or originating from the site.

3. Quantification of exposure, magnitude, frequency and duration of exposure for each pathway identified. This step is most often conducted in two stages: estimation of exposure concentrations and calculation of intakes (equation 1) [1].

$$I_{ni} = C_i * \frac{CR_n * EF * ED}{BW} * \frac{1}{AT} \quad \text{(Equation 1)}$$

Where:
I_n – *Contaminant (i) intake for the exposure n (mg/kg body weight/day)*
C_i – *Contaminant (i) concentration (mg)*
CR_n – *Contact rate for the exposure pathway n (mg/kg)*
EF – Exposure Frequency (days/year)
ED – Exposure Duration (years)
BW – Body Weight (kg)
AT – Average Time (days)

Toxicity assessment – gathering toxicity information for non-carcinogenic effects (target organs or critical effects) and carcinogenic effects (carcinogen class). Consists in identifying important toxicity values such as:

(i) *chronic oral reference doses* (*RfDs*) for non-carcinogenic effects;
(ii) *oral cancer slope factors* (*SF*) for carcinogenic effects [1].

Toxicity evaluation means specific toxicity of a chemical element considering its adverse effects on human health associated with exposure to the element. To evaluate specific toxicity it is necessary to evaluate the relation between the magnitude of exposure, the type of adverse effect and the probability of a compound to produced cancer in individuals over the exhibition. For this purpose toxicological databases are the basis for information on toxicology and respective adverse effects to health.
This step can be divided into two main steps:

- identification of the adverse effects - type and magnitude of the adverse effect on health that is caused by exposure to a specific toxic agent;

- determination of the dose-response - procedure for quantitative evaluation of the toxicity, relating to the dose of the contaminant that was received with the incidence of adverse effects to health in a given population exposed.

Risk characterization - summarizes and combines outputs of the exposure and toxicity assessments into a quantitative and qualitative expression of risk**.**
The *carcinogenic risk* is the probability of an individual to develop cancer over a lifetime, and is estimated from calculated intakes (I_{ni}) and chemical-specific *Slope Factor* (*SF*) (equation 2) [1].

$$CR = I_{ni} * SF \quad \text{(Equation 2)}$$

Where:
CR – Carcinogenic Risk
SF - Slope Factor

The *Hazard Quotient* (*non-carcinogenic risk*) is the probability of an individual to develop a non cancer disease over a lifetime and is estimated from calculated *Intakes* (I_{ni}) and the *Reference-Dose* (*RfD*) (equation 3) [1].

$$HQ = \frac{I_{ni}}{RfD} \qquad \text{(Equation 3)}$$

Where:
HQ – Hazard Quotient (non-carcinogenic risk)
RfD – Reference Dose

2.2 Stage 2 – Risk mapping

After the estimation of *carcinogenic risk* (*CR*) and *hazard quotient* (*HQ*) values for each local sample, it is necessary to estimate the morphology and extension of the risk areas to identify the priority areas to remediate.
The most appropriate geostatistical method for estimating the morphology of a certain phenomena in space is the indicator kriging [2]. This method transforms the original variable in a new binary variable – indicator variable, represented by *zero* (0) or *one* (1), respectively, if values are below or above a certain threshold (*z*), as indicated in equation 4 [2].

$$I(x_i) = \begin{cases} 1 \text{ if } x >= z_i \in X \\ \qquad\qquad \text{and } i = 1,...,N \\ 0 \text{ if } x < z_i \in X^C \end{cases} \qquad \text{(Equation 4)}$$

$I(x_i)$ – indicator variable at point x_i;
i, ... N - number of samples
z – threshold value

Hence, risk values are transformed into indicator variables *(I(x))* and probability risk maps are obtained by kriging the indicator variables, considering each scenario and exposure pathway.
The structure and dimension of the risk areas could be measured by the indicator variogram $\gamma_I(h)$, defined as (equation 5):

$$\gamma_I(h) = \frac{1}{2N(h)} \sum_{i=1}^{N(h)} \left[I(x_i) - I(x_i + h) \right]^2 \qquad \text{(Equation 5)}$$

h – distance between samples
$N(h)$ – number of data pairs within the class of distance h
i – sample location; 1…. N
x_i – value of sample at location i
γ_I (h) – indicator variogram value for lag distance h

Indicator variogram is a measure of how often two *z* values, separated by a vector *h*, are on opposite sides of a threshold value.
The probability of each point x_0 ($x_0 \in X_{n.m}$, regular grid) to be below or above a cut-off is estimated as a linear combination of the neighbouring samples of x_0 $I_z(x_\alpha)$, α = 1 to *N*, as (equation 6):

$$\left[I_z(x_0) \right]^* = \sum_{\alpha=1}^{N} \lambda_\alpha(z) I_z(x_\alpha) \qquad \text{(Equation 6)}$$

x_0 - location of the point to estimate;
λ_α - weight of each sample $\alpha = 1, \ldots, N$.
$I_z(x_\alpha)$ – probability of x_0 belong to X

For the characterization of priority risk areas the indicator kriging [2] geostatistical approach is used considering the following cut-off for risk values [1]:

- If $CR \geqq 1E^{-4}$; carcinogenic risk is assumed for industrial/commercial use (probability of 1 individual in 10 thousand, to have cancer);
- If CR 1E-6; carcinogenic risk is assumed for residential use (probability of 1 individual in 1 million, to have cancer);
- If $HQ \geqq 1$; non-carcinogenic risk is assumed.

3 The Case Study

The study area is an old industrial site located near Lisbon, on the left margin of Tagus River (Barreiro - Portugal). The priority area, with an extension of about 30 ha, has been scenario of several chemical industries over the last one hundred years. Nowadays, the area is almost deactivated and designated for urban redevelopment [3].
Selection of relevant data was based on soil samples where chemical content exceed the reference values for residential use, in accordance with Ontario guidelines [4]. In conformity, the selected contaminants are: arsenic (As), copper (Cu) and lead (Pb).
Figure 2 presents a map locating the 59 pit/boreholes used as data source for the risk evaluation.

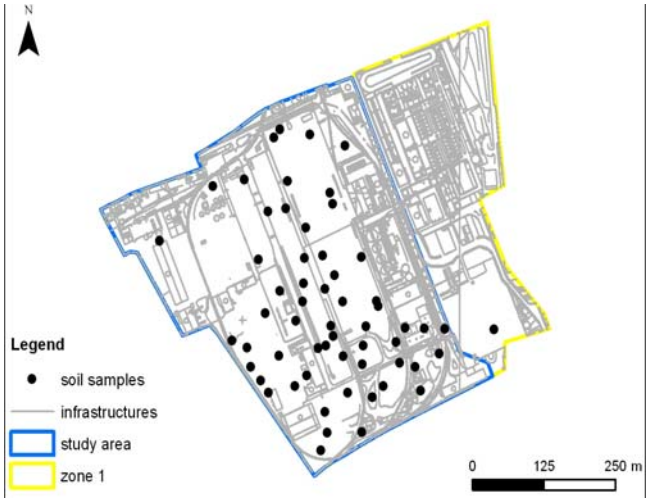

Fig.2 – Location of the 59 soil data samples

The evaluation was developed considering different scenarios for present and future use of the area: residential child, residential adult, industrial or commercial worker and construction worker.
The contact between the receptor and the contaminant may occur through the following exposure pathways (figure 3): ingestion of soil, dermal contact with soil and ingestion of vegetables.

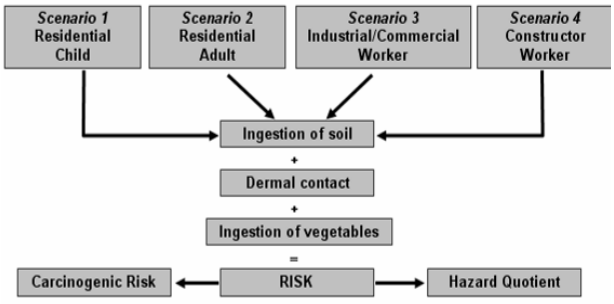

Fig.3 – Land use scenarios for risk assessment

Stage 1 – Risk assessment

To calculate the risk values the *RISCWorkbench* software was used [5] based on the Manual "Risk Assessment Guidance for Superfund (RAGS)" of the U. S. Environmental Protection Agency [1], and the GIS software ArcGIS from ESRI [6].
Risk values - carcinogenic risk (*CR*) and hazard index (*HQ*), were calculated for each sample location, using equations 1, 2 and 3.
For the evaluation of *exposure* there were used the parameters presented on table 1 (for *soil ingestion* and *dermal contact* exposure pathways) and table 2 (for *ingestion of vegetable*s exposure pathway). The values used to calculate toxicity are presented on table 3.

Table 1 – Exposure parameters for *soil ingestion* and *dermal contact* pathways [1]

Parameters	Residential		Commercial	Constructor
	Child	Adult	Worker	Worker
Lifetime (years)	70	70	70	70
Body weight (kg)	15	70	70	70
Exposure frequency (days/year)	130	40	125	250
Exposure duration (years)	6	9	8	1
Soil ingestion rate (mg/day)	90	40	40	100
Total skin surface (cm^2)	6.800	18.400	18.400	23.000
Fraction skin surface exposed to soil	0.13	0.11	0.11	0.57
Soil to skin adherence factor (mg/cm^2)	0.2	0.2	0.2	0.3

Table 2 – Exposure parameters for *ingestion of vegetables* pathway [1]

Parameters	Residential		Commercial	Constructor
	Child	Adult	Worker	Worker
Lifetime (years)	70	70	70	70
Body weight (kg)	15	70	70	70
Exposure frequency (days/year)	130	40	125	250
Exposure duration (years)	6	9	8	1
Exposure frequency for ingestion of vegetables (days/year)	350	350	0	0
Exposure duration for ingestion of vegetables (years)	6	9	0	0
Veg.ingestion rate (underground) (g/year)	48.5	87.5	0	0
Veg.ingestion rate (aerial)(g/day)	55.8	127	0	0

Table 3 – Toxicity parameters [1]

Chemicals	As	Cu	Pb
CAS	7440382	7440508	7439921
Molecular weight (g/mole)	74.9	63.5	0.0
EPA classification	A	D	B2
Slope factor (SF) - Oral	$1.5E^{+0}0$	-	-
Slope factor (SF) - Inhalation	$1.5E^{+01}$	-	-
Reference-dose (RfD) - Oral	$3.0E^{-04}$	$4.0E^{-02}$	$3.6E^{-03}$
Absorption – (Oral – Soil)	1	1	1
Absorption (Dermal – Soil)	0.03	0.01	0.01
Skin permeability coefficient	$1.0E^{-03}$	$1.0E^{-03}$	$0.0E^{+00}$

Stage 2 – Risk mapping

For estimating the morphology of the risk areas it was used the indicator kriging geostatistical method:

1 - Risk indexes (*CR* and *HQ*) were transformed into an indicator variable by using equation 4, as following:

- If $CR \geqq 1E^{-6}$; then $CR_{Indicator} = 1$; otherwise 0
- If $HQ \geqq 1$; then $HQ_{Indicator} = 1$; otherwise 0

Example of carcinogenic risk indexes (*CR*) transformed on indicator variables are shown from figures 4 to 10.

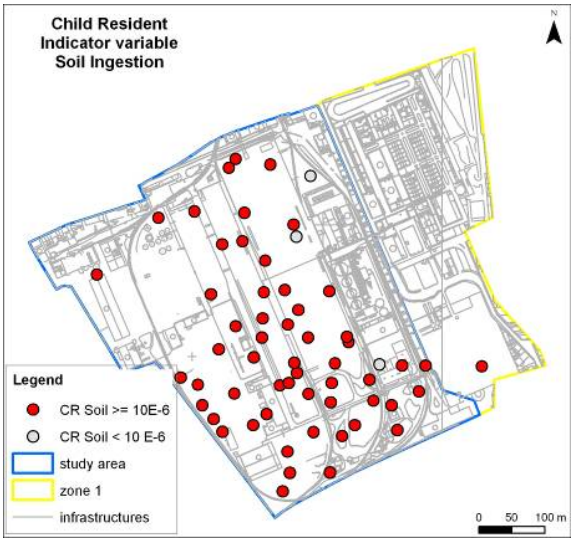

Fig. 4 – Scenario 1- Indicator variable for *Soil ingestion*(in [3])

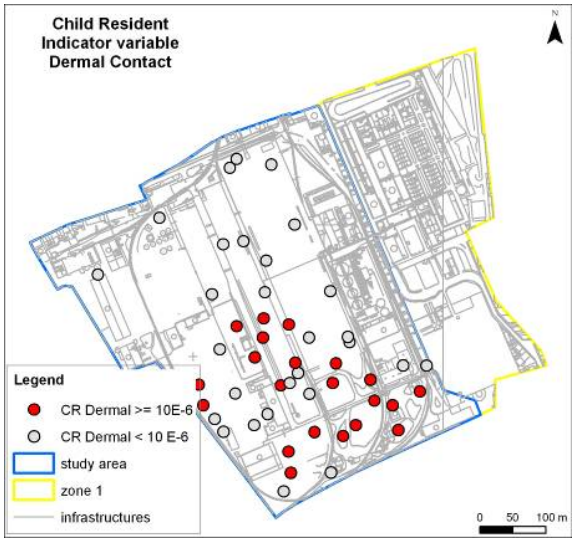

Fig. 5 – Scenario 1 - Indicator variable for *Dermal contact* (in [3])

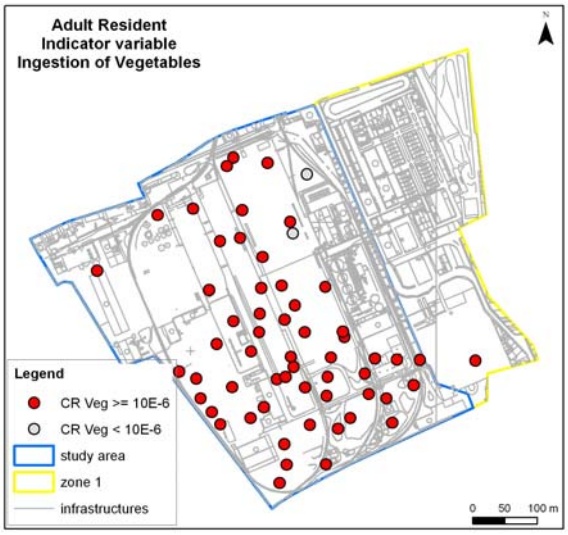

Fig. 6 – Scenario 1 - Indicator variable for *Ingestion of Vegetables* (in [3])

Total risk is obtained by the sum of individual risk values.

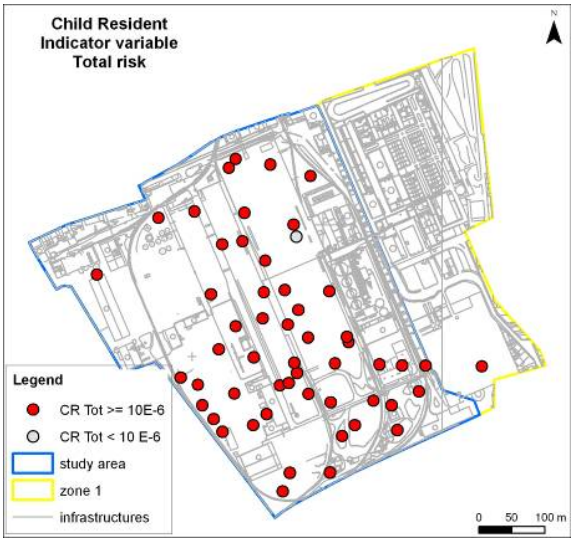

Fig. 7 – Scenario 1 - Indicator variable for *Total risk* (in [3])

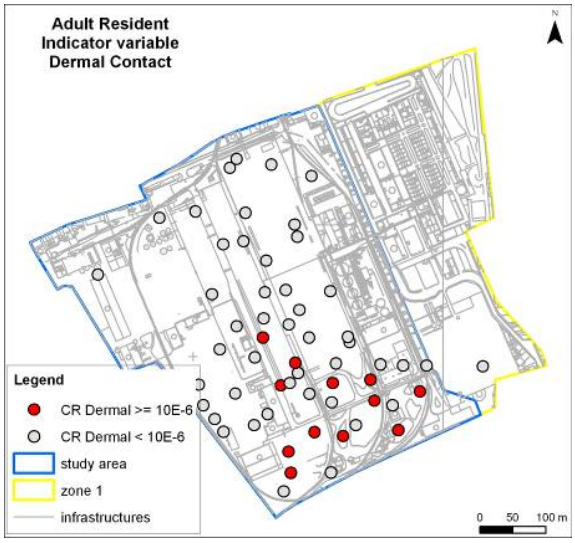

Fig. 9 – Scenario 2- Indicator variable for *Dermal contact* (in [3])

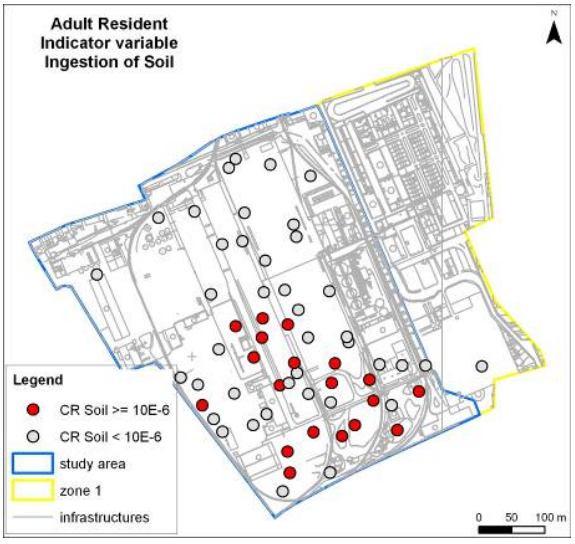

Fig. 8 – Scenario 2- Indicator variable for *Soil ingestion*(in [3])

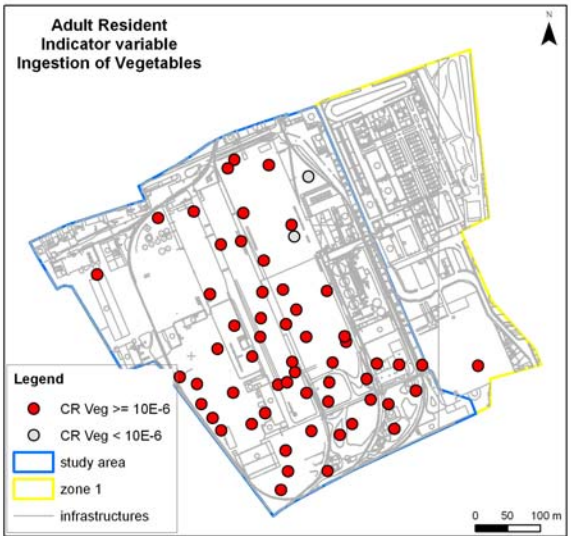

Fig. 10 – Scenario 2 - Indicator variable for *Ingestion of Vegetables* (in [3])

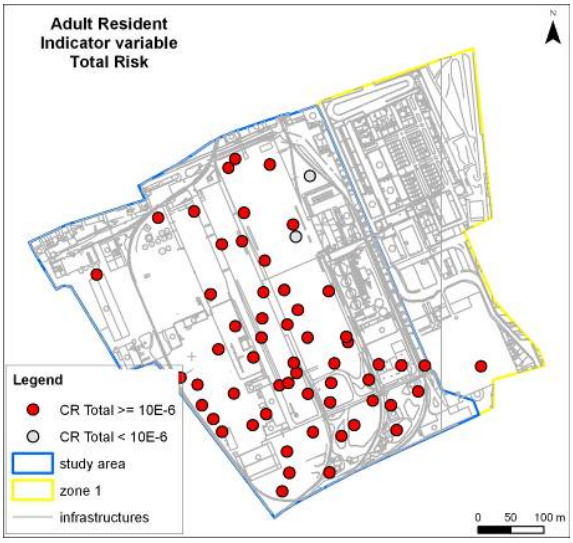

Fig. 11 – Scenario 2 - Indicator variable for *Total risk* (in [3])

2 - Risk maps are estimated by calculating, for each scenario, the experimental indicator variograms, for each scenario and exposure route, using equation 5. Probability maps are obtained by equation 6 and morphological maps by transforming the probability maps into indicator maps [2]. This geostatistical step was performed using software *Geoms* [7].

Estimated risk maps for scenario 1 (child-residential) are presented on figures 12, 13 and 14 for (i) ingestion of soil; (ii) dermal contact and; (iii) ingestion of vegetables, exposure pathways, respectively.

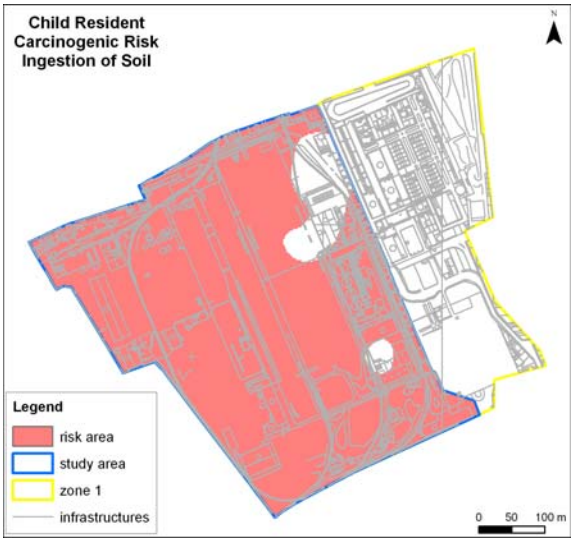

Fig. 12 – Scenario 1- *Soil ingestion*(in [3])

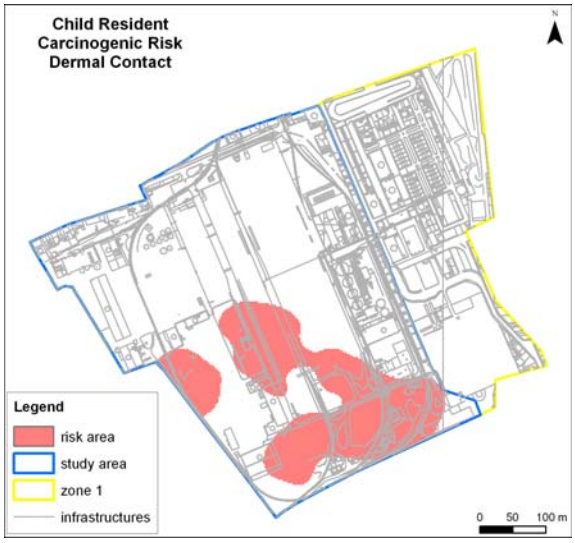

Fig. 13 – Scenario 1 - *Dermal contact* (in [3])

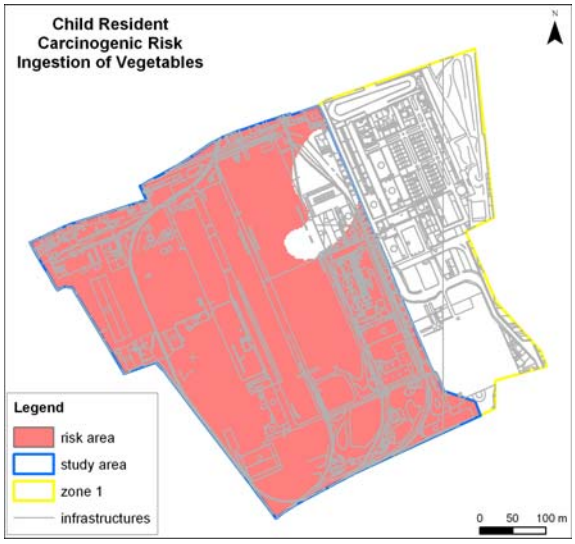

Fig. 14 – Scenario 1 - *Ingestion vegetables* (in [3])

Total risk is obtained by the sum of individual risk maps (figure 15).

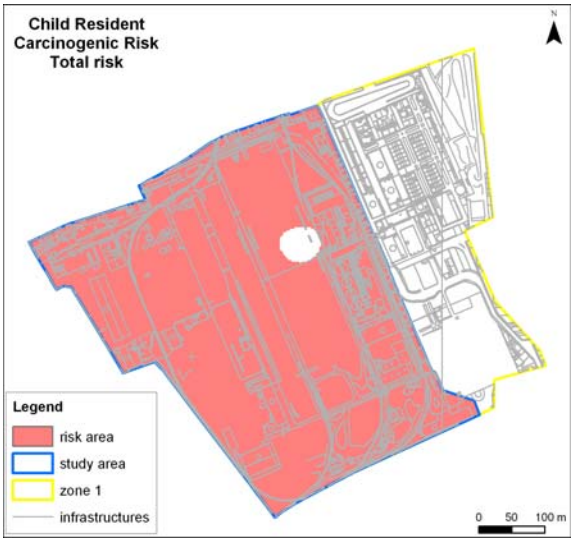

Fig. 15 – Scenario 1 - *Total risk* map (in [3])

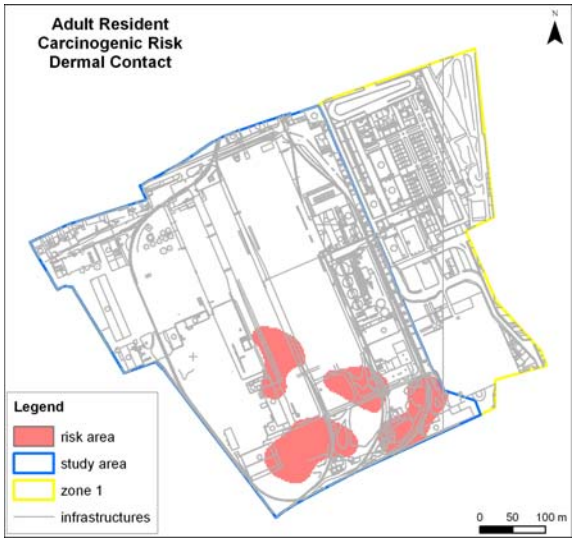

Fig. 17 – Scenario 2 - *Dermal contact* (in [3])

Figures 16, 17 and 18 present the estimated risk maps for scenario 2 (adult-residential), considering the same exposure pathways as for scenario 1: (i) ingestion of soil; (ii) dermal contact and; (iii) ingestion of vegetables, respectively.

The estimation of risk maps allowed not only the quantification of risk areas but also the weighting of exposure pathways.

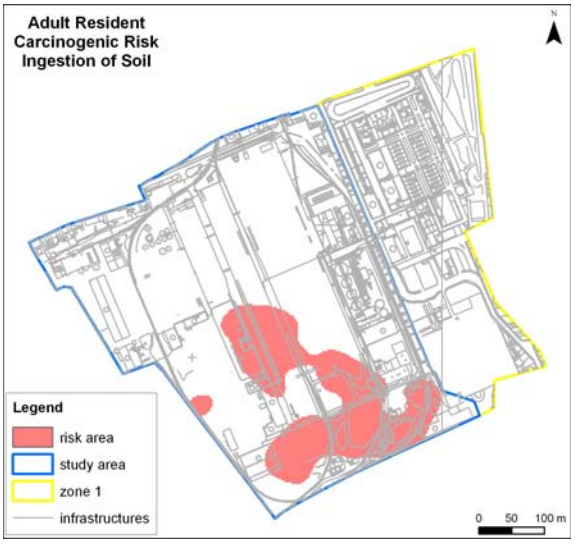

Fig. 16 – Scenario 2- *Soil ingestion*(in [3])

Fig. 18 – Scenario 2 – *Ingestion vegetables* (in [3])

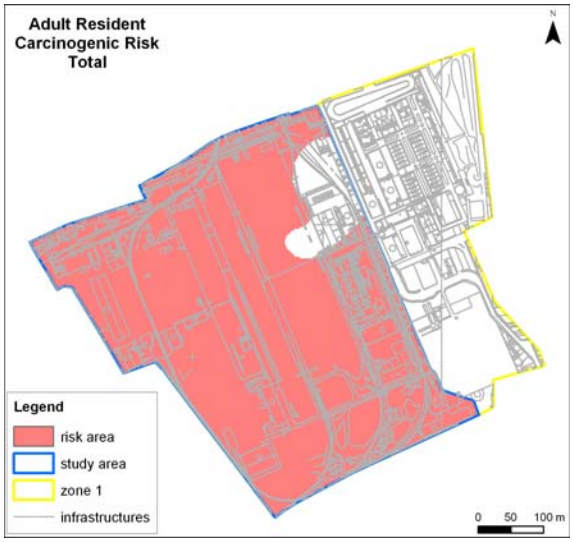

Fig. 19 – Scenario 2 - *Total risk* map (in [3])

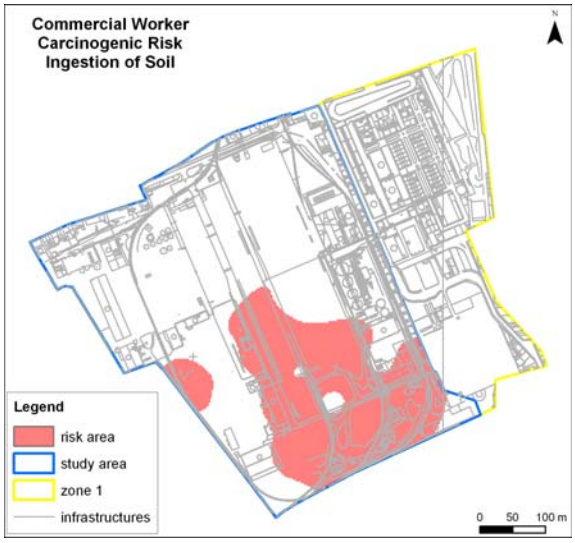

Fig. 20 – Scenario 3- *Soil ingestion*(in [3])

Concerning residential receptor

- the carcinogenic risk for the ingestion of vegetables is much higher than the risk caused by the ingestion of soil (figures 16 and 18);
- The risk for *dermal contact* (figure 17) is reduced, compared with the other exposure pathways.

The study concluded that, in the hypothesis of eliminating the risk for *ingestion of vegetables* (through a mere restrictive measure of use), the *carcinogenic risk* of the study area is reduced to about 2/3 of the total risk in the case of a child and to about 5% in the case of the adult.
In terms of total carcinogenic risk (sum of the 3 exposure pathways), it can be concluded that almost the entire area should have some kind of intervention in terms of soil remediation or use restriction for residential use (figure 19).
Figures 20 to 25 illustrate the risk maps obtained for scenarios 3 and 4. For these scenarios the exposure pathway for *ingestion of vegetables* is not considered.

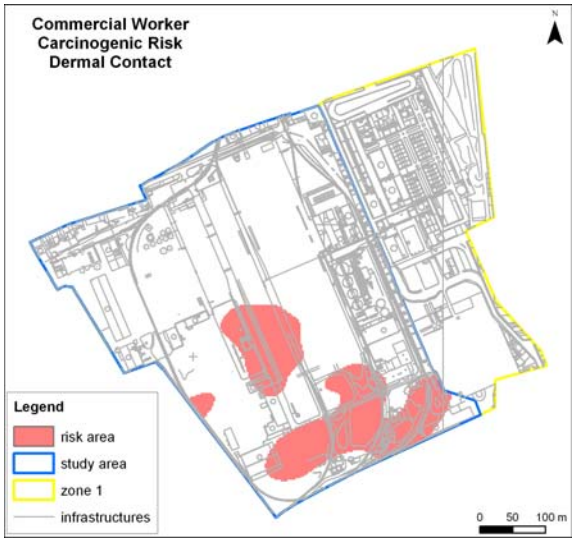

Fig. 21 – Scenario 3 - *Dermal contact* (in [3])

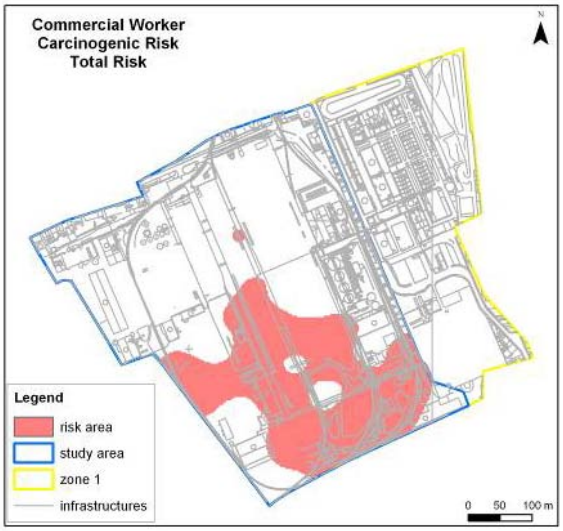

Fig. 22 – Scenario 3 – *Total risk map* (in [3])

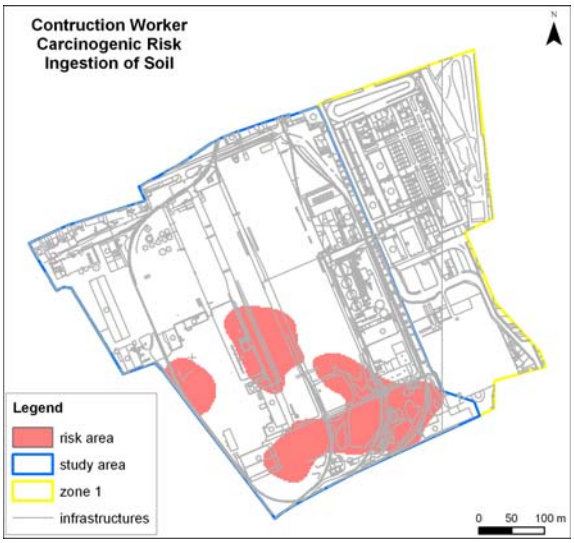

Fig. 23 – Scenario 4 - *Soil ingestion*(in [3])

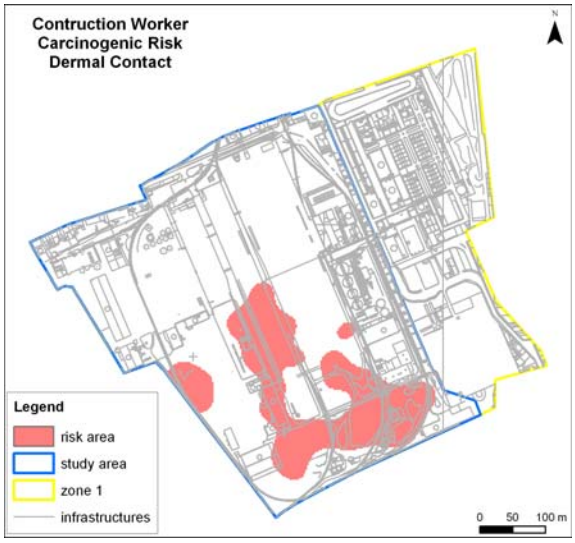

Fig. 24 – Scenario 4 - *Dermal contact* (in [3])

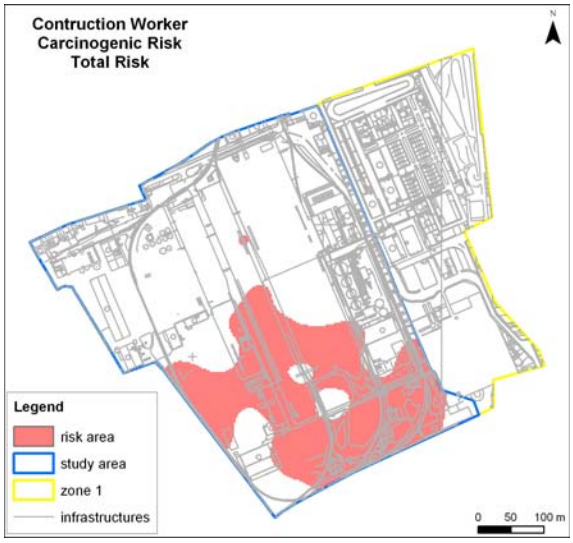

Fig. 25 – Scenario 4 – *Total risk map* (in [3])

The *construction worker* has a similar risk for both ways of exposure as a consequence of direct manipulation of materials (figures 10 and 11).
Concerning scenarios 3 and 4 (*commercial worker* and *construction worker*), the results show that they are subject to a total *carcinogenic risk* in approximately 1/3 of the area. The *commercial worker* has a risk of *ingestion of soil* higher than the risk of *dermal contact* (figures 20 and 21).

Concerning the non carcinogenic risk the results are not presented here but it can be said that the contribution of the factors of exposure are very similar to those of the total carcinogenic risk.

4 Conclusions

The integration of geostatistical techniques as a second stage for site risk assessment allowed the characterization and quantification of the priority areas to be remediated and, consequently, minimized the respective environmental costs.
The methodology contributes to the planning of human occupation and identifies potential restrictive actions to implement in order to minimize the identified risks.

This methodology allows the comparison of risk areas resulting from the different exposure pathways related to human occupation. In the particular case of this study, it was possible to reduce the total risk area to about 2/3 by the application of restrictive landuse rules (restriction of agriculture practices) to the site.

5 Acknowledgments

The authors are thankful to Quimiparque – Parques Empresariais, S.A

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