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Cattle Consumption Model for the Wonderfonteinspruit Related to Human Health Risk

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Abstract: Polluted cattle feed not only impacts on animal health but also on the health of humans. The aim of this study is therefore, to develop a cattle consumption model based on a control and experimental group. Animal tissue analysed for both groups were samples from cattle, 6 years of age. The experimental group had been exposed to elevated levels of pollutants emanating from the gold mining industry in South Africa. The cattle consumption model takes into account the intake of soil, grass and water by the animal. A human health risk assessment (toxic and carcinogenic) was performed based on the results of the cattle consumption model. It was shown that no toxic risk exits for both the control and experimental groups if an intake rate of 0.13 kg of meat per day is assumed. For the same consumption rate, only muscle consumption in the control group yielded an acceptable carcinogenic risk of the occurrence of one person getting cancer out of a million.

Key words: Wonderfonteinspruit, cattle consumption model, health risk assessment, cancer, South Africa

INTRODUCTION

The quality of cattle feed not only impacts on animal health but more importantly, also potentially on human health (Van der Fels-Kle *et al*., 2011). It is therefore, crucial to analyse the elemental concentrations found within cattle feed in areas exposed to pollutants and in turn relate the results to the impacts on human health. It is expensive and impractical to have analyses on each animal when they are slaughtered for the consumer market.

The aim of this study was to develop a cattle consumption model based on a control and experimental group. The experimental group is exposed to elevated levels of pollutants emanating from the gold mining industry in South Africa. The term cattle consumption model takes into account intake of soil, grass and water by the animal. In turn the predictive cattle consumption model is used to perform a human health risk assessment based on the Environmental Protection Agency (EPA) methodology.

Background: The Wonderfonteinspruit (WFS) takes its origin at the surface water divide immediately to the South of Krugersdorp in the Gauteng Province, South Africa and flows into the Mooi River, close to Potchefstroom in the Northwest Province (Coetzee, 2004). It forms part off the Mooi River (Kromdraai) catchment which constitutes an important component of the Vaal River System. The name wonderfonteinspruit means "wonderful-fountain-stream", a name it derived from the large volumes of dolomitic groundwater that once fed the stream via karst springs. It drains a catchment area of approximately 1600 km^2 and flows for approximately 90 km through an area known to have the richest gold deposits in the world. The WFS has been divided into the upper and lower WFS areas (Winde, 2010). A map of the catchment can be seen in Fig. 1.

The upper WFS originates at Tudor Dam, South of Krugersdorp and ends in Donaldson Dam near Westonaria. The West Rand goldfield, has produced >1900 ton of gold and was first mined in 1887, only a year after the discovery of gold in the Witwatersrand (Coetzee, 2004; McCarthy, 2006). Most of the mines in the upper WFS have been closed or abandoned and the area is covered with unrehabilitated tailings dams, rock dumps and sand dumps (Coetzee, 2004).

The lower WFS starts below Donaldson Dam, at the beginning of the 1 m pipeline which was constructed in 1977 to transport water from various gold mines over three dewatered dolomitic compartments (Oberholzer, Venterspos and Bank). The pipeline was constructed to prevent recirculation of the water that was pumped to the surface from underground mine workings. The 1 m pipeline stretches for approximately 32 km and

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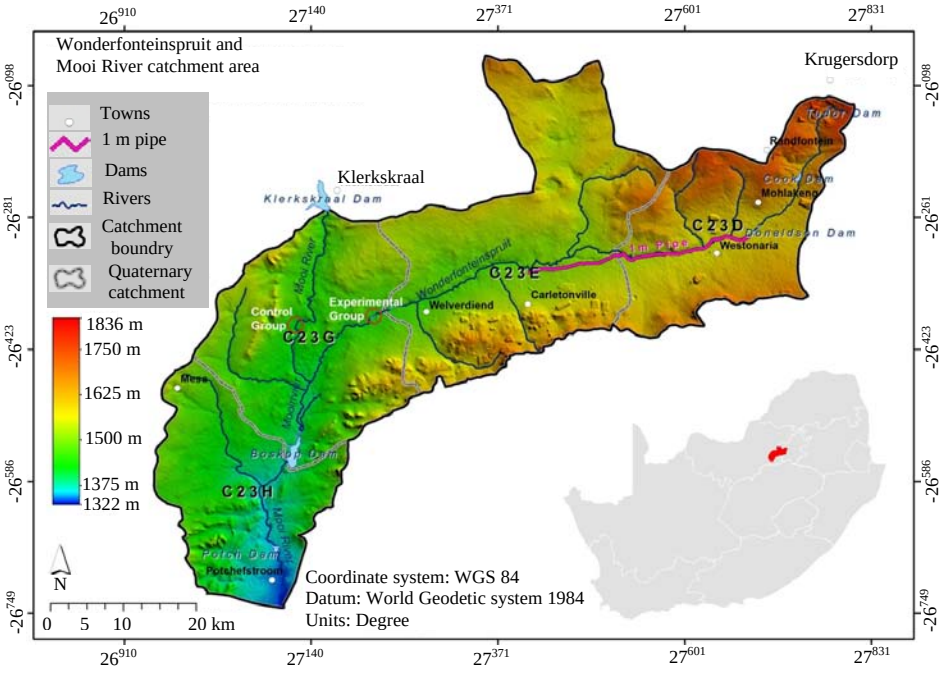

Fig. 1: Map of the wonderfonteinspruit, surrounding areas and the Mooi River catchment

ends immediately to the North of Carletonville. The lower WFS area comes to an end at its confluence with the Mooi River above Boskop Dam (Coetzee, 2004).

The goldfield in this area is often referred to as the West Wits Line and has supported 10 major mines that produced >7300 ton of gold (McCarthy, 2006). Mining only commenced after the dolomitic compartments were dewatered in the 1930's. Several of the gold reefs in this area also contained elevated concentrations of uranium which led to large-scale uranium production in the early 1950's. The mining activities gradually transformed the lower WFS into a barren streambed with several scattered sinkholes and four dried up springs (Swart *et al*., 2003).

Mining which involves the extraction and processing of ores, generally affects relatively small areas. It is the tailings and waste rock deposits close to the mining site which is the source of metal pollution of water resources (Salomons, 1995).

The pollution in the WFS has already reached headlines in local and international media (Winde, 2010). It has also been the subject of numerous studies and reports by the National Nuclear Regulator, Department of Water Affairs and the Council of Geoscience (Coetzee, 2004). Mining and processing of uraniferous gold ores are mainly responsible for radioactive and heavy metal pollution in the WFS (Coetzee, 2004). The main mechanisms for release of metals from mine wastes are through leaching into surface and groundwater, fugitive dust emissions and from tailing solutions (Spitz and Trudinger, 2009).

A control and experimental group of cattle (Fig. 1) was selected in the WFS study area. The control group is situated along the Mooi River which is considered unpolluted and the experimental group is situated along the polluted WFS.

MATERIALS AND METHODS

Measured field data from both the control and experimental groups were fit to a consumption model to predict concentrations in the liver, kidney and muscle of the animal based on the intake of soil, grass and water. The predictive model was then employed to perform a human health risk assessment based on the animal intake and the human consumption for various scenarios. A schematic of the modelling process is shown in Fig. 2.

Available data: Data collected from the control and experimental groups are presented in Table 1 and 2, respectively. Note that multiple samples were analysed for each element and only the average values are presented here. Furthermore, the animal age group were approximately 6 years for both the control and experimental groups and all concentrations expressed in mg/kg represent the dry weight concentrations.

Table 1: Control group data

Variables	Co	Ni	Cu	Zn	Se	Cd	Pb	U
Soil (mg kg^{-1})	39.5500	153.0000	46.0750	28.9750	0.5040	0.1050	25.5250	0.7150
Grass (mg kg^{-1})	0.8733	5.5725	10.2000	32.5250	0.6950	0.0400	0.9000	0.0400
Water (mg L^{-1})	0.0000	0.0019	0.0038	0.0028	0.0069	0.0000	0.0005	0.0008
Liver (mg kg^{-1})	0.3321	0.3018	159.3400	104.2900	0.6759	0.1636	0.1064	0.0425
Kidney (mg kg^{-1})	0.1073	0.0017	18.1165	78.0500	6.5710	1.1820	0.0261	0.0039
Muscle (mg kg^{-1})	0.0062	0.2044	3.3095	154.7400	0.3895	0.0174	0.1515	0.0485

Table 2: Experimental group data

Variables	Co	Ni	Cu	Zn	Se	Cd	Pb	U
Soil (mg kg^{-1})	39.5500	153.0000	46.0750	28.9750	0.5040	0.1050	25.5250	0.7150
Grass (mg kg^{-1})	0.8733	5.5725	10.2000	32.5250	0.6950	0.0400	0.9000	0.0400
Water (mg L^{-1})	0.0000	0.0019	0.0038	0.0028	0.0069	0.0000	0.0005	0.0008
Liver (mg kg^{-1})	0.3321	0.3018	159.3400	104.2900	0.6759	0.1636	0.1064	0.0425
Kidney (mg kg^{-1})	0.1073	0.0017	18.1165	78.0500	6.5710	1.1820	0.0261	0.0039
Muscle (mg kg^{-1})	0.0062	0.2044	3.3095	154.7400	0.3895	0.0174	0.1515	0.0485

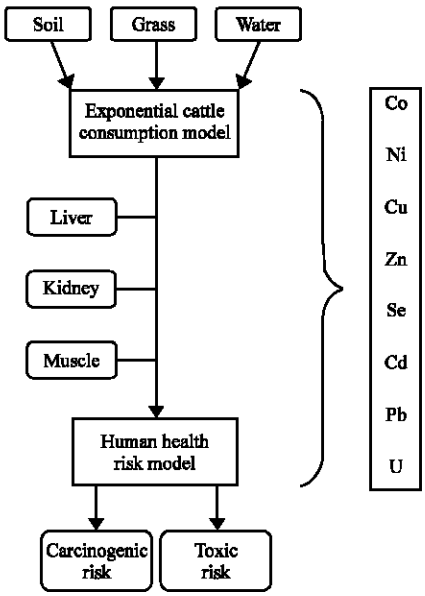

Fig. 2: Schematic of the modelling process

Cattle consumption model: Two general approaches to modelling accumulation in animal organs are found in literature namely the linear accumulation and the steady state model using an exponential relationship (Van der Fels-Kle *et al*., 2011). The linear model assumes an irreversible accumulation with no excretion path and usually represents worst case scenarios. The formulation of the linear model is given in Eq. 1 (Van der Fels-Kle *et al*., 2011):

$$C_t = BTR \times DI \times t \quad (1)$$

Where:
C_t = Concentration at time t (mg kg^{-1})
BTR = Biotransfer Rate
DI = Daily Intake (mg day^{-1})
t = Time (days)

The exponential model assumes a steady state scenario where accumulation takes place but an excretion path is also present. The formulation for the exponential model is given in Eq. 2-3 (Van der Fels-Kle *et al*., 2011):

$$C_t = C_0 e^{-\lambda t} + C_{ss}\left(1 - e^{-\lambda t}\right) \text{with } \lambda = \frac{\ln(2)}{T_{1/2}} \quad (2)$$

$$C_{ss} = BTF \times DI \times F_{abs} \text{ with } BTF = \frac{COR}{W\lambda} \quad (3)$$

Where:
C_t = Concentration at time t (mg kg^{-1})
C_0 = Initial concentration (mg kg^{-1})
λ = Elimination time constant
$T_{1/2}$ = Half-life time (days)
C_{ss} = Steady state concentration (mg kg^{-1})
BTF = Biotransformation Factor (day kg^{-1})
DI = Daily Intake (mg day^{-1})
F_{abs} = Absorption in alimentary canal (%)
COR = Carry Over Rate
W = Weight of organ (kg)
t = Time (days)

The predictive cattle consumption model not only includes organs but also muscle, bone and hair, even though the health risk assessment will only be conducted on liver, kidneys and muscle as the main sources of human ingestion. Making use of the similarity between Eq. 1 and 3 and applying an exponential response to the model in analogy to Eq. 2 the following general equation is formulated:

$$C_t = C_0 e^{-\lambda t} + (BTR \times DI)\left(1 - e^{-\lambda t}\right) \text{ with } \lambda \frac{\ln(2)}{T_{1/2}} \quad (4)$$

Where:
C_t = Concentration at time t (mg kg^{-1})
C_0 = Initial concentration (mg kg^{-1})

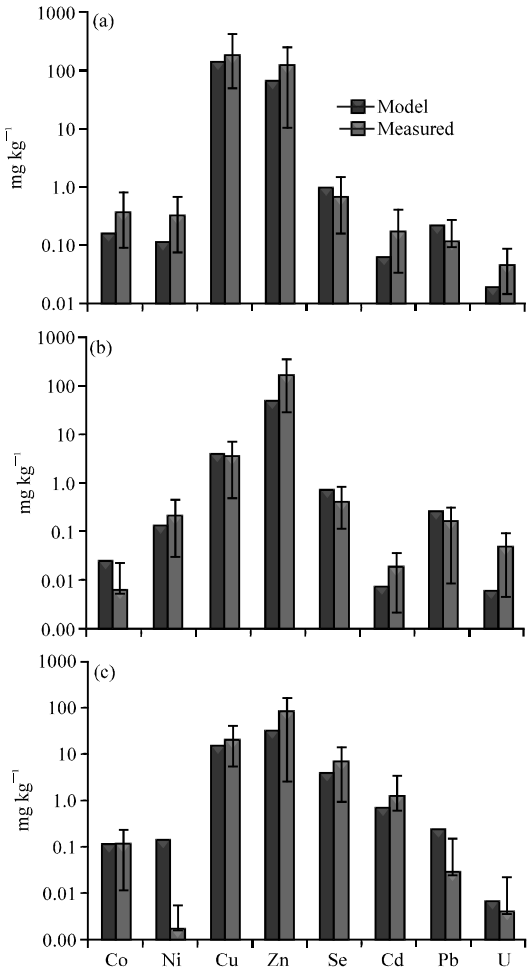

Fig. 3: Control group-measured vs. modeled values: a) Liver; b) Muscle; c) Kidney

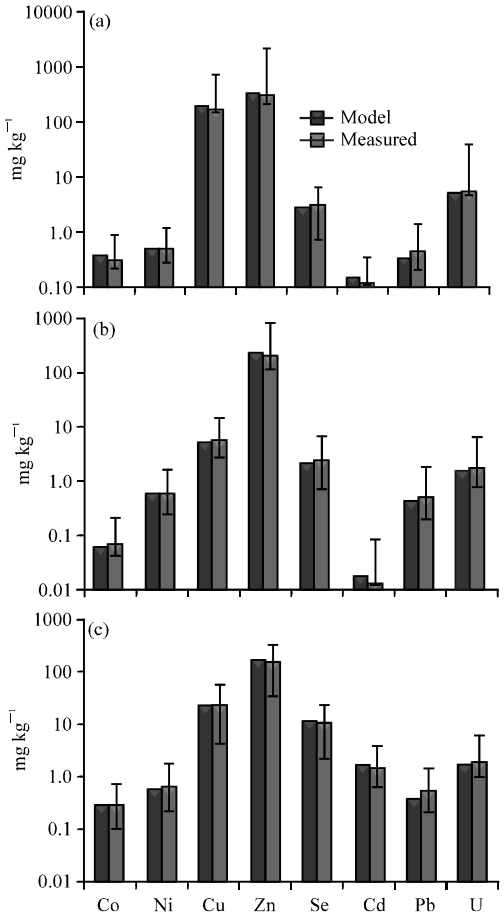

Fig. 4: Experimental group-measured vs. modeled values: a) Liver; b) Muscle; c) Kidney

λ = Elimination time constant
$T_{1/2}$ = Half-life time (days)
BTR = Biotransformation factor (day kg^{-1})
DI = Daily Intake (mg day^{-1})
t = Time (days)

Model calibration: The initial concentration at day zero of the animals tested was not available for this study and was assumed zero for the purposes of this study. An initial concentration is however, expected in a new-born calf whose mother has been subjected to polluted feed and water. Due to the fact that two groups of data exist (control and experimental) requires the simultaneous solution of two equations in two unknowns (BTR and λ) for each component of interest. The equation set for a single element is shown in Eq. 5:

$$C_{t_{con}} = BTR \times DI_{con} \times \left(1 - e^{-\lambda t}\right)$$
$$C_{t_{exp}} = BTR \times DI_{exp} \times \left(1 - e^{-\lambda t}\right) \quad (5)$$

The solution of the equation set in Eq. 5 was achieved using MATLAB® with an initial BTR guess as the average BTR obtained using the linear model in Eq. 1 for each element of interest.

The observed versus simulated results for the control group and experimental group is shown in Fig. 3 and 4, respectively. From the results it appears that the predictive model estimates the experimental group better than the control group. Note that concentrations are presented on a log scale in Fig. 3 and 4 and the error bars indicate the minimum and maximum of the samples analysed with respect to the average value measured. The correlation between observed and simulated values when the control and experimental groups are combined is shown in Fig. 5. The three sources used in the health risk assessment with their associated correlations are as follows:

- Liver (98%)
- Muscle (93%)
- Kidney (87%)

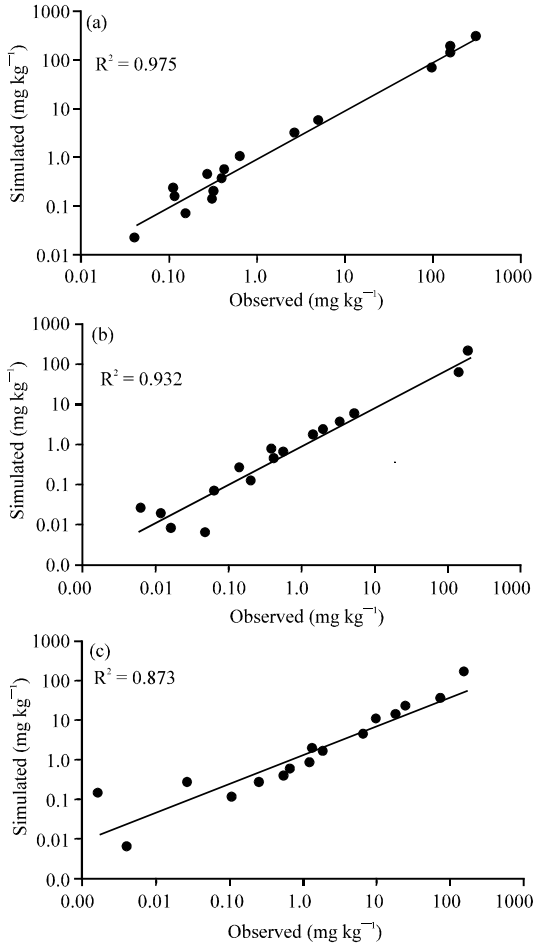

Fig. 5: Correlation between observed and simulated values for both control and experimental groups: a) Liver; b) Muscle; c) Kidney

Table 3: Model predictions outside the minimum-maximum range measured

Variables	Co	Ni	Cu	Zn	Se	Cd	Pb	U
Liver								
Kidney		x					x	
Muscle	x							

All model predictions for the control group were observed to be between the measured minimum and maximum values with the exception of those listed in Table 3. All model predictions for the experimental group grouped between the measured minimum and maximum values.

Non-unique solution: Of particular importance is to note that the solution of two equations in two unknowns are non-unique, hence the initial guesses to ensure a solution in the correct solution space (Grosan and Abraham, 2008). Example responses of kidneys to cadmium are presented in Fig. 6 to illustrate this point. The model can be fit to obtain the same concentration at year 6 for various

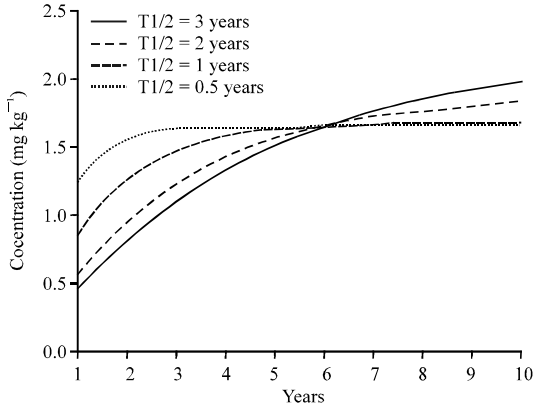

Fig. 6: Example kidney response to Cd

half-life values. The only way to achieve a fit with a high confidence is to consider similar data sets at different times to better describe the curve. Unfortunately, additional datasets were not available for this study to achieve the earlier mentioned.

It has already been shown that sufficient correlation between measured and observed values are obtained from the predictive model, calibrated for data at year 6. This is seen as an upper limit for the risk assessment as 6 years are the general age at which these animals are slaughtered.

Model fitness: The influence of the intake of water was proven to be negligible based on measurements of cadmium in drinking water (Van der Fels-Kle *et al*., 2011). The predictive model fitness was tested by setting the intake of water and soil to zero, leaving grass as the only source in the cattle consumption model. The model fitness under these conditions, expressed as the correlation between observed and simulated values are presented in Fig. 7.

It is clear from the data presented in Fig. 7 that only small changes in model correlation have occurred and only liver and spleen has shown a decrease in correlation whereas all the other components actually showed an increased correlation. This confirms that the predictive model can be used effectively with grass as the only source.

Risk Assessment Model: The EPA (Environmental Protection Agency) methodology for carcinogenic and toxic risk is implemented. Toxic and carcinogenic assessments take into account the following routes of exposure: Ingestion, inhalation and dermal sorption. For the purpose of this study only the ingestion pathway is considered.

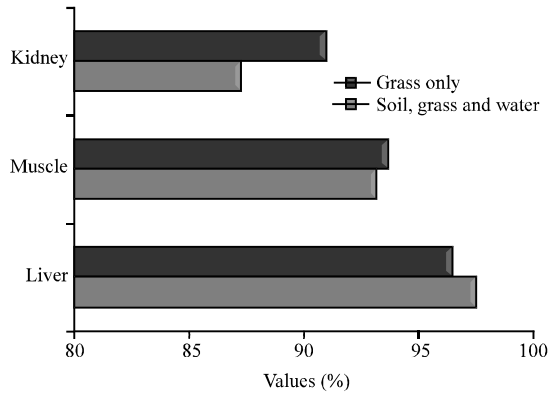

Fig. 7: Model fitness when grass is used as the only source; correlation between observed and stimulated values

Methodology: Before calculating the risks associated with carcinogenic and toxic assessments, the total dose, average daily dose and lifetime average dose have to be defined. The equations used to define risks associated with human exposure to a pollution are generally based on those specified in the EPA "Risk Assessment Guidance for Superfund" (USEPA, 1989).

For each pathway, the total dose that will reach a human has to be calculated. The total dose is defined as:

$$\text{Dose} = C \times IR \times ED \quad (6)$$

Where:

Dose = Total dose (mg)
C = Maximum concentration (mg kg^{-1})
IR = Initial cincentration (kg day^{-1})
ED = Exposure Duration (days)

The Average Daily Dose (ADD) is determined by dividing an estimate of the total dose accrued during the exposure duration from a pathway by an averaging time or an expected lifetime:

$$ADD \frac{Dose}{BW \times ED} \quad (7)$$

Where BW avarage body weight over exposure period (kg).

Carcinogenic risk: Carcinogenic risk assessments are determined over a human's lifetime. Therefore, the Lifetime Average Daily Dose (LADD) is calculated as:

$$LADD \frac{Total Dose}{BW \times Lifetime} \quad (8)$$

The carcinogenic risk calculation is based on a Poisson Model:

Table 4: Available cancer potency factors and reference doses

Factors	Co	Ni	Cu	Zn	Se	Cd	Pb	U
Cancer Potency Factor (CPF)								
IRIS	-	-	-	-	-	-	-	-
ATSDR	-	-	-	-	-	-	-	-
RAIS	-	-	-	-	-	-	0.0085	-
Reference Dose (RfD)								
IRIS	-	0.002	-	0.30	0.005	0.001	-	0.003
ATSDR	-	-	0.90	-	0.005	-	-	0.006
RAIS	0.003	0.020	0.04	0.30	0.005	0.001	-	0.003

IRIS = Integrated Risk Information System; ATSDR = Agency for Toxic Substances and Disease Registry; RAIS = Risk Assessment Information System

$$Risk = 1 - e^{-LADD \times CPF} \approx LADD \times CPF \quad (9)$$

Where CPF is Cancer Potancy Factor (mg/kg/day). The potency factor is the slope of the percentage of animals developing cancer versus the dosage level of a particular chemical. The slope of this curve is then extrapolated to the low doses expected to be encountered by humans who may be exposed to the same chemical. Carcinogenic risk is expressed as a probability of the occurrence of cancer cases, e.g., a calculated risk of 10^{-6} is interpreted as the occurrence of one case in a million. The measure of 10^{-6} as an acceptable risk is sited in various literature. According to Kelly (1991), this figure should be redefined on scientific, social and economic basis. For the purpose of this study, the 10^{-6} was recognised as an acceptable risk estimate.

Toxic risk: The toxic risk is calculated as:

$$Risk = \frac{ADD}{RfD} \quad (10)$$

Where RfD is Reference Dose (mg/kg/day). The reference dose is an estimation of daily exposure to the population (including sensitive subgroups), likely to be without an appreciable risk of deleterious effects during a lifetime. Toxic risks compare the average daily dose of a pollutant to a reference dose calculated for that specific pollutant. Once the average daily dose is equal to or greater than the reference dose, the risk of a person suffering toxic effects due to exposure to the particular pollutant is 99%.

Cancer Potency Factors (CPF) and Reference Doses (RfD): Available sources consulted on CPF and RfD with the associated values for the elements in question are shown in Table 4.

RESULTS

The input data (soil, grass and water) as given in Table 1 and 2 were applied to the predictive cattle

Table 5: Exposure rates of cattle

Variables	Values
Soil (kg day^{-1})	0.75
Grass (kg day^{-1})	12.38
Water (l day^{-1})	38.50

Table 6: Inputs for the human risk model

Variables	Values
Average human weight (kg)	70
Average lifetime (years)	70
Exposure duration (years)	50

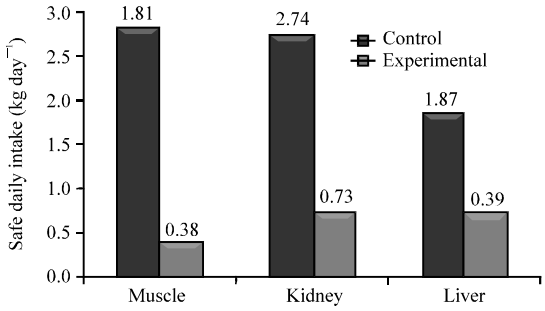

Fig. 8: Safe daily intake for toxic risk assessment (human of 70 kg with a 50 years exposure

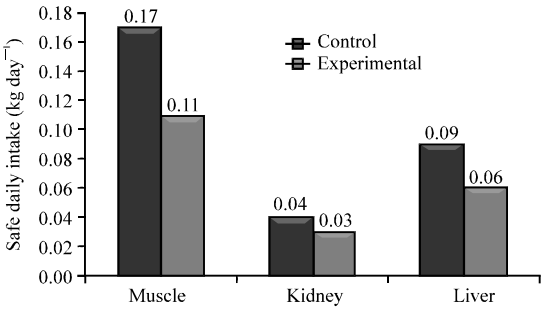

Fig. 9: Safe daily intake for carcinogenic risk assessment (human of 70 kg with a 50 years exposure)

consumption model using the following exposure rates (Table 5) assuming the age of the cattle to be 6 years.

The predicted elemental concentrations of the muscle, liver and kidneys were then used in the risk assessment model. Both the human toxic and carcinogenic risks were calculated for the consumption of muscle, liver and kidneys for both the control and experimental groups. Table 6 lists the assumed inputs for the risk model.

The resultant safe daily intake to avoid toxic risk is presented in Fig. 8. A pronounced difference was found to exist between the control and experimental groups with regard to the toxic risk. On average South Africans consume 49 kg of meat per annum (FAO, 2010) which translates to 0.13 kg day^{-1} and includes all types of meat. Using this figure for daily ingestion of meat, a person will be safe from toxic risks for both the control and experimental groups.

The resultant safe daily intake to avoid carcinogenic risk is indicated in Fig. 9. The daily intake of 0.13 kg of meat a day will only comply with acceptable carcinogenic risk for ingesting muscle of the control group. Note that the acceptable risk is defined as the occurrence of cancer in one person out of a million. Furthermore, the carcinogenic risk assessment is only related to lead as this was the only element in the list of considered elements for which a cancer potency factor was obtained (Table 4).

CONCLUSION

It was shown that a predictive cattle consumption model was developed and calibrated from data gathered from a control and experimental group. Animal matter analysed for both groups were related to the cattle age of 6 years. Although, good correlation between observed and simulated values was achieved, the exiting model fit is non-unique. To obtain a more precise model fit a similar dataset is required for both groups but at a different age.

The predictive model also showed that if only grass were to be used as input, there were no significant changes in the correlation between observed and simulated values. This has a huge advantage in terms of costs associated with laboratory analyses as the analysis of grass will be sufficient for using the model.

A human health risk assessment (toxic and carcinogenic) was performed based on the results of the cattle consumption model. It was shown that no toxic risk exits for both the control and experimental groups if an intake rate of 0.13 kg of meat per day is assumed. For the same consumption rate only muscle consumption in the control group yielded an acceptable carcinogenic risk of the occurrence of one person getting cancer out of a million.

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