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Health Risk Assessment
of Plasticizer in Wastewater Effluents
and Receiving Freshwater Systems

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1. Introduction

A variety of human activities e.g. agricultural activities, urban and industrial development, mining and recreation, significantly alter the quality of natural waters, and changes the water use potential (Spinks et al., 2006; Madungwe and Sakuringwa, 2007). The key to sustainable water resources is, therefore to ensure that the quality of water resources are suitable for their intended uses, while at the same time allowing them to be used and developed to a certain extent. Water quality management, therefore involves the maintenance of the fitness for use of water resources on a sustained basis, by achieving a balance between socio-economic development and environmental protection. Approximately 40 000 small-scale farmers, 15 000 medium-to-large-scale farmers, 120 000 permanent workers, and an unknown number of seasonal workers are involved in irrigation farming, which consumes approximately 51 to 61 % of South Africa's water on some 1,3 million hectares (Backeberg, 1996; Blignaut and Heerden, 2008). Irrigation farming contributes 25 to 30 % of South Africa's agricultural output. Agriculture is crucially important to the basic food security of the poor, who constitute 40 % of the population of 42 million, and who are overwhelmingly concentrated in rural areas and (peri-) urban townships (Blignaut and Heerden, 2008).

Like many countries in the world, water scarcity is becoming a major problem in South Africa (Marcucci & Tognotti, 2002; Oweis & Hachum, 2009; Komnenic et al., 2009) as dams serving communities with drinking water and water for daily household use, have been less than 30% full in recent years (Qiao et al., 2009; Malley et al., 2009). River water, in combination with groundwater, effluents from wastewater treatment plants, is considered a suitable alternative as a utilisable and potable water source (Blignaut and Heerden, 2008). To complement scare water resources, there has been increase in the number of wastewater facilities in many countries. This is to forestall the outbreak of environmental pollution and spread of diseases, remove conventional pollutants (such as ammonia and phosphate), and to maintain and restore the biologic integrity of surface waters (Wang et al., 2005; Sun et al., 2008). Domestic and industrial wastewaters are significant sources of endocrine disrupting chemicals

(EDCs) to the receiving surface, coastal waters and regional environments (Ahel *et al.*, 1994; Ahel *et al.*, 1996; Ying *et al.*, 2002; Vethaak *et al.*, 2005; Voutsa *et al.*, 2006; Zuccato *et al.* 2006).

South African rivers are steadily becoming more contaminated and in some cases even toxic, due to urbanization, industrialization and malfunctioning of wastewater treatment plants in the cities (Fatoki et al., 2004; Jackson et al., 2007; Jackson et al., 2009). Water quality in South Africa has been a major debate considering the water consumption trend in the country both for agricultural development, recreational purposes and domestication usage. Of the major water pollutants that have been relegated to the background in South Africa is Phthalate esters (PE). There was no local interim guidelines for PE in freshwater systems in South Africa, thus pollution of freshwater systems through industrial activities could not be punished. However, water quality is of paramount importance in this country.

Phthalate ester is synthetic compound commonly used as a plasticizer to impart flexibility, workability, and durability to polymers such as polyvinyl chloride. Also, this compound is used in a wide variety of products such as paints, adhesives, inks and cosmetics (Ling et al., 2007; Huang et al., 2007). As a result, PE has become ubiquitously distributed in the environment and easily finds their ways into the river systems through both dry and wet deposition (Yuan et al., 2002; Yuan et al., 2008). PE is considered to be a potential carcinogen, teratogen, and mutagen. Their toxicity to human beings and aquatic organisms is of deep concern (Mylchreest et al., 1999; Awal et al., 2004; Fatoki et al., 2010).

Furthermore, PE acts as endocrine disruptors, which could alter reproductive functions and exert distinct effects on male reproductive organs due to anti-androgenic effects (Latini et al., 2006; Lambrot et al., 2009; Vo et al., 2009). The aim of this study was to assess the potential human impacts health associated with PE found in the final effluent from wastewater treatment plants and river water receiving effluent wastes.

1.1 The risk assessment framework

In recent decades, the interest about environmental issues has increased very quickly. Not only to the natural scientists, but other active members of the society (politicians, industrialists and the general public), have paid much attention in all aspects related to the environment, in general, and environment protection, in particular. In this context, environmental pollution has been one of the fields where more efforts have been aimed to control. Because of the lack of environmental consciousness and technical capacity, many industries released toxic substances into the air, water and soil, for a number of years. As a first consequence, levels of pollution in areas surrounding industrial sites became much higher than background (unpolluted) zones. Recently, implementation of legislative measures carried out by public administrations has obliged to companies to improve their production processes in order to reduce the pollutant emissions.

The concern resulting from the potential exposure to contaminants initiated the development of methodologies that evaluate the consequences that those contaminants can have on environment and human health. Among these methods, risk assessment has been one of the most widely used. Risk assessment is a formalized process for estimating the magnitude, likelihood, and uncertainty of environmentally induced health effects (Sexton *et al.*, 1995). In 1983, the US National Research Council (NRC), in the so-called "Red Book", defined a series of principles to be considered for human health risk assessment, and

defined it as a process in which information is analyzed to determine if an environmental hazard might cause harm to exposed persons and ecosystems (NRC, 1983).

In addition to definition, NRC proposed a framework for human health risk assessment, which involved 4 basic steps (NRC, 1993). The four steps of the process are:

1. Hazard identification
2. Dose-response assessment
3. Exposure assessment and
4. Risk characterization.

1.1.1 Hazard identification

This step can be defined as the qualitative determination of whether or not a particular hazardous agent is associated with health effects of sufficient importance to warrant further scientific investigations. Different kinds of tools (QSAR, short-term toxicity test) are used in order to estimate the chemical damage of a single substance. When establishing the hazard from industrial sources, the chemicals are also identified according to measurements of amount and typology of emissions.

1.1.2 Dose-response assessment

This component is focused on examining quantitative relationships between the magnitude of the exposure (or dose) and the probability of occurrence of adverse effects in the population. Usually, dose-response assessment is based on extrapolations from data about laboratory animals, which have been given high-doses of toxicant and monitored accordingly.

1.1.3 Exposure assessment

Exposure assessment may be defined as the quantitative determination of the extent of exposure of the population to the hazardous agent in question. Since they provide a real knowledge of the state of pollution of an area, data obtained in the environmental monitoring are commonly used as a starting point. Factors that need to be considered include frequency and duration of exposure, rates of uptake or contact, and rate of absorption (NRC, 1993). Other factors in assessing exposure include release patterns, cumulative versus non-cumulative exposure, persistence, failure of exposure controls, quality of data and quality of models.

1.1.4 Risk characterization

This fourth component can be defined as the description of the nature and magnitude of the risk, expressed in terms which are comprehensible to decision makers and the public. Information acquired in the previous 3 steps is integrated in order to communicate the overall meaning of, and confidence in, the hazard, exposure, and risk conclusions. Risk is expressed as a probability of suffering a particular kind of harm from a hazard to a specified group of population (Bennion *et al.*, 2005). Moreover, qualitative and quantitative uncertainty related to risk must be also supplied.

The project aimed at determining the potential health risks that may be associated with using river water and treated effluent from wastewater treatment plants in Cape Town. Since phenols and phthalate esters were placed on the United State Environmental Protection Agency list as priority pollutants, both phenols and phthalate esters congeners were analyzed in water samples. However, emphasis is more on the phthalate esters congeners. The derivatization of the phenolic congeners did not in any way affect the intensity of the phthalate esters congeners included in this study (Olujimi et al., 2011b).

2. Materials and method

2.1 Study areas

Influents and effluents from six wastewater treatment plants namely; Athlone, Bellville (which consist of the Old and New plants), Kraaifontein, Potsdam, Stellenbosch and Zandvliet) were investigated for the occurrence of seventeen organic compounds (eleven priority phenols and six phthalate esters). Five of these wastewater treatment plants (WWTPs) were located in the City of Cape Town, while one is located in Stellenbosch. Rivers associated with each treatment plant are: Athlone - Vygekraal River; Bellville - Kuils River; Kraaifontein -Mosselbank River; Potsdam - Diep River; Zandvliet - Kuils River and Stellenbosch -Veldwachters River. Five of the WWTPs and associated rivers investigated are presented in Figure 1. Samples were taken at the point of discharge, as well as upstream and downstream from point of discharge (about 1-2km) to evaluate the possible impact of effluent on organic compounds load on the aquatic environment. The geographical location, population equivalent and treatment processes of the investigated treatment plants are presented in Table 1.

WWTPID	Geographical Location of plant	People equivalent	Source	Treament Process	River
A	S33.5709° E18.3048°	900,000	Domestic Industrial	S + G + Sed + AS (BNR) + Sed + Chl + AD + Dew	Vygekraal River
B	S33.5923° E18.4332°	591,000	Domestic Industrial	S + G + EAAS (N) + Sed + UVdis + Dew	Kuils River 1
C	S33.82539° E18.70442°	133,000	Domestic	S + G + Sed + AS (N) + Sed + Chl + AD + Dew	Mosselbank River
D	S33.5070° E18.3108°	385,000	Domestic Industrial	S + G + Sed + AS (N) + Sed + Chl + AD + Dew	Diep River
E	S33.94345° E18.82492°	N/K	Domestic Industrial	S + G + Sed + FB + AS (BNR) + Sed + Chl + AD + Dew	Veldwatchers River
F	S34.0312° E18.4259°	400,000	Domestic Industrial	S + G + Sed + AS (N) + Sed + Chl + AD + Dew	Kuils River 2

Abbreviations: S = Screening; G = Grit removal; Sed = Sedimentation; AS = Activated Sludge; EAAS = Extended Aeration Activated Sludge; N = Nitrogen; BNR = Biological nutrient removal; Chl = Chlorination; UVdis = UV disinfection; AD = Anaerobic digestion; FB = Filter bed; N/K = Not known; WWTP ID = Wastewater treatment plant identification.

Table 1. Description of the six wastewater treatment plants investigated.

Fig. 1. Map showing five of the five of the wastewater treatment plants.

2.2 Chemicals and reagents

Analytical grade phenol (PH) 99.9 %, 2-nitrophenol (2-NP) 99 %, 4-nitrophenol (4-NP) 99 %, 2,4-dinitrophenol (2,4-DNP) 99.7 %, 4,6-dinitro-2-methylphenol (DNMP) 98 %, 2,4-dimethylphenol (2,4-DMP) 98 %, 2-chlorophenol (2-CP) 99.8 %, 4-chlorophenol (4-CP) 99 %,

2,4-dichlorophenol (2,4-DCP) 100 %, 4-chloro-3-methylphenol (4-C-3MP) 99 %, pentachlorophenol (PCP) 99.6 %, dimethyl phthalate (DMP), diethyl phthalate (DEP) 99 %, benzybutyl phthalate (BBP) 98 %, dioctyl phthalate (DOP) 99 %, diethylhexylphthalate (DEHP) 99 %, dibutyl phthalate (DBP) 99 % were purchased from Superlco (Bellefonte, PA USA). Helium (99.999 %) is supplied by Afrox gas, South Africa, Potassium Carbonate, acetic anhydride were supplied by Separations (South Africa). The solvents (methanol, n-hexane, acetone and acetonitrile) were of analytical grade from Sigma Aldrich and were further purified by distillation. Separate stock solutions (1000 mgl^{-1}) of individual congeners were prepared in methanol. A working mixture containing each compound at 10 mgl^{-1} was also prepared and stored at 4°C in the dark. Milli-Q water used was from apparatus Millipore (Bedford, MA, USA).

2.3 Derivatization procedure

Some EDCs such as phenols with hydroxyl group within the molecule have to be derivatized with N-Methyl-N- (Tert-Butyldimethylsilyl) trifluoroacetamide (MTBSTFA), which results in the formation of tert-butyltrimethylsilyl (TBMS) derivatives. The high polarity of the phenolic compounds gives rise to poor chromatographic performance and as a consequence derivatization was carried out. The phenol-silylate is more volatile and affords better detection limits when using gas chromatography (GC). The standard mixture was derivatized according to Olujimi et al. (2011b). Briefly, 1 ml of the standard mixture (phenols and phthalate esters) was measured into sample vial and blown to dryness under gentle flow of nitrogen gas. The dried standard mixture was reconstituted with 50 μl acetonitrile and 50 μl silylating reagent N-Methyl-N- (Tert-Butyldimethylsilyl) trifluoroacetamide (MTBSTFA) and mixed in a vortex for 90 s. The solution was derivatized at 90 ºC for 20 min in a GC oven. The sample was cooled down to room temperature and 1 μl was injected into the GC-MS for analysis. The stepwise derivatization procedure is shown in Figure 2. The GC-MS parameters used for the analysis is presented in Table 2 after initial optimization studies.

2.4 Determination of limits of detection and quantification GC-MS

Lower concentration standards were prepared through serial dilution of individual standard of phenols and phthalate esters as well as the mixture standards. 1 μl aliquots of each of the standard was injected into GC, to determine the lowest concentration. Different procedures for the determination of limits of detections (LODs) and limit of quantifications (LOQs) are reported in the literature. These limits can be experimentally estimated from the injection of serially diluted standard solutions or extracts of fortified water samples until the signal-to-noise ratio (s/n) ratio reaches a value of three. LOD was estimated as three times the noise level of the baseline in the chromatogram, while the limit of quantification (LOQ) is set at three times the LOD. For this study, LOD and LOQ were calculated using the equations below:

$$LOD = 3.3 \times Sb/a \tag{1}$$

and

$$LOQ = 10 \times Sb/a \tag{2}$$

where a is the slope and Sb is the standard deviation of the y-intercept (De Sousa et al., 2003).

2.5 Solid phase extraction (SPE) for water samples

C18-E cartridges (strata, 500 mg/ 6 ml) from Separations Limited were used for the extraction of phenols and phthalates from water samples based on recoveries obtained for phenols using HPLC (Olujimi et al., 2011a). Prior to the sample processing, the cartridges were fitted onto a vacuum manifold (Supelco) connected to pump. The cartridges were conditioned with 5 ml of n-hexane:acetone (50:50, v/v), followed sequentially by 5 ml of methanol and 10 ml of Milli-Q purified water (purified by Milli-Q System, Millipore, Bedfore, MA, USA). Prior to extraction of each 500 ml, water samples were filtered on vacuum using a 0.22 μm filter to remove suspended particulate matter that might block the SPE cartridges. Hydrochloric acid (37 %) was used to adjust the pH of the water sample to pH ≤ 3 before passing it through the conditioned cartridge. The cartridge were then rinsed with 5 ml of Milli-Q water and left on the vacuum manifold for 30 min to dry (-70 Kpa). The retained analytes of interest were eluted with 3.5 mL of methanol followed by 3.5 ml of n-hexane:acetone (50:50, v/v) into 10 ml glass vials. This was blown to dryness on hot plate at 70 °C under gentle flow of nitrogen gas. The retained analytes were then derivatized according to the procedure described in section 2.3 (Figure 2).
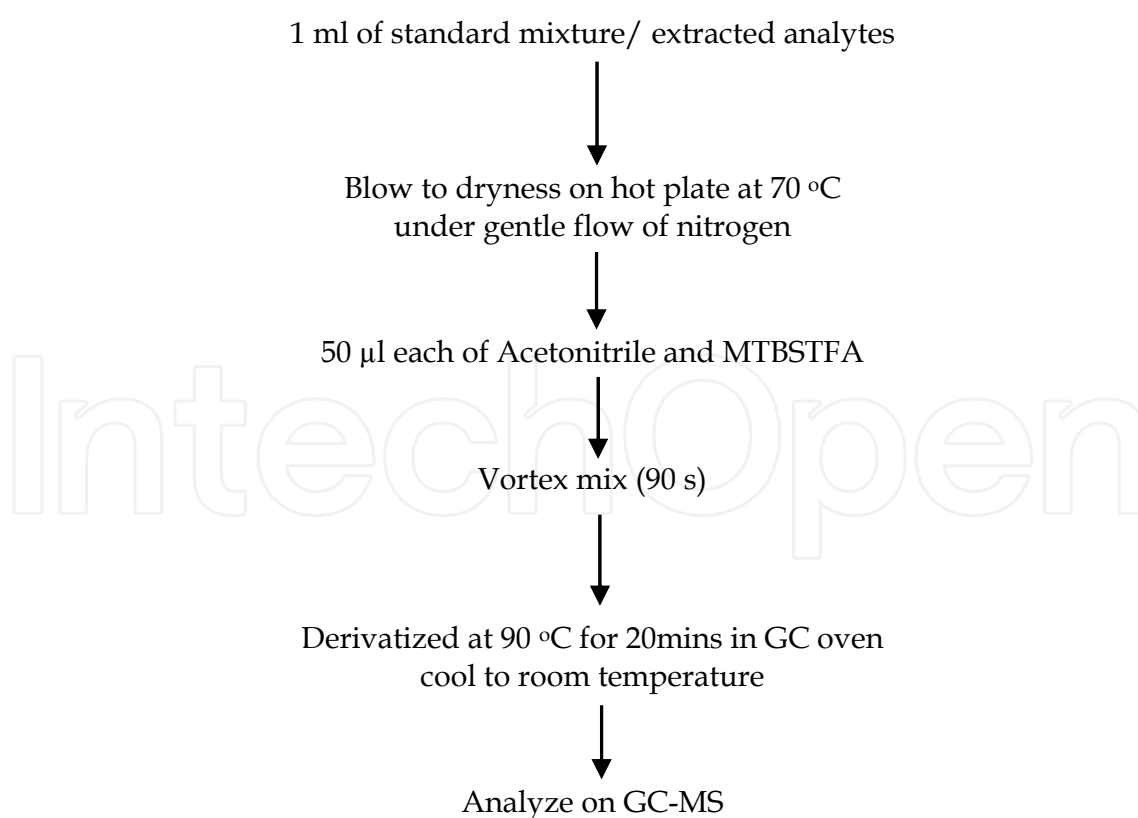
1 ml of standard mixture/ extracted analytes

↓

Blow to dryness on hot plate at 70 °C
under gentle flow of nitrogen

↓

50 μl each of Acetonitrile and MTBSTFA

↓

Vortex mix (90 s)

↓

Derivatized at 90 °C for 20mins in GC oven
cool to room temperature

↓

Analyze on GC-MS

Fig. 2. Derivatization procedure for silylation.

Gas chromatography		Mass spectrometer	
GC-MS	Agillent 6890N	5975	
Capillary Column	DB-5MS, 30 m x 0.25 mm i.d. (0.25 μm film thickness)	Ion source	Electron impact ionization, 70 ev
Carrier gas	Helium, purity: 99.999 %	Ion source temperature	230 °C
Injector parameters	1 μl splitless, injection temperature 260 °C	Inlet temperature	260 °C
Oven temperature	80 °C (1 min) -5 °C min^{-1} 150 °C held for 1 min, then to 280 °C at 12 °C (7 min), carrier gas flow rate: 1.0 mlmin^{-1} Post run temperature: 300 °C (2 min)	Transfer line Scan mode (m/Z)	280 °C 50-450

Table 2. Gas Chromatography and Mass Spectrometer Parameters.

2.6 Seasonal sampling protocol

Water samples for organic compounds analysis were collected from the wastewater treatment plants and rivers on quarterly basis. This was to observe the possible impact of seasonal variation on organic compounds in wastewater treatment plants and possible impact this could have on the concentration of congeners in the freshwater systems. Sampling started in April 2010 and ended in March 2011.

2.7 Quality assurance and quality control (QA/QC) for GC-MS

Spiked procedural blanks, solvent blanks and control samples were included in each batch of analyses. Blanks and controls were treated similarly as the samples and analyzed after every sample injection. A calibration standard solution of 50 μgl^{-1} was injected in duplicate to monitor the instrumental sensitivity and reproducibility every time before sample analyses.

2.8 Health risk assessment

A human health risk assessment was conducted to provide an indication of whether the organic compounds or heavy metals detected in the water samples tested may cause adverse health effects to human. The methodology used to assess this potential human health risk was that described by US-EPA (1988, 1996) and the WHO (2002). The exposures considered in the assessment include:

a.   Ingestion through drinking of final effluents or river water,
b.   Dermal absorption due to daily washing/bathing in the river water,
c.   Irrigating farm lands with final effluent or river water,
d.   If fish from these areas is consumed.

Human exposure to toxic effects are expressed in terms of average daily dose (ADD) which is the amount of substance taken into the body on daily basis during the exposure period calculated

$$ADD = (C_{medium} \; x \; IR \; x \; ED \; x \; F_c) \, /BW \; x \; AT \; (mg/kg \cdot d) \tag{5}$$

where:

ADD = average daily dose C_{medium} = concentration in the contaminated water
IR = daily intake rate
ED = exposure duration
F_c, = the fraction contaminated
BW = body weight
AT = lifetime averaging time

For risk of carcinogens for exposures that last less than lifetime, the dose is adjusted using the formula:

$$LADD = ADD \; x \; (ED/Lft) \tag{6}$$

where: Lft is lifetime

2.8.1 Non-cancer toxic effects (Hazard Quotient)

For agents that cause non-cancer effects, a Hazard Quotient (H.Q) was calculated, comparing the expected exposure to the agent to an exposure that is assumed not to be associated with toxic effects.

For oral or dermal exposures, the Average Daily Dose (ADD) was compare to a Reference Dose (RfD):

$$\text{H. Q.} = \text{Average Daily Dose} \, / \, \text{Reference Dose} \tag{7}$$

Any Hazard Quotient less than 1 is considered to be safe for a lifetime exposure.

2.8.2 Cancer risk

For chemicals that may cause cancer if ingested, risk is calculated as a function of Oral Slope Factor and can was calculated by using the formula:Risk = Oral Slope Factor * Lifetime Average Daily Dose (8)

2.8.3 Cross-media transfer equations used to generate exposure estimates

The formulae used to generate the contaminant exposure concentration in water were those described by the US-EPA (1990) for water to fish; vegetables; dairy and meat concentrations. The formula for the consumption of recreationally caught fish and shellfish-water to edible tissue is presented in equations below:

$$C(f) = BCF * (\frac{fat}{3}) * C(w) \tag{9}$$

$$BCF = [0.79 * \log (Kow)] - 0.40 \tag{10}$$

Where:C (f) = concentration in fishC (w) = concentration in waterC(sd) = Concentration in Sediment

DN = Sediment Density (Relative to Water Density of 1.0 kgl^{-1}) (1.90)
OC = Organic Carbon Fraction of Sediment (4.00 %)
Koc = Octanol-Carbon Partition Coefficient of the Compound
Kow = Octanol - Water coefficient of the compound
BCF = Bioconcentration factor

2.8.4 Homegrown fruit and/or vegetables – Water to root (root uptake)

Limitations: not applicable to polar species, where RCF(w) = 0.82

$$C(r) = RCF (w) * C(w) \qquad (11)$$

$$\log(RCF(w) - 0.82) = (0.77 * \log(Kow)) - 1.52 \qquad (12)$$

Where:

C(r) = Concentration in root Calculated
C(w) = Concentration in water Chemical Specific
Proportion in root 100.00 %
RCF = Root concentration factor
Kow = Octanol-water partition coefficient of the compound Chemical Specific

2.8.5 Homegrown fruit and/or vegetables – Water to transpiration stream (root uptake)

$$C(st) = TSCF(w) * C(w) \qquad (13)$$

$$TSCF(w)= 0.784 \exp -((\log(Kow) - 1.78) *2)/2.44) \qquad (14)$$

Where:

C(st) = Concentration in Stem Calculated
C(w) = Concentration in Water Chemical Specific
Proportion in Stem: 100.00 %
TSCF = Transpiration Stream Concentration Factor
Kow = Octanol-water partition coefficient of the compound Chemical Specific

2.8.6 Homegrown meat or dairy – Water to edible tissue

Limitations: If either of these conditions occur, BCF = 0, Log Kow < 3.5, Log S > 4

where S is the water solubility of the compound.

$$C(t) = BCF(f) * F * C(w) \qquad (15)$$

$$\log(BCF(f)) = -3.457 + 0.5 (\log(Kow)) \qquad (16)$$

Where:

C(t) = Concentration in edible tissue calculated

C(w) = Concentration in water chemical Specific
F = Fat content in tissue (dairy) 4.00 %
F = Fat content in tissue (Meat) 14.00 %
BCF = Bioconcentration factor for tissue fat chemical specific
Kow = Octanol-water partition coefficient of the compound chemical Specific

2.8.7 Exposure parameters used to calculate exposure estimates

The dose estimates in this assessment, as well as the risk estimates derived from them, refers only to the specific exposures that have been described in Table 3. The average daily dose was calculated taking into account the concentration of the chemicals in water, sediment, for a 70 Kg adult, assuming an intake of 0.054 kg fish on a daily basis (equivalent to 378 g per week). A range of risks is presented making use of average and 95th percentile concentrations of chemicals detected in water, calculated to represent concentrations expected in fish. The 95th percentile represents the "reasonable maximum" risk.

Exposure parameter	Amount
Events per year	350
Kg fish per day	0.054
Kg dairy	0.4
Kg meat per day	0.1
L water per day	2
Body weight	70 kg
Exposure duration	30 years

Table 3. Exposure parameters used to generate exposure estimates.

3. Result and discussion

The LOD of each compound for the analytes was determined as three times the standard deviation of seven independent replicate analyses. LOQs were determined as 3.3 times of LODs. Instrument detection limits ranged from 0.6 µgl⁻¹ (DEHP) to 3.16 µgl⁻¹ (4-NP) and the LOQs varied from 1.9 µgl⁻¹ (DEHP) to 10.44 µgl⁻¹ (4-NP) as presented in Table 4. The LODs and LOQs values are adequate for environmental monitoring of the target compounds and low enough compared to previous work on the analytes of interest (Fatoki and Noma, 2002; Yuan *et al.*, 2002; Cortazar *et al.*, 2005; Zhou *et al.*, 2005; Kayali *et al.*, 2006; Ling *et al.*, 2007) taking into account the complexity of the samples and the low sample amounts used. For wastewater and river samples, the LODs achieved in the present work were at similar levels or lower than those obtained in previous studies with GC–MS (Yuan *et al.*, 2002; Cortazar *et al.*, 2005; Kayali *et al.*, 2006). The chromatogram of the derivatized phenols and phthalate esters congeners are presented in Figure 3.
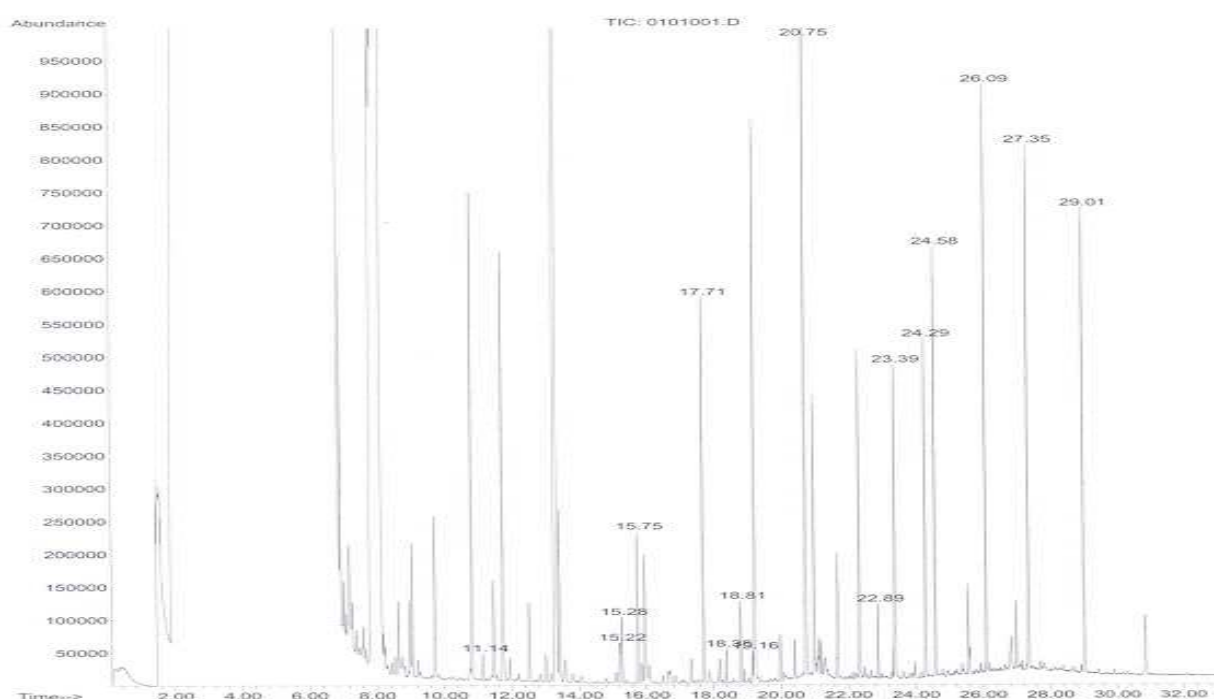
Fig. 3. Chromatogram of derivatized phenols and phthalate esters.

Five point calibration curves were constructed using triplicate injections of the derivatized standard. The retention time, target ion monitored, and the SPE recovery of the selected phenols and phthalates are presented in Table 4. Analysis of the result demonstrated the concordance of the response with a linear model as shown in Table 4, where the regression coefficient ranges from 0.976 to 1.000. The method precision and accuracy were satisfactory. The detectable concentration range was from 2.5 to 1000 μgl^{-1}. Due to non-availability of reference materials, the validation of the analytical method for extraction and elution was assessed through the recovery of standard mixtures of the target analytes in Milli-Q water. For the efficient quantification of the target compounds, analysis was performed within the linear portion of the calibration curve.

3.1 Health risk assessment

There are many associated adverse health effects if people are exposed to these chemical contaminants in excess doses. Where possible the study looked at whether people might be exposed to excessive concentrations through various pathways, such as if water were used for domestic purposes, if the water were used to irrigate vegetables, if fish living in the water were eaten on a regular basis, if the rivers were used for recreational swimming and lastly if meat were consumed from the area making use of the water. The classic example of a population that differs from the norm is subsistence fishers, who may consume as much as 10 times the amount of freshwater fish that most citizens do.

This population is of particular concern when evaluating surface water contamination in areas that are economically depressed or if the immune systems of the people in the area are compromised. The methodology used to asses this potential human health risk was that described by the US-EPA (1988, 1996) and the WHO (2002), making use of the risk assessment programme, Risk Assistant ™ (Thistle Publishers, 1996). DEHP and DBP were

the only organic chemicals of those tested that could be included in the quantitative health risk assessment.

Compound	Retention Time (min)	Target ion (m/z)	Reference ion (m/z)	SPE Recovery (%)	LOD (µgl⁻¹)	LOQ (µgl⁻¹)	Correlation Coeffient R^2
Phenol	11.14	151	208	93.43 ± 0.05	2.2	7.18	1.000
2-CP	15.21	185	149, 93	98.21 ± 4.38	1.9	6.34	0.988
DMP*	15.27	163	77	83.72 ± 6.03	2.2	7.43	0.993
2,4-DMP	15.74	179	163, 149, 105	98.69 ± 8.43	1.4	4.78	0.987
4-C,3MP	17.71	199	93	76.21 ± 5.28	2.96	9.77	0.989
DEP*	18.38	149	177, 104, 77	98.46 ± 11.31	1.58	5.22	0.993
2,4-DCP	18.81	219	183, 125,93	94.1 ± 7.16	1.11	3.66	1.000
2-NP	19.15	196	180, 151, 136, 91	95.39 ± 11.68	1.36	4.47	1.000
4-NP	20.74	196	150, 135	88.19 ± 10.29	3.16	10.44	0.999
2,4,6-TCP	20.76	255	217, 159, 93	73.21 ± 0.05	2.81	9.63	0.999
DBP*	22.89	149	207	98.99 ± 8.27	0.9	2.9	0.978
2,4-DNP	23.39	241	225, 195, 137	96.34 ± 2.93	1.63	5.36	0.986
2-M, 4,6-DNP	24.29	255	239, 209 179, 149	90.33 ± 6.18	1.48	4.87	0.976
PCP	24.58	323	93	92.64 ± 11.39	2.23	7.37	0.998
BBP*	26.09	149	206, 91	97.43 ± 18.31	0.6	2.9	0.987
DEHP*	27.35	149	279, 167	101.32 ± 0.21	0.6	1.9	0.989
DOP*	29.01	149	279, 57	90.77 ± 5.39	1.41	4.65	0.988

*Compound not affected by MTBSTFA derivatization

Table 4. Retention time, target ion, limits of detection and quantification in GC-MS of the selected phenols and phthalates recoveries (n = 7).

The average concentrations detected in all the sample sites over the sampling period of a year was used as a most likely scenario to determine what risks (if any) were involved as a screening risk assessment. If a chemical was found to be responsible for risks considered by the US-EPA and WHO to be unacceptably high, a more detailed assessment for that chemical was investigated, making use of the spread of the data, averages, and identifying which sampling site was responsible for the highest concentrations detected. The following graphs (Figures 4 & 5) illustrates the average concentrations of the chemicals detected at the sampling sites used in the primary screening for human health risk assessment.

DBP was found at highest concentrations in both river water samples and wastewater effluents, followed by nitro-phenol (NP) and DEP (Figures 4 & 5). Human dose-response data was available for DEHP and DBP to allow a quantitative health risk assessment to be performed (ATSDR, 1995; 2001; 2002). The results of the exposure calculations are given in the Table 5 and are presented as both Average Daily Dose (ADD) and Lifetime Average Daily Dose (LADD) in mg/kg/d.
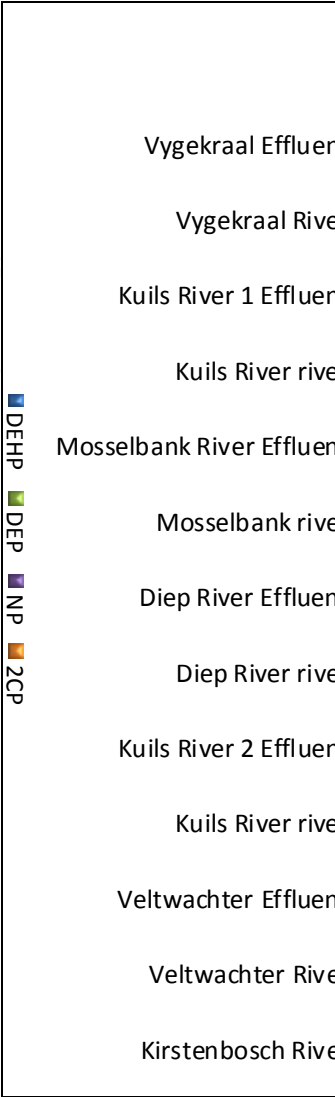
Fig. 4. Concentrations (µgl⁻¹) of phthalate and phenolic congeners detected in river and WWTP effluents at different sites (excluding DBP).

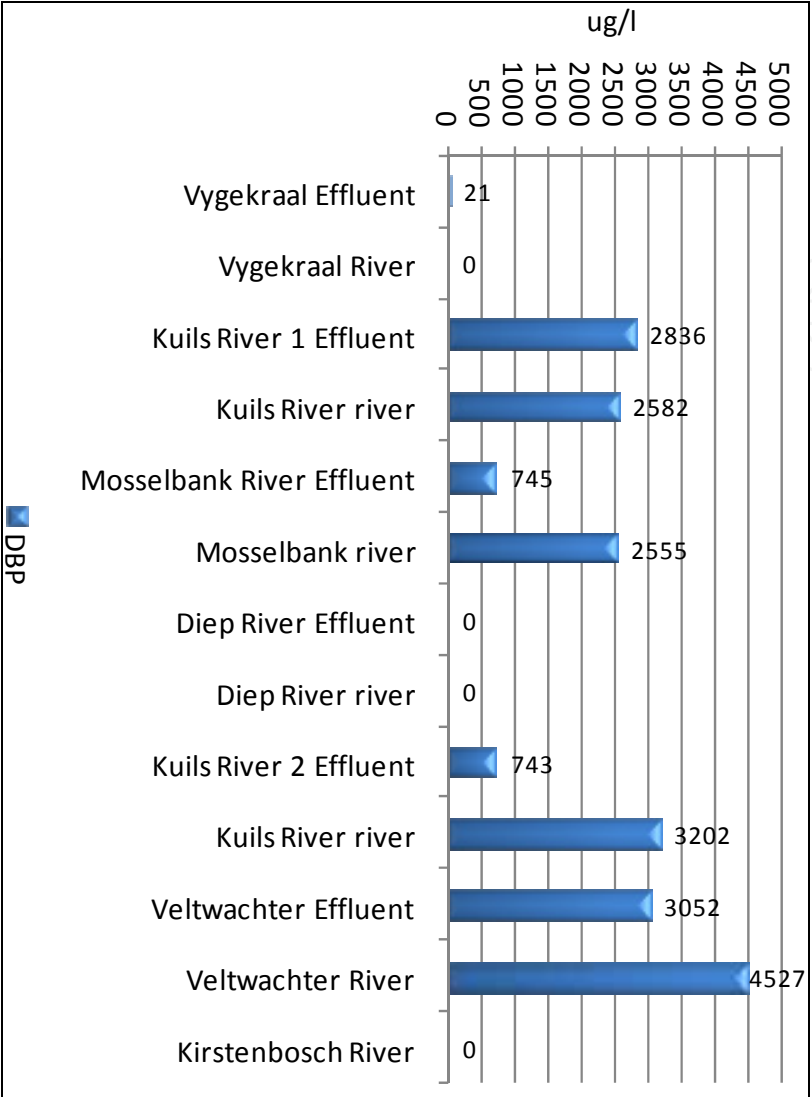
Fig. 5. DBP concentrations (µgl⁻¹) detected in effluent and river water samples at the different sites.

Based on the exposure assumptions described in the section above, risks of developing cancer and toxic effects were calculated for the various phthalate chemicals where sufficient data was available. Most of the chemicals were found at concentrations to be below those where "unacceptable" risks, as defined by both the WHO and US-EPA, are anticipated. However, risks of developing cancer may be as high as 2 in one thousand resulting from exposure to DEHP (Figure 6) resulting predominantly from exposure through vegetables that have been irrigated with the contaminated water, and to a lesser extent, through the consumption of fish, grown in the contaminated water.

DEHP was detected at high concentrations at Kirstenbosch, Kuils River, Mosselbank River and Vygekraal River. In general, river waters contained higher concentrations than treated effluents of the waste water treatment works (Figure 6). This risk would result if the water were used to irrigate vegetables or if fish grown in the water were consumed on a regular basis.

Site	Chemical	ADD (mg/kg/d)	LADD (mg/kg/d)
Vygekraal River	DEPH	0.02474	0.0106
	DBP	0	0
Vygeraal Effluent	DEHP	0.007983	0.0003421
	DBP	0.003242	0.001389
Kuils River 1	DEHP	0	0
	DBP	0.3895	0.1708
Kuils River (1) Effluent	DEHP	0.007982	0.003421
	DBP	0.4377	0.1876
Mosselbank River	DEHP	0.06066	0.0206
	DBP	0.3943	0.169
Mosselbank Effluent	DEHP	0.03233	0.01385
	DBP	0.115	0.04927
Diep River	DEHP	0.003592	0.001539
	DBP	0	0
Diep River Effluent	DEHP	0.003991	0.00171
	DBP	0	0
Kuils River (2)	DEHP	0.1014	0.0435
	DBP	0.4941	0.2118
Kuils River (2) Effluent	DEHP	0.1189	0.05097
	DBP	0.1147	0.04914
Veldwachter River	DEHP	0.04191	0.1796
	DBP	0.6986	0.02497
Veldwachter Effluent	DEHP	0.05827	0.02497
	DBP	0.471	0.2019
Kirstenbosch Stream	DEHP	0.1278	0.1278
	DBP	0	0

Vygekraal Effluent = Athlone WWTP effluent; Kuils River (1) Effluent = Bellville WWTP Effluent; Mosselbank Effluent = Kraaifontein WWTP Effluent; Kuils River (2) Effluent = Zandvliet WWTP Effluent Veldwachter Effluent = Stellenbosch WWTP Effluent; Kirstenbosch Stream = Control Site.

Table 5. Predicted total average daily doses and lifetime average daily doses, based on average concentrations of phthalates.
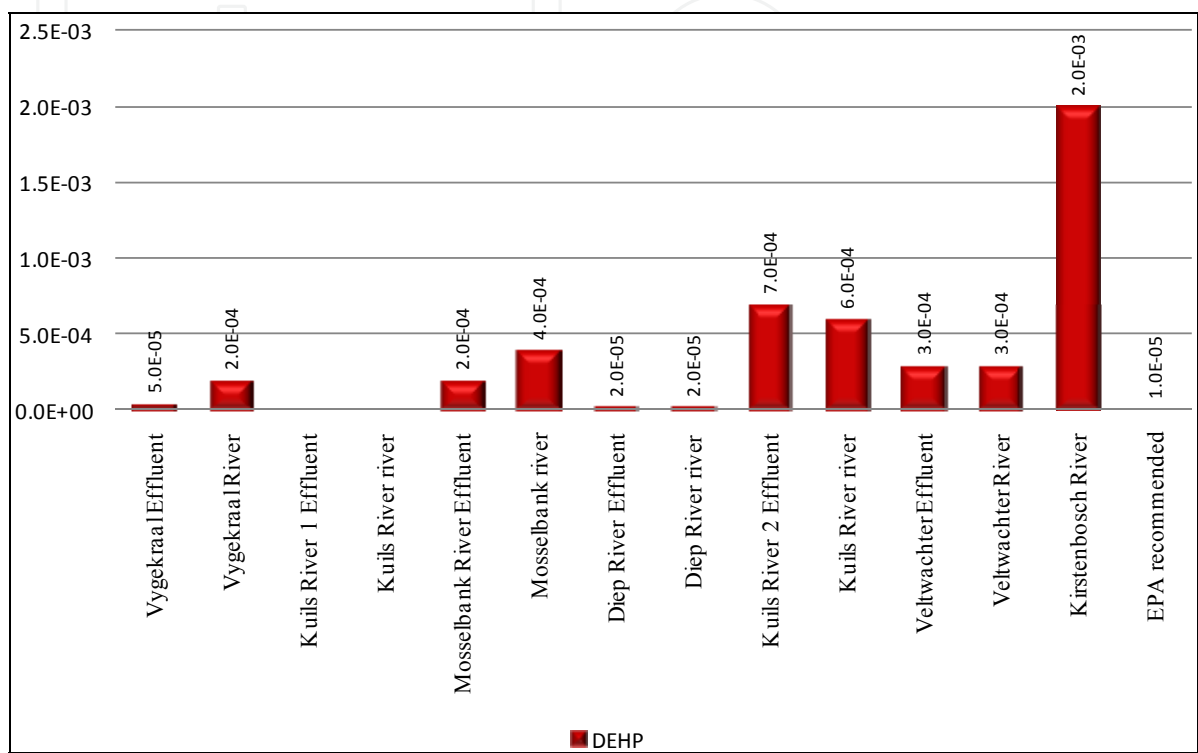
Fig. 6. Cancer risks from DEHP exposure.

Toxic risks could be anticipated resulting from exposure to both DEHP and DBP with individual exposure concentrations predicted at up to 14 times that considered to be safe for a lifetime exposure (Figure 7 & 8). However, the certainty of the reference dose, or the dose considered to be safe, has a safety factor of 100 built into it for both DEHP and DBP (ATSDR, 2002; and ATSDR, 2001 respectively). The safety factors built into the reference doses for DEHP and DBP are to allow for extrapolation from animals to humans (a factor of 10) and to allow for variability within humans (another factor of 10) (ATSDR 2001 & 2002). The predicted risks indicate that a possible risk exists and does not indicate a definite risk as the exposures are modelled and not based on actual measurements.

The driver of the human health risk was identified through this exercise. The chemicals responsible for the risks include DEHP and to a lesser extent, DBP (Figures 6 & 7). DEHP was found to be the major contributor of risk of developing cancer in this screening health risk assessment. The highest potential risks were observed at Kirstenbosch resulting from DEHP detected in the river water. The potential risk through the use of this water is if it were used to irrigate vegetables.

This section examined whether possible human health effects might be anticipated based on chemical contaminants detected in wastewater effluents and in rivers throughout the Western Cape, South Africa. In order to determine whether this is possible, a human health risk assessment was conducted by modelling the chemical contaminant concentrations expected in vegetables, fruit, fish and meat based on levels detected in water. Trans-media calculations (water to fish; water to fruit and vegetables and water to meat) were conducted based on individual chemical parameters described in the earlier sections.

The screening risk assessment identified the chemicals that could be responsible for adverse health effects if drinking the untreated water or eating fish , fruit, vegetables or meat , over a 30 year period were to occur. Although not present at the highest concentrations, the chemicals that were of principal concern were identified as DEHP and to a lesser degree, DBP and arsenic. The type of adverse effect that might result was also identified as predominantly carcinogenic, with possible reproductive system toxic effects being anticipated, as the predicted doses were well below those considered safe by the WHO and US EPA.
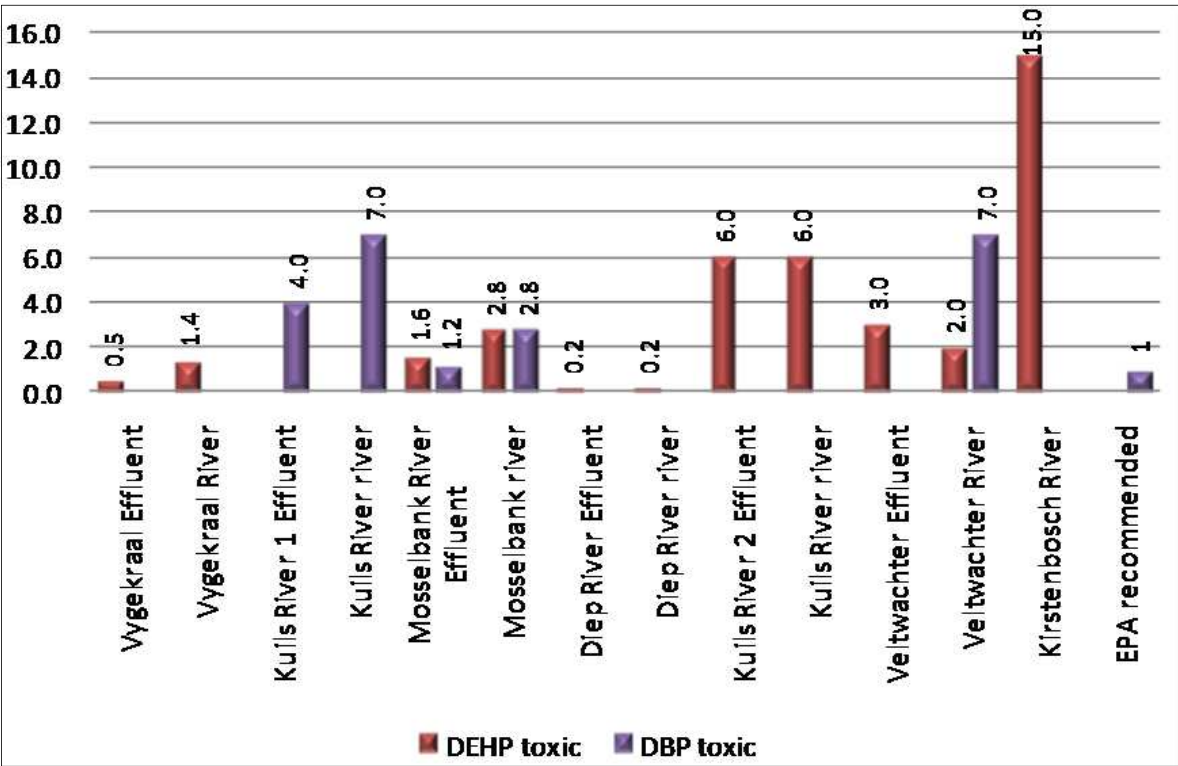
Fig. 7. Hazard quotients for individual phthalates.
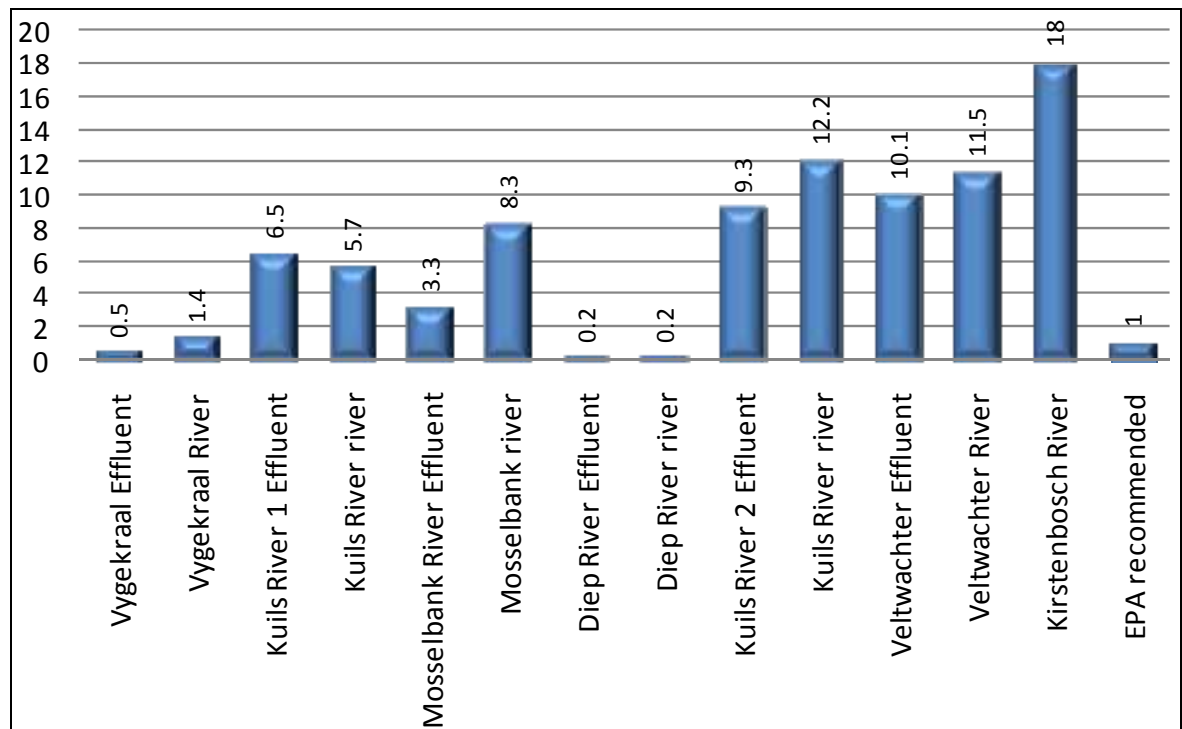
Fig. 8. Hazard quotients for total phthalates.

4. Conclusion

This screening risk assessment has highlighted that possible health risks can be anticipated resulting from ingestion of vegetables irrigated with the water and ingestion of fish from the rivers on a regular basis. There are many uncertainties in any health risk assessment, and this study presents a screening or rapid human health risk assessment. Seasonal and spatial variations were considered in this health risk assessment as the average concentrations tested over the 4 seasons were used in the average daily dose calculations. In addition to sample variation, dose calculations also represent uncertainty, based on the assumption of the number of times a year that people eat certain foods and the amount of that food eaten. Future investigations need to focus on verifying the uptake of phthalates into vegetables and fish via water as this has highlighted that although levels were considered to be safe in the water, bio-accumulation is possible into both fish and vegetables to levels considered to be unacceptable by the US EPA and WHO.

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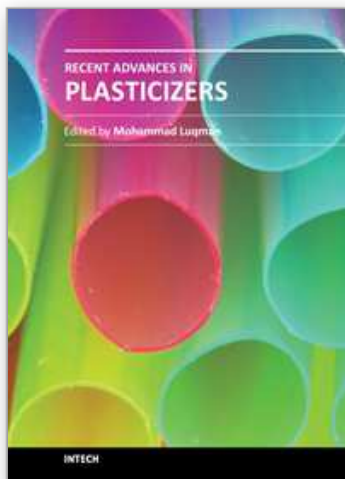
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