Effect of Seawater Intrusion on Human Health Risk and Toxicity of Disinfection By-products

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Objectives

1. To evaluate the formation of trihalomethane (THM) under varying levels of seawater intrusion (SWI).
2. To determine the human health risks associated with THMs in coastal groundwater with varying levels of SWI.
3. To calculate the toxicity of THMs to mammalian cells due to increasing degrees of SWI.

Introduction

With increasing human activities and climate change, SWI is becoming a growing concern for coastal communities. The quality and characteristics of such waters differ from those of surface and groundwater. SWI imparts unusual concentration of bromide, a major precursor of brominated THMs. THMs are known to be carcinogenic and have been linked to various health issues, including reproductive problems, developmental delays, and cancer.

Methodology

SWI was simulated using synthetic seawater (ASTM D1141-98). Deionized water (100 mL) was mixed with SSW at 0%, 0.25%, 0.5%, 1%, 2%, and 3% seawater by volume.

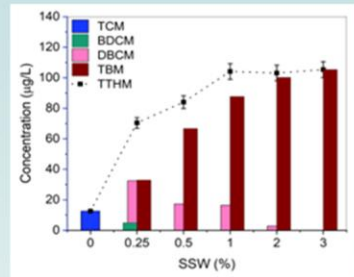
Chlorination of these samples was carried out as per uniform formation condition (UFC). THM concentration was measured after 24 hours of chlorination using a gas chromatograph equipped with an ECD.

Human health risk assessment models recommended by the USEPA was adopted with several modifications to calculate cancer risk (CR). To predict the toxicity of THMs, the methodology utilized the LC_{50} values of CHO cells. The toxicity values and CR were then correlated with the varying levels of seawater intrusion (SWI) to determine the degree of toxicity associated with increasing levels of SWI.

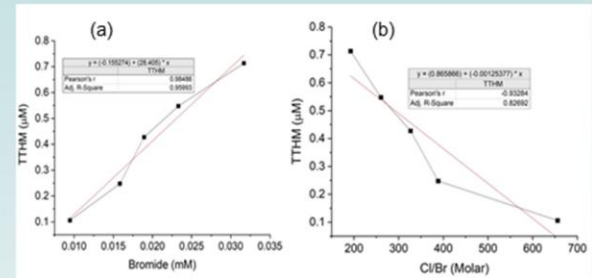
Monte Carlo simulation was utilized for both assessments.

Details are given in Supplementary Materials.

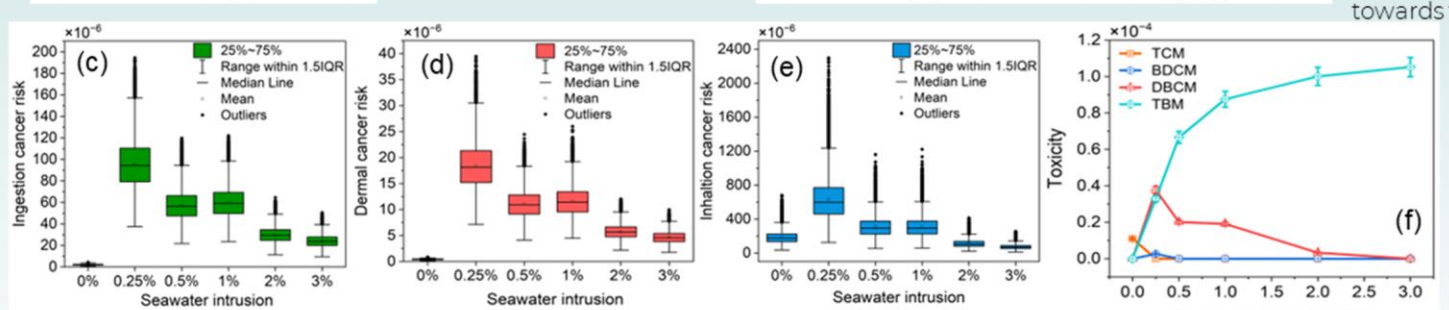
Results



THM formation and speciation with increase in SWI. **(a)** TTHM concentration was positively correlated with bromide concentration and **(b)** negatively with Cl/Br ratio.

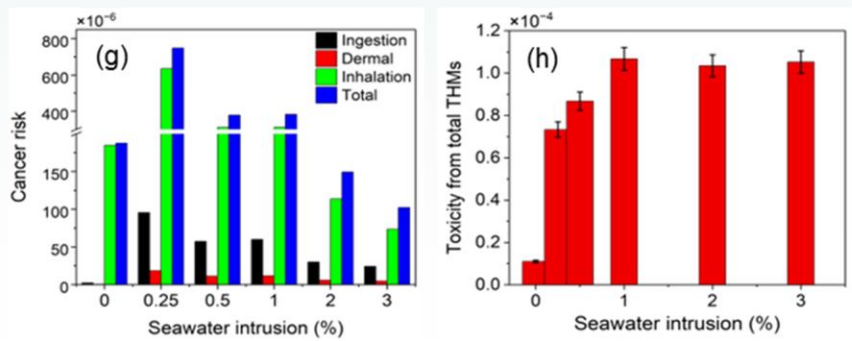


The TTHM concentrations increased from 12.64 µg/L to 105.34 µg/L after 24 h and to 115.8 µg/L after 48 h as SWI increased from 0% to 3%. In the case of no intrusion, only TCM was formed as there was no bromide in reaction mixture. Thereafter, a shift towards the Br-THMs was observed with increasing SWI. Cancer risk and toxicity increased to certain extend. High intensity of SWI resulted in negligible conc. of chlorinated THMs. As a result, reduction in CR and toxicity was observed.



Cancer risk through **(c)** ingestion, **(d)** dermal contact, and **(e)** inhalation of THMs

Increase in toxicity to CHO cells by four THMs **(f)**



(g) Total cancer risk from TTHM and **(h)** toxicity to CHO cells from TTHM

Conclusion

- SWI significantly increased the formation of THMs during chlorination.
- Monte Carlo simulations show that the total cancer risk increased by 4 times for an increase of SWI from 0% to 0.25%.
- The toxicity of THMs to CHO cells also increased with increasing degrees of SWI, with the highest total toxicity observed at SWI = 1% by volume.
- The findings by policymakers and water managers to make informed decisions about coastal water management.

Sharing is encouraged

THM concentration in water under the influence of SWI

THM (ug/L)	Seawater Intrusion (% by volume)					
	0	0.25	0.5	1	2	3
TCM	12.64	0	0	0	0	0
BDCM	0	5.00	0	0	0	0
DBCM	0	32.48	17.31	16.46	2.86	0
TBM	0	32.95	66.71	87.67	100.21	105.34

Cancer Risk

Reference: Parveen and Goel 2023, 10.3390/toxics11040295

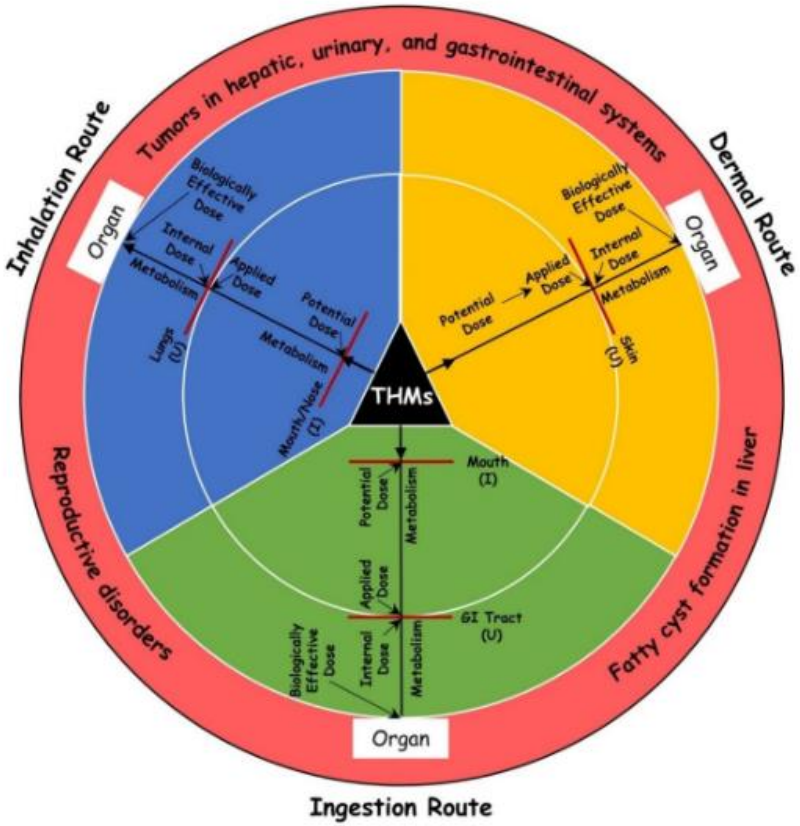


Figure 1. Schematic of source-to-effect of three exposure pathways of THM in human exposure models. Red lines are either exposure surface (a surface on a receptor where an agent is present) or absorption barrier (any exposure surface that can retard the rate of penetration of an agent into a receptor). I: intake; U: uptake; potential dose: the quantity of agent that go into a receptor after passing an exposure surface that is not an absorption barrier; applied dose: the amount of agent at an absorption barrier; internal dose: the amount of agent that enters a receptor by crossing an exposure surface acting as an absorption barrier; biologically effective dose: the quantity of agent that reaches the target internal organ or tissue where the harmful outcomes arise.

Cancer Risk

CR is the cancer risk, *CDI* is the chronic daily intake of THM (mg kg^{-1} day^{-1}), and *CSF* is the cancer slope factor associated with each THM ((mg kg^{-1} day^{-1})$^{-1}$).

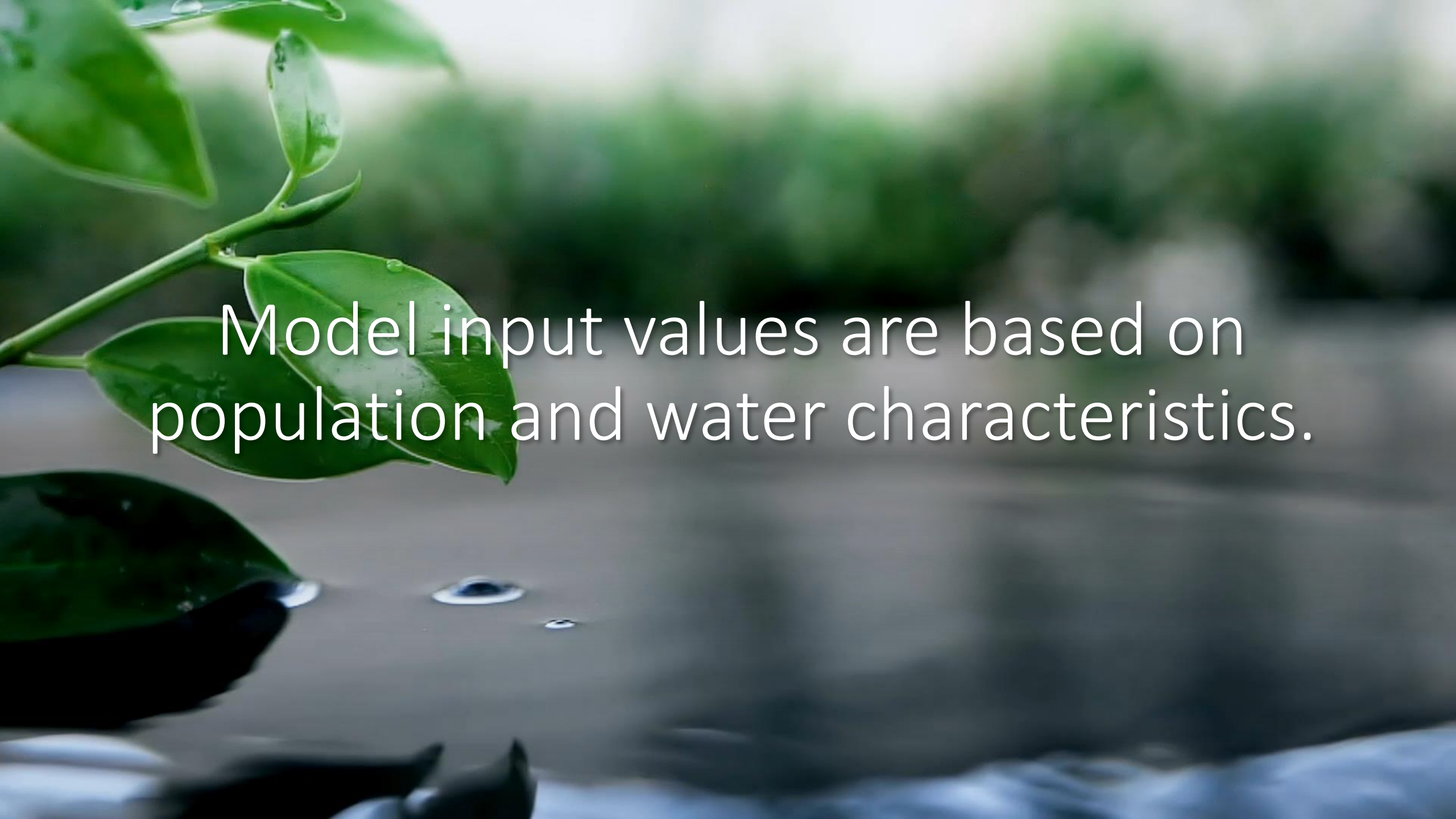
$$CR = CDI \times CSF$$

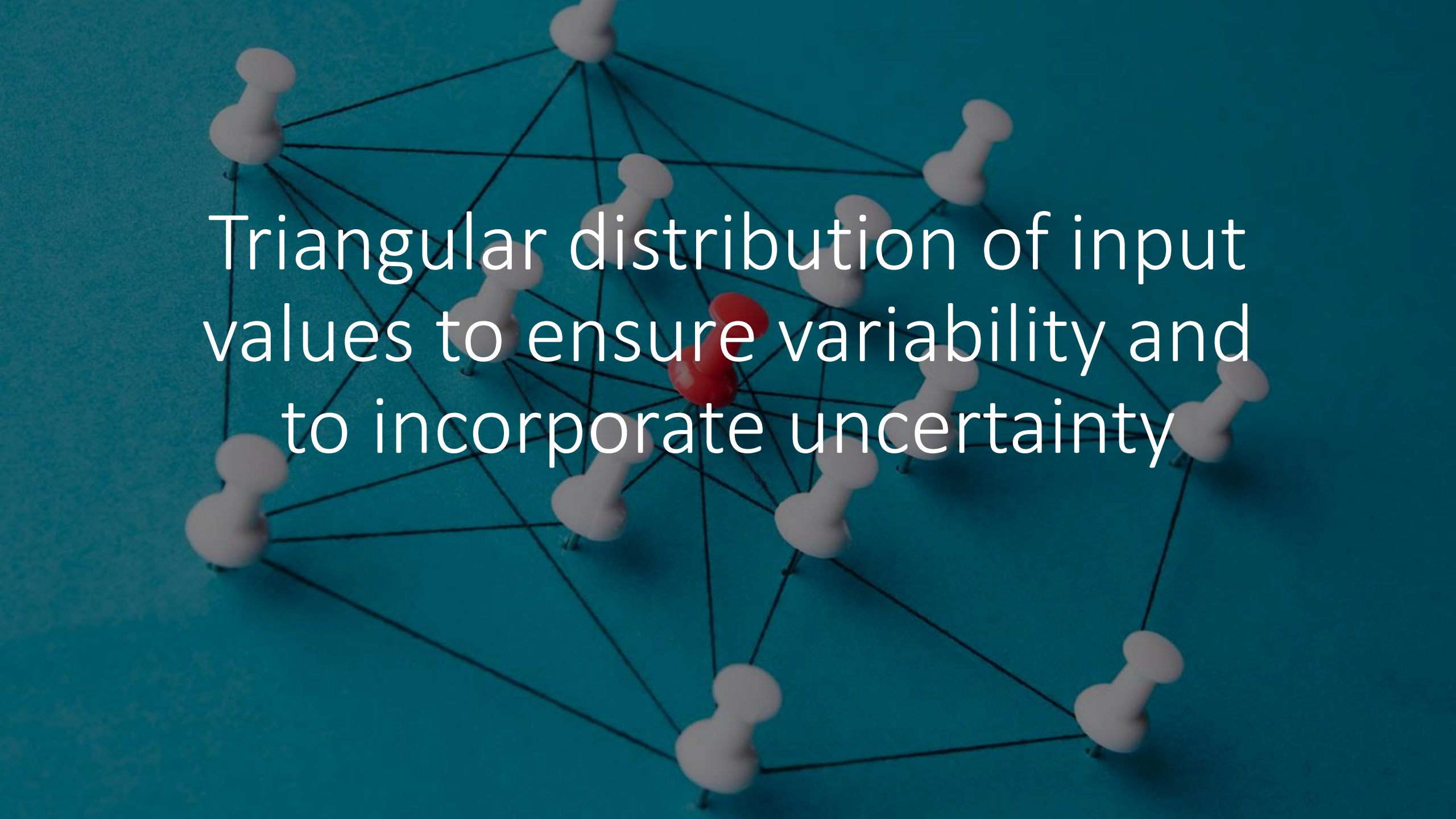
Cancer Risk Assessment

- Ingestion
  - $CDI_{ing,i} = \frac{Cw_i \times IR \times EF \times ED \times CF}{BW \times AT}$
- Dermal Contact
  - $CDI_{der,i} = \frac{Cw_i \times SA \times Pd \times t \times F \times EF \times ED}{BW \times AT}$
- Inhalation
  - $EC_i = \frac{Cair_i \times ET \times EF \times ED}{AT}$
  - $CR_i = EC_i \times IUR_i$

Where,

$CDI_{ing,i}$ is the chronic daily intake of ith THM through ingestion (mg kg^{-1} day^{-1}), Cw_i is the concentration of ith THM in water (µg L^{-1}), IR is the ingestion rate of drinking water (L day^{-1}), EF is exposure frequency (days $year^{-1}$), ED is exposure duration (year), CF is the conversion factor from µg to mg (0.001), BW is body weight (kg), AT is averaging time (days) $CDI_{der,i}$ is the chronic daily intake of ith THM through dermal absorption (mg kg^{-1} day^{-1}), Cw_i is the concentration of ith THM in water (µg L^{-1}), SA is the skin surface area (m^2), Pd is the THM permeability of human skin (m min^{-1}), t is the showering duration (min $events^{-1}$), F is the showering frequency (events day^{-1}), $Cair_i$ is the concentration of ith THM in shower air (µg m^{-3}), EC_i is the exposure concentration of ith THM in the air (µg m^{-3}), ET is the exposure time (h day^{-1}), AT is the averaging time (h), CR_i is the inhalation cancer risk, and IUR_i is the inhalation unit risk associated with ith THM ($(\mu g\ m^{-3})^{-1}$)


Model input values are based on population and water characteristics.



Triangular distribution of input values to ensure variability and to incorporate uncertainty

Toxicity to CHO cells

THM	LC_{50} Values* (µM/L)
TCM	9620
BDCM	11500
DBCM	4130
TBM	3960

* From Wagner and Plewa (2017), https://doi.org/10.1016/j.jes.2017.04.021

The overall toxicity of THM4 for varying intensity of sweater intrusion was calculated as:

$$Toxicity = \sum_{1}^{i} \frac{C_i}{LC_{50,i}}$$

C_i is the concentration of THM in µM/L and LC50 is the lethal concentrations that reduced the CHO cell density by 50%