Adjusting Relative Source Contribution

What is Relative Source Contribution (RSC)

EPA introduces the concept of relative source contribution (RSC) as applied to human health water quality criteria in its 2000 Human health methodology (EPA, 2000). RSC is a means to account for non-water sources of exposure, in other words, the fact that people are exposed to toxic substances through a variety of pathways, or sources, only some of which are (can be) addressed by water quality criteria. As EPA explains, those other sources should be acknowledged to ensure overall protection of human health, resulting in a reduction in the reference dose allocated to setting human health water quality criteria. While this reduction can be done quantitatively with enough information, on a chemical specific basis, it can also be expressed relatively, as a fraction of the whole allowed dose; like pieces of an exposure pie.

Since the idea of the RSC is to keep the exposure pie from exceeding the reference dose (RfD), if any part of the pie grows, e.g. greater fish consumption, then the pieces of the pie must be reapportioned to keep the sum of relative exposures at unity. Exposure is a product of quality and quantity, thus any change in the FCR or DI used in human health criteria calculation implies a different RSC.

Factors Affecting RSC

RSC is ultimately about the masses of contaminant an individual receives from various sources, reduced to proportions, fractions of the whole. While these fractions vary by each person’s habits, for criteria purposes of protecting a population, they are taken to be a representative characteristic of the population to be protected. Furthermore, the mass contribution from any one source is a product of source quality and quantity. The focus of this paper is change in quantity. Assuming that quantity and quality are independent variables, increasing one’s consumption of fish or intake of water does not change their quality.

Because each contaminant has different chemical and physical properties affecting how it moves through and among environmental media, an RSC is contaminant specific. A big factor in this specificity is the rate at which a contaminant bioaccumulates in the food chain, represented by its bioaccumulation factor (BAF). BAF acts as an exposure magnifier, e.g. a BAF of 100 means eating 20 g of fish delivers as much contaminant mass as 2000g (also ml) of water. Higher bioaccumulation not only means an increasing fraction of exposure comes from fish rather than water; it may also mean fish consumption dominates all other sources of exposure. This appears to be the case with mercury.
With respect to fish consumption and water intake, there are two sets of human health criteria – one for fish only exposure and another for fish plus water exposure – representing decidedly different exposure scenarios. These correspond to different uses that may be made of surface waters; recreational use – which in Idaho includes fishing – and domestic water supply where drinking of the water and fishing may both occur. The RSC for fish plus water exposure is clearly greater than for fish only exposure. How much so depends upon the strength of bioaccumulation, with a smaller bioaccumulation factor (BAF) leading to more of a difference.

Finally, if the regulatory fish consumption rate increases, then the relative amount of exposure due to fish must increase.

Because food chains (and thus bioaccumulation), patterns and types of chemical release, and people’s fish consumption and other lifestyles are regional, RSC also varies by where we live.

Our fish consumption habits shouldn’t alter our non-diet exposures. On the other hand, fish consumption is part of a broader diet, maybe the dominant component of our diet.

**EPA’s Default RSC**

The following figure represents EPA’s default breakdown in sources between fish and water included in criteria development (RSC = 0.2) and other sources (RSC= 0.8):

Other sources can be divided into other diet and other non-diet exposures (non-diet including inhalation, dermal, etc.). The following figure depicts a hypothetical breakdown of all other sources, in addition to fish and water included in the regulatory FCR:
This breakdown of other sources is purely hypothetical; EPA offers no default proportions as they do for RSC.

Do other sources matter? They do because fish consumption is part of a broader diet, maybe the dominant component of our dietary exposure to contaminants. Therefore, unless our caloric intake is increasing, an increase in fish consumption means less exposure from other dietary exposures, and a greater proportion of exposure from fish consumption. On the other hand, our fish consumption habits shouldn’t alter our non-diet exposures. Thus increasing FCR without also increasing RSC implies a receptor is getting more contaminants from other sources than is the case. This will come again later.

When looked at in a relative sense, various sources can be seen as pieces of a pie, which must add up to one-the whole. If one slice increases, another must shrink, at least relatively. This also leads one to consider that the relative size of the pie slices does not necessarily have to reflect how sources currently proportion; they could instead represent how we want to allocate dose among sources.

**Review of EPA’s Exposure Decision Tree Approach**

EPA establishes RSC as a matter of risk management policy. That policy applies RSC to non-carcinogens as well as carcinogens with a non-linear dose response. For carcinogens with a linear low-dose extrapolation, EPA explains that criteria are set based on “the incremental lifetime risk posed by a substance’s presence in water, and is not being set with regard to an individual’s total risk from all sources of exposure.”

The RSC policy establishes “… a “ceiling” level of 80 percent of the RfD and a “floor level” of 20 percent …” This range appears to be justified based on this statement “The underlying
objective is to maintain total exposure below the RfD (or POD/UF) while generally avoiding an extremely low limit in a single medium that represents just a nominal fraction of the total exposure.” EPA then provides an Exposure Decision Tree for deciding which of three values, 0.8, 0.5, or 0.2 should be used for RSC. The decision tree appears to be focused on variability in the quality component of exposure for a fixed quantity of fish consumption and/or drinking water intake, namely 17.5 g/day and 2.0 L/day respectively.

EPA’s 2000 Human Health methodology also speaks to “the need to bridge the gap between the differences in the risk assessment and risk management approaches used by EPA’s Office of Water for the derivation of AWQC under the authority of the CWA and Maximum Contaminant Level Goals (MCLGs) under the Safe Drinking Water Act (SDWA).” Thus the floor and ceiling values are borrowed from EPA’s drinking water program. No rationale beyond the above is provided for the three RSC policy values.

EPA’s discussion speaks of water and non-water sources, and water sources as consisting of drinking water intake and fish ingestion. Upon reading EPA’s entire discussion one is left with understanding that water sources means local water sources, and non-water sources include everything else, including non-local sources of fish and water. This is most evident in the exclusion of marine fish from EPA’s national fish consumption rate, and statements that “the marine portion should be considered an other source of exposure when calculating an RSC for dietary intake” and that marine fish may be included “provided that the marine intake component is not double-counted with the RSC estimate.” Hereafter, when we refer to ‘water’ exposure we mean the included fish & water incorporated in criteria calculation in the sense used by EPA in their 2000 HH Guidance.

Non-water sources can be broken down into excluded fish and water, other dietary, and non-dietary, the latter including things such as inhalation and dermal exposures. So some of the excluded dietary exposure may, does in EPA’s national 304(a) human health criteria, take in consumption of some fish and water, or water containing fluids, not sourced in local surface waters criteria will be applied to.

The proportion of exposure from both water intake and fish ingestion attributable to local water resources versus other non-local dietary sources is fungible.

**Adjustment of RSC**

Idaho has decided to include only local fish and shellfish in its consumption rate used to derive human health criteria for toxic substances. We know that there is consumption of other, non-local fish. For example, NOAA tracks sources of consumption of fish in the US and reports 84% of the fish consumed in the US is imported (NOAA, 2014), and their [Fish Watch](https://www.fishwatch.noaa.gov) web page puts this at 90% for seafood. This imported fish is presumably sold in the marketplace. In Idaho we
do not yet know how much fish is bought versus caught from local waters. In addition, some of
the fish caught in Idaho are anadromous and may be considered non-local in so far as their
accumulation of contaminants is concerned (DEQ, 2014). Under EPA’s scheme for RSC non-
local fish is accounted for in other exposures. Although we do not know yet for Idaho the
breakdown, i.e. relative consumption of non-local versus local fish, we can say that as local fish
consumption increases its relative piece of the exposure pie must increase as well.

Consideration of the above leads DEQ to propose a simple adjustment of the RSC. We follow
EPA Exposure Decision Tree to step 4. There we rely upon “generalized information available”
to propose reapportioning EPA’s default 0.2/0.8 split between water and non-water exposure (or
more accurately local-water and all-other, including some other water exposure), to adjust for
difference between Idaho’s local fish consumption rate and EPA’s national default fish
consumption rate of freshwater and estuarine fish.

EPA makes no distinction in applying their RSC for fish only versus fish + water criteria.
Although logically the actual relative contribution cannot be the same, we are nonetheless left
with a default RSC of 0.2 in the case of both sets of criteria. This illustrates the arbitrary nature
of EPA’s policy decision of a fixed default range for RSC.

In order to add fish and water together, and take into account the magnifying effect BAF has on
exposure to fish borne contamination, we introduce the idea of ‘Exposure gram equivalents’, or
EGE:

\[
One \ ml \ of \ water \ weighs \ one \ gram \ and \ provides \ 1 \ EGE, \ 2000 \ ml \ provides \ 2000 \\
EGEs. \ One \ gram \ of \ fish, \ at \ a \ BAF = 1 \ also \ provides \ 1 \ EGE, \ and \ 17.5 \ g \ provides \\
17.5 \ EGE. \ Let \ ‘water’ \ EGE = (FCR*BAF) + DI. \ So \ the \ combined \ EGE \ is \ 2018.
\]

Here is our premise:

1) EPA’s default RSC of 0.2 is associated with a nationally recommended fish consumption
rate of 17.5 g/day based on consumption of freshwater and estuarine fish, and intake of
2.0L of water.

2) EPA recognizes that this default RSC should be adjusted upward if marine fish are
included in a regulatory RSC, or double accounting of sources would occur.

3) Increase in exposure is proportional to increase in consumption, if there is no change in
concentration. So while the fish consumption and drinking water intake may depend
somewhat on quality, we assume quality of fish and water consumed is not altered by
choosing a higher regulatory FCR or DI. Thus, increased ‘water’ exposure to
contaminants is directly proportional to the increase in fish consumption and water
intake.

4) We also assume increased ‘water’ exposure’ does not change other exposures. Thus our
exposure pie increases in size by the amount of increased exposure equivalents. This may
not be entirely true; increasing water exposure may come at the expense of other dietary exposures when it comes to fish consumption. Allowing the exposure pie to grow is a conservative approach we’ll come back to.

Thus a regionally greater fish consumption rate would imply a necessary adjustment of the RSC, as magnified by BAF. So does an increase in drinking water intake imply an needed RSC adjustment, but without the magnifying effect of BAF.

Here is how our proposed adjustment works for the fish + water criteria:

I. We take EPA’s default RSC of 0.2 as a starting point. This default RSC of 0.2 means all other sources account for 4 times as much exposure as does the accounted for fish + water consumption (0.8/0.2 = 4).

II. With 17.5 g/day as the regulatory FCR at a BAF of 1 and 2000 ml DI, we could express included exposure in EPA’s 2002 human health criteria, as 2018 EGE and other exposure 4 times that ((0.8/0.2) x 2018) or 8070 EGE. And the overall exposure is 10088 EGEs. Call that our base exposure.

III. Now let’s say we learn that the fish consumption rate we account for in water quality criteria has increased to 22g/day, and drinking water intake to 2400 ml.

IV. Included fish + water now accounts for 2422 EGE’s, while total EGES increase to 10492. We are looking at this relatively, so the RSC for included fish + water is now 2422 / 10492 = 0.23.

This simple adjustment accounts for the necessary increase in relative contribution when fish consumption and drinking water intake included in the regulatory increases, and other sources are held constant. This is depicted graphically below:
A modest increase in RSC reflecting the dominance of exposure by water at a BAF =1.

In this simple adjustment we have let the pie grow, from 10088 EGEs to 10492 EGEs. In reality the pie may not grow, or grow as much. This would occur if as we increase included fish consumption other dietary consumption decreased, through diet substitution. When fish consumption gets large enough it will account for much of our caloric need, consequently diminishing our other dietary sources.

We could recognize this as a constraint by fixing the size of the pie at the starting base size of 10088 EGE. If we did so other sources would shrink in EGE’s by as much as included fish and water consumption increased. With our example above fish we would get RSC = 2422 / 10088 = 0.24. Graphically:

On the surface this is more realistic, as it represents shifting of exposure from other fish and water to included fish and water, or from other sources period. But we do not know the breakdown of other sources, how much is excluded fish and water or something else, and we don’t have EPA guidance on the matter, as we do with the 20/80 split. If we allow that the exposure pie in fact grows with greater fish consumption, as we did in our first example approach, we are being more conservative (less of a change in RSC). We also avoid the possibly of adjusted EGE’s for included fish and water eclipsing the starting pie size. Thus we have decided it is best in this simple adjustment to keep other sources fixed, and allow the whole to increase in size as included exposure grows with increasing FCR, DI, and BAF.

**Effect of increasing BAF**

The above two example were at a BAF of 1, no bioaccumulation. Bioaccumulation magnifies exposure due to fish consumption – eating 1g of fish at a BAF of 10 is the same as eating 10g of
fish with a BAF of 1. And the range in BAF is much greater than the range in fish consumption, can be an overriding factor in exposure, why we worry about highly bioaccumulative substances.

Going back to EPA’s default RSC of 0.2 we see that it applies to both the fish only and fish + water criteria, even though they provide different ‘water’ exposures. How much different depends on BAF, which interacts with FCR. An increase in BAF does not change exposure from water but does change exposure from consumption of fish. At a BAF of 100 the same 17.5g of fish provides 1750 EGE.

Since BAF is a magnifier of exposure due to fish tissue, the separate fish and water contributions reach balance at a BAF of 114, where 17.5 g of fish consumption is equal in contribution to 2.0 liters of water. At lower BAFs water exposure becomes increasingly dominant and exceed >90% of combined exposure due to fish + water when BAF drops to ~12.7. At higher BAFs fish exposure becomes increasingly dominant and exceed >90% of combined exposure due to fish + water when BAF rises above 1029. Let’s round those to 100, 10, and 1000 call them, moderate, low and high bioaccumulation respectively.

The table below shows how RSC changes taking into account increase in FCR with rising BAF, holding drinking water intake at 2.4L/day.

Table 1. RSC adjusted from EPA 2002 base by FCR and BAF

<table>
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<tr>
<th>BAF</th>
<th>FCR 6.5 g/day</th>
<th>FCR 17.5 g/day</th>
<th>FCR 22 g/day</th>
<th>FCR 44 g/day</th>
<th>FCR 88 g/day</th>
<th>FCR 110 g/day</th>
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<td>1.00</td>
</tr>
</tbody>
</table>

It can be seen from this table that BAF is more important than fish consumption in altering relative ‘water’ exposure.

The adjusted RSC can be calculated for any combination of BAF, FCR and DI as follows:

\[
((\text{FCR} \times \text{BAF}) + \text{DI}) \div \left[\left(\left(17.5 \times 1\right) + 2000\right) \times 4\right] + ((\text{FCR} \times \text{BAF}) + \text{DI})
\]

Where an adjustment of RSC has already been made by EPA using their decision tree approach the effect of BAF should be accounted for, but not the effect of increased FCR or DI. In this case it is suggested that an adjustment at BAF =1 be applied to the already adjusted RSC.
Reference:

