

Practical considerations on food safety objectives

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Abstract

The concept of food safety objectives (FSO) is very strong in that it may make food safety transparent and quantifiable. This brings a major advantage in that one can ensure food safety at the process where it is the most effective in meeting the overall integrated objective. A practical overview is given how to derive FSOs from population health goals, through product group health objectives. Then these FSOs can be used to assign the responsibilities over the various parts of the food chain, and within one part of the chain over the various process stages, linking finally the limits of the CCPs in HACCP to the overall public health objective. Determination of characteristic numbers (log change in numbers) can help to supply the quantification of the various parts. Finally, the impact of the statistical distribution of the concentration of pathogens in foods is taken into account, and how it impacts compliance to an FSO.

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1. Introduction FSO: food safety objective

The principle of food safety objectives as proposed by the ICMSF (2002) and CCFH (Codex Alimentarius Commission (2004)) is very simple, and that is its power. By integrating the changes in a hazard from the initial level (H_0) minus the sum of the reductions (R) plus the sum of growth (G) and (re)contamination (C) one arrives at a concentration/prevalence that at consumption must be lower than a food safety objective (FSO) (Fig. 1).

This FSO is a concentration and/or a prevalence based on the so-called appropriate level of protection (ALOP). The ALOP clearly shows the philosophy that greater public health good is achieved by setting a public health goal and then determining the frequency and/or level of a hazard in food that is compatible with that goal rather than trying to eliminate all hazards from

the food supply. One can better set an “appropriate” level and assure that it is not exceeded, than to mislead people believing that zero risk exists. The word appropriate can also be considered as dynamic and that in the future one might set another level.

Definitions and the correct representation of units is of importance:

- First of all the equations are based on log values. (For clarity FSO is only used on log basis and 10^{FSO} is used if not on log basis).
- To avoid misunderstanding one should always clearly distinguish between concentration and dose and it is important to report units: concentration (organisms per gram) or dose (organisms per serving, for example 100 g, differs by a factor 100).
- One should clearly define the end-point and the corresponding/appropriate units of risk: whether it is infection, illness, or death (endpoint), and the population that is considered. Whether risk is measured as health outcome per consuming occasion, year, or

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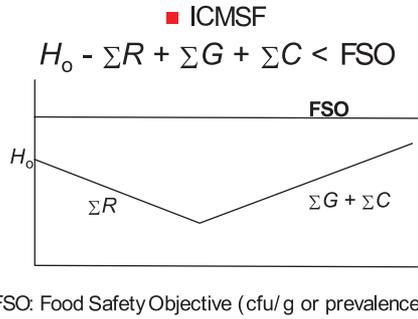


Fig. 1. Schematic representation of the FSO-concept.

lifetime exposure is of very large importance. This seems obvious, but in many publications the unit cannot be figured out, therefore much more emphasis on the correct reporting is necessary. Reporting of a number without defining the “case” and the population on which it is based and the time frame, is of no use.

2. Setting of the FSO: distribution of ALOP over product groups and translation of health burden to concentration

In order to think of the way to translate an ALOP into an FSO, one should first consider about the flow of information in a structured way. If one accepts this procedure, one can include in this process additional factors like stochastic behaviour or safety factors, but first one should look at the basic steps.

If one has set for example an ALOP for *Listeria monocytogenes* mortality of 5 deaths/million/year for the total population, one may first attribute this ALOP over the various sources (food, water, person–person etc.) or product groups, since an FSO can be based on different specific product groups (for example ready-to-eat foods, raw meats, etc.).

$$\text{ALOP} = \sum \text{ALOP}_p \quad (1)$$

In words, the total number of cases (ALOP), equals the sum of the cases per source or product group (ALOP_p), the number of cases per million per year in this example. It should be realised that if the FSO is defined for a specific group of food products or even a very global group of food products, there always may be additional product groups and other sources determining the health objective.

If, for example, one tolerates 1 death/million/year for smoked fish, 3 deaths/million/year for ready to eat meats and 1 deaths/million/year for raw milk cheeses (note: assuming that these three products are the sole responsible for listeriosis; $1 + 3 + 1 = 5$), one can translate this level (1 death/million/year) to an FSO for raw milk cheese. To determine this FSO, one needs the total consumption per year and the infectivity of the cell:

$$\begin{aligned} \text{ALOP} &= \text{deaths per million per year} \\ &= \text{servings per million per year} \\ &\quad \cdot \text{probability of mortality per serving} \\ &= \text{servings per million per year} \\ &\quad \cdot \text{probability of mortality for one cell} \\ &\quad \cdot \text{dose} = S \cdot 1\text{E}6 \cdot r \cdot D \end{aligned} \quad (2)$$

with S being the number of servings per person per year, r the probability of mortality following exposure to 1 organism, and D the dose consumed. This holds if the dose is in the range where the probability is proportional to the dose. With the dose equal to the product of the mass per serving (M) and the concentration, which is 10^{FSO} (since FSO is the log of the concentration) one gets:

$$10^{\text{FSO}} \cdot M \cdot S \cdot 1\text{E}6 \cdot r = \text{ALOP} \quad (3)$$

If for instance the per person consumption of raw milk cheese is 50 servings of 30 g/year and the probability of mortality after consumption of 1 *L. monocytogenes* is $7.2\text{E}-12$ (assuming 20% risk group * $1.2\text{E}-10$ probability of illness (Buchanan, Damert, Whiting, & van Schothorst, 1997) * 30% mortality) and using the equation:

$$\begin{aligned} 10^{\text{FSO}} \cdot \frac{30 \text{ g}}{S} \cdot \frac{50S}{p \cdot \text{year}} \cdot \frac{1\text{E}6p}{\text{million}} \cdot 7.2\text{E}-12 \frac{\text{death}}{\text{List}} \\ = 0.0108 \frac{\text{death}}{\text{year} \cdot \text{million}} \cdot \frac{\text{g}}{\text{List}} \cdot 10^{\text{FSO}} = 1 \frac{\text{death}}{\text{year} \cdot \text{million}} \end{aligned}$$

with S meaning serving and p persons.

So this results in an FSO of 2 ($10^{\text{FSO}} = 100$ *Listerial* g). Graphically this is represented in Fig. 2. It should be noted that the curvature in Fig. 2 depends on both the infectivity of the organism (r) and the total consumption per year ($1\text{E}6 \cdot M \cdot S$).

This is the basic calculation scheme. But there are important attention points:

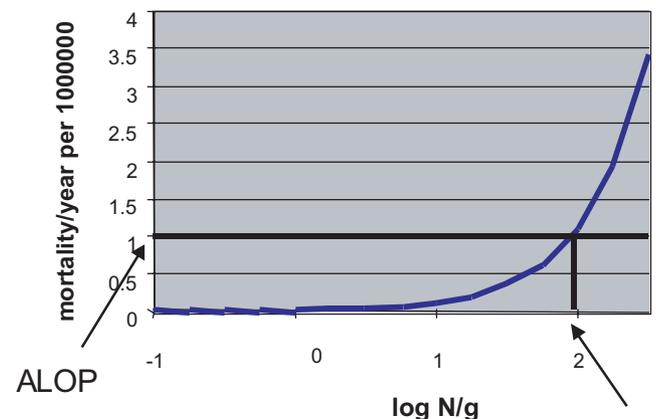


Fig. 2. Relation between ALOP and FSO.

- (1) The dose response relation is not linear for high doses (what a high dose is depends on the infectivity of the organism. As long as $D \cdot r < 0.1$ the error of assuming that the probability is proportional to the dose is below 5% for the exponential model).
- (2) All variables are stochastic and this needs to be taken into account.
- (3) The concentration in food in the distribution chain is often assumed to be log normally distributed. Therefore, it are often the high doses with low probability that are determinant for the response. So one should not set an FSO on a level only, but on an average value (or a probability lower than x that it is higher than a certain level).
- (4) If prevalence is lower than 100% this factor needs also been taken into account. The FSO is then not a log concentration, but can be set as the concentration multiplied with the prevalence. So if the concentration is 500 cfu/g in 20% of the cases (and 0 in the remaining 80%), this is considered equal as 100 cfu/g, so $FSO = 2$ (this holds if one is still in the linear part of the dose–response curve, i.e. 20% probability of a five times higher illness probability, results in an equal risk).

3. Distribution over the chain

In order to meet the FSO at the end of the chain, one can set performance objectives (PO) along the chain. The PO is a term equivalent to the FSO but indicates the targets at earlier stages; targets that will allow the FSO to be met. In this manner responsibilities and specifications of all partners in the chain may be quantified, agreed, and transparent. This has a great advantage in that one can do the main interventions at the stage where it is the most effective. Within a single segment of the chain one can subdivide again the PO over the various steps in the process with performance criteria (PC), for example for a specifically required reduction (e.g. 10^6 reduction). This goes along with the establishment of process criteria (for example 71.5 °C, 15 s) or product criteria (for example pH < 4.5). In this manner process and product criteria in for example HACCP are all together interconnected to the FSO and thus to the overall ALOP (Fig. 3).

4. Quantitative methods

To estimate the values in the FSO equation one can use microbiological methods or use quantitative microbiology. Characteristic numbers (Zwietering, 2002) showing the change in log numbers, can supply the necessary numbers for the equation in a direct way for every

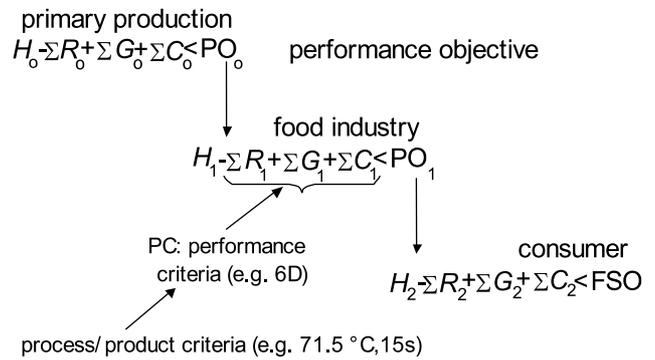


Fig. 3. Link of process/product limits with FSO.

stage in the chain, with the first characteristic number the Step Characteristic (SC):

$$SC = \frac{kt}{\ln(10)} \text{ for growth (G) or inactivation (R)} \quad (4)$$

In which k is the specific growth rate or inactivation rate and t is the time.

Secondly a Contamination Characteristic (CC) can be defined:

$$CC = \log \left(\frac{N_{in} + R_c}{N_{in}} \right) \text{ for (re)contamination (C)} \quad (5)$$

in which N_{in} is the numbers entering the stage and R_c is the (re)contamination rate.

It should be noted that SC is only “condition” dependant, i.e. the effect of a heat treatment remains the same whether the initial level of microorganisms is 10^3 organisms/g or 1 organism/g, e.g. a 6D reduction. Therefore growth and inactivation are “additive” on a logarithmic scale. CC on the contrary is also state dependant, depending on the number of entering microorganisms. Contamination is “additive” on a linear scale and not on a logarithmic scale. An example of the quantification of characteristic numbers is given in Fig. 4.

5. Difference between growth/inactivation and (re)contamination

As noted above, SC (or $\sum G, \sum R$) is only condition dependant and the order of the increases or decreases is not of relevance. If growth and inactivation processes are considered to follow first order kinetics, it is possible to express a process without recontamination as

$$N = N_0 \cdot \exp(k_1 t) \cdot \exp(k_2 t) \cdot \exp(k_3 t) \cdot \exp(k_4 t) \dots \quad (6)$$

with k the specific growth or inactivation rate, depending on the actual conditions in the stage.

On a log scale these kinetics become additive:

$$\begin{aligned} \log(N) &= \log(N_0) + \frac{k_1 t}{\ln(10)} + \frac{k_2 t}{\ln(10)} + \frac{k_3 t}{\ln(10)} + \frac{k_4 t}{\ln(10)} \\ &= H_0 + SC_1 + SC_2 + SC_3 + SC_4 \end{aligned} \quad (7)$$

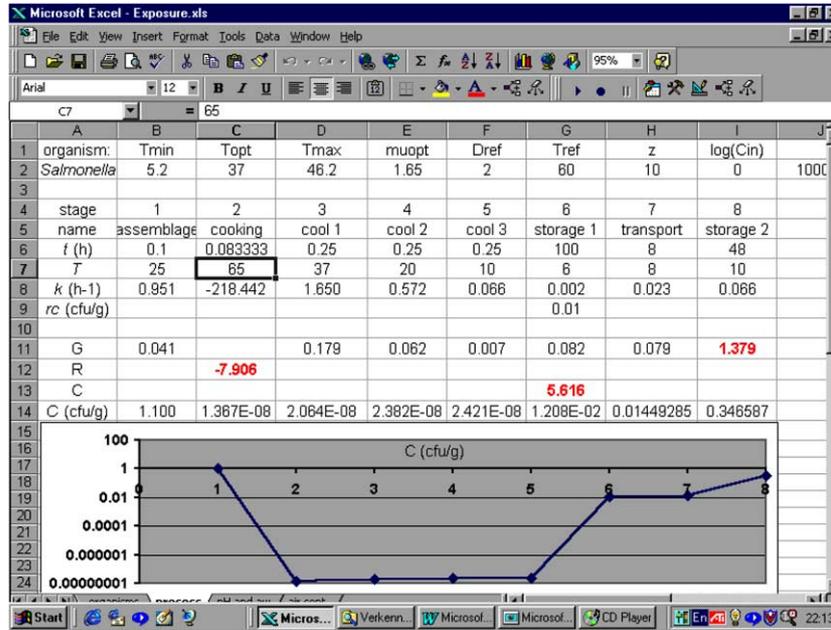


Fig. 4. Quantification of $\sum G$, $\sum R$, and $\sum C$ in an example process line.

If for example SC_2 is an inactivation, and the other 3 growth, $\sum G = SC_1 + SC_3 + SC_4$ and $\sum R = SC_2$. In principle the outcome will be equal if process steps are interchanged. It does not matter if first a 4 log growth and then a 6 log reduction takes place, or first a 6 log reduction and then 4 log growth, the result will in any case be an overall 2 log reduction. This can also be seen from the fact that in Eq. (4) the effect is only dependant on k and not on the actual level.

There are three exceptions:

- (1) If within growth the stationary phase is reached, but this is generally not the case for pathogens (and should not be).
- (2) If the number of organisms in a product unit becomes smaller than 1. Even in that case for large numbers of product units and proportional dose response relations without threshold, this does not have an overall effect on the outcome of the risk estimate.
- (3) History effects may make a dependence between stages.

On the other hand, contamination is additive on a linear scale but not on a logarithmic scale. This results in the fact that CC (or $\sum C$) is state dependant. For a case where in all stages of the process both growth or inactivation and contamination can take place one gets:

$$N = (((N_0 + R_{c1}) \cdot \exp(k_1t) + R_{c2}) \cdot \exp(k_2t) + R_{c3}) \cdot \exp(k_3t) + R_{c4}) \cdot \exp(k_4t) \dots \quad (8)$$

In this case the final effect can be totally different in case contamination occurs at stage 1, 2, 3 or 4 (for example

before or after pasteurisation). This can also be seen from Eq. (5) where the characteristic number depends on the recontamination level (R_c) and on the actual state (N_{in}). A recontamination with 10 cells per gram is much more important if the actual concentration is 1 cfu/g than if it is already 100 cfu/g.

6. Overall picture

Finally the characteristic numbers (SC , CC or $\sum G$, $\sum R$, $\sum C$) of all process steps of all parts of the chain are combined (Fig. 5) with the initial number ($N_0 = 10^{Ho}$) to determine the concentration at consumption (N_i). This allows defining the exposure (dose = concentration * serving size), which is translated, with the dose–response relation, into a probability of illness or death based on one serving, the risk per serving (RpS). This probability is multiplied with the total number of servings per year per million people, resulting in the probability of one case or the number of cases per year per

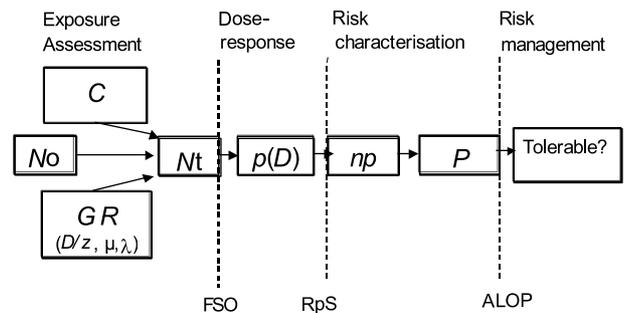


Fig. 5. Overall analysis of the connection between exposure assessment, dose–response, risk characterisation and management.

million people. It is then up to risk managers to evaluate this value and to decide whether it is tolerable/appropriate.

In this, Eq. (3) is used: $10^{\text{FSO}} \cdot M \cdot S \cdot 1\text{E}6 \cdot r = \text{ALOP}$, but with $D = 10^{\text{FSO}} \cdot M$, the dose; $n = S \cdot 1\text{E}6$, the total numbers of servings per year per million people, $p(D) = rD$, the probability given dose D , this can also be written as

$$D \cdot n \cdot r = \text{ALOP} \quad (9)$$

If the prevalence is lower than 100% this means that there is a probability that the concentration or dose is present in the product unit, but there are also units without any pathogen. In such a case the equation to be used is

$$D \cdot P \cdot n \cdot r = M \cdot C \cdot P \cdot n \cdot r = M \cdot 10^{\text{FSO}} \cdot n \cdot r = \text{ALOP} \quad (10)$$

with $10^{\text{FSO}} = C \cdot P$, so $\text{FSO} = \log(C \cdot P) = \log(C) + \log(P)$.

7. Distributions of exposures

In many cases prevalences of various levels are known. In these cases the highest concentrations are usually the ones determining the main number of cases of illness. In these cases the contribution of the various concentration ranges can be added:

$$M \cdot \sum_i (C_i \cdot P_i) \cdot n \cdot r = \text{ALOP} \quad (11)$$

If we have, for instance:

- (1) 57% no *L. monocytogenes*,
- (2) 30% around 10/g *L. monocytogenes*,
- (3) 10% around 100/g *L. monocytogenes*,
- (4) 3% around 1000/g *L. monocytogenes*.

The number of people getting ill from eating this product per 1 million product units of 100 g (assuming that 20% of the people is in risk groups, and that for these risk groups the r of the exponential dose response relation equals $1.2\text{E}-10$) results in:

- (1) 0 cases,
- (2) $M \cdot C \cdot P \cdot n \cdot r = 100 \cdot 10 \cdot 0.3 \cdot 1\text{E}6 \cdot 0.2 \cdot 1.2\text{E}-10 = 0.0072$,
- (3) $M \cdot C \cdot P \cdot n \cdot r = 100 \cdot 100 \cdot 0.1 \cdot 1\text{E}6 \cdot 0.2 \cdot 1.2\text{E}-10 = 0.024$,
- (4) $M \cdot C \cdot P \cdot n \cdot r = 100 \cdot 1000 \cdot 0.03 \cdot 1\text{E}6 \cdot 0.2 \cdot 1.2\text{E}-10 = 0.072$.

The total is therefore 0.103 cases per million servings. The highest concentration range gives the largest contribution (70%), albeit the low prevalence. If the contamination of this 3% could be prevented, the health burden

would be reduced by a factor 3.3 in this hypothetical example. Note that in this example the endpoint of the case is illness (mortality is not taken into account) and that it is expressed in per million servings, and not per million people per year.

With, for instance, 50 servings per year per person, this would result in 5.2 cases of illness per year, and assuming 30% mortality, 1.5 deaths per million per year. The number 0.103 (illnesses per 1E6 servings) seems to be lower than in the earlier example (1 death per million per year), but it is not.

Maybe in reality there are also products at concentrations of 10,000/g, but that go undetected. And if these are present in more than 0.3% of the cases they are the most relevant (and the probability of detecting this 0.3% if present, would even with 60 samples only be 16%).

8. Combination of prevalence, and the statistical distribution of the concentration into an FSO

If an FSO is set at 2 (100 cfu/g) or -2 (1 cfu/100 g), one can achieve this goal by controlling the average level, the spread of the distribution and the prevalence. If one has the simple case of a log normal distribution of organisms in a product, which is often a good approximation, one can determine the mean concentration by

$$\log(\bar{C}) = \overline{\log(C)} + 0.5\sigma_{\log}^2 \cdot \ln(10) \quad (12)$$

Note that the log of the mean concentration ($\log(\bar{C})$) is larger than the mean log concentration ($\overline{\log(C)}$).

Together with the prevalence P (presence/absence) one gets:

$$(\overline{\log(C)} + 0.5\sigma_{\log}^2 \cdot \ln(10)) + \log(P) = \text{FSO} \quad (13)$$

So if the FSO is set at 2 (100/g) equal results can be obtained (Table 1) for example with an exact concentration of 100/g, with prevalence 100%, or by a mean $\log(C)$ of 0.85 with a standard deviation of 1 (so $\log C = \text{normal}(0.85, 1)$) and a prevalence 100%. This again results in the same as a mean log concentration of 2, standard deviation of 1 and a prevalence of 7%. These calculations only hold if the range of doses are in the linear part of the dose–response relation, that is for doses where for the exponential model $1 - \exp(-D \cdot r) \approx D \cdot r$ (as long as $D \cdot r < 0.1$, the error is below 5%).

The equation also holds for lower, and even unmeasurable FSOs. For example if absence in 100 g is set as level, $\text{FSO} = -2$ (Table 2). The same equations can be used resulting in the same phenomena as Table 1.

9. Definitions

Another point is the harmonisation of definitions. In the food safety literature various definitions do exist.

Table 1
Various mean log concentrations, standard deviations and prevalences, resulting in the achievement of the same FSO

Mean log(C)	σ	P	FSO
2	0	1	2
1.93	0.25	1	2
1.71	0.5	1	2
0.85	1	1	2
2	0.25	0.847	2
2	0.5	0.515	2
2	1	0.071	2
3	0	0.1	2
3	0.25	0.085	2
3	0.5	0.052	2
3	1	0.0071	2

For example, ALOP (Appropriate Level of Protection), TLR (Tolerable Level of Risk), ALR (Acceptable Level of Risk) are all used to express the same public health goal. It is important to select only one term to avoid confusion. Level of protection has a more positive aspect since it uses protection and not risk, but on the other hand tolerable shows a more dynamic behaviour than the word acceptable. Recently, CODEX (Codex Alimentarius Commission, 2004) selected the term ALOP. Secondly the FSO is defined at the point of consumption. This is important because when the FSO is moved away from the point of consumption, it becomes less related to the public health objective and interventions after the point at which the limit is set can have an impact on public health. Also for performance objectives and performance criteria, their exact definition have been decided on (Codex Alimentarius Commission, 2004):

Food safety objective (FSO): The maximum frequency and/or concentration of a hazard in a food at the time of consumption that provides or contributes to the appropriate level of protection (ALOP).

Performance Objective (PO): The maximum frequency and/or concentration of a hazard in a food at a specified step in the food chain before the time of con-

sumption that provides or contributes to an FSO or ALOP, as applicable.

Performance Criterion (PC): The effect in frequency and/or concentration of a hazard in a food that must be achieved by the application of one or more control measures to provide or contribute to a PO or an FSO (Codex Alimentarius Commission, 2004).

10. Conclusions

- The FSO concept is very strong in that it makes food safety transparent and quantifiable.
- This brings a major advantage in that one can control safety in that part where it is the most efficient but keep to an integrated objective.
- Correct use and reporting of units is of great importance.
- Characteristic numbers can supply the necessary quantification of the various parts of the equation.

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Table 2
Various mean log concentrations, standard deviations and prevalences, resulting in the achievement of the same FSO

Mean log(C)	σ	P	FSO
–2	0	1	–2
–2.07	0.25	1	–2
–2.29	0.5	1	–2
–3.15	1	1	–2
–2	0.25	0.847	–2
–2	0.5	0.515	–2
–2	1	0.071	–2
–1	0	0.100	–2
–1	0.25	0.085	–2
–1	0.5	0.052	–2
–1	1	0.0071	–2

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