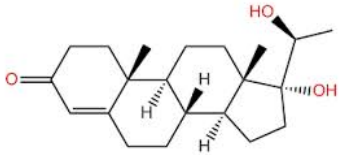
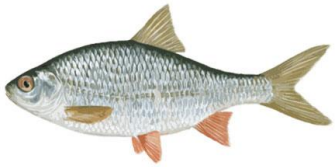
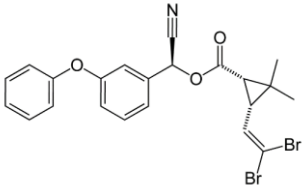
HOW SHOULD WE ASSEMBLE EVIDENCE TO DECIDE WHETHER OUR ENVIRONMENT IS BEING DAMAGED BY CHEMICALS?

Andrew Johnson







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Duties of those working on chemicals in the environment

A. Ensuring that the characteristics of new chemicals and likely exposure levels would not harm the environment (prospective risk assessment)

B. To assure ourselves that the environment is not being damaged by the chemicals in current use (retrospective risk or impact assessment)

Duty A is the constant concern of industry, academics and regulators, however, this vital topic is not the subject of this talk.

Duty B largely falls on environmental scientists informing policy makers, which is the topic we deal with here.

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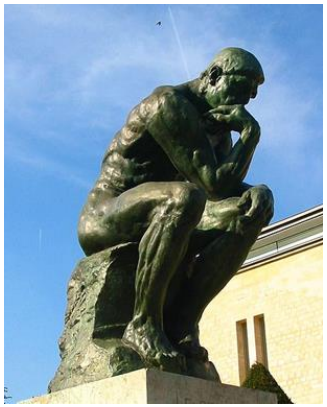
The philosophical bit

Can we ever be certain?

- Many authors warn against the desire for proof and certainty when trying to discuss the subject of causation
- In the philosophy of science from Popper, only falsehoods can be disproved whilst those theories which survive multiple tests may be accepted as merely provisional.
- As Hill (1965) would say, ‘all scientific work is unfinished’ and so knowledge can never be complete
- Thus, attempting to demonstrate causation is an activity of building our confidence in the likelihood of a cause



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Lack of certainty has been used as a ‘get out clause’ in the past

Gilbertson(1997) recounts examples where industry and authority chose not to respond due to lack of certainty over the causative agent:

- Scepticism from US and Canadian Governments over acid rain and loss of trout in upland lakes in the 1980s
- Great Lakes Water Quality board scepticism over which chemicals and which sources were responsible for decline of wildlife in the lakes
- Delays by Industry to health restrictions on smoking due to demands that the causative chemicals responsible for lung cancer be specifically identified

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Gilbertson M. Advances in forensic toxicology for establishing causality between Great Lakes epizootics and specific persistent toxic chemicals. Environmental Toxicology and Chemistry 1997; 16: 1771-1778.

But we can take action without certainty over the causative agent if we know the source

- Dr Snow removing the handle of a drinking water pump stopped his local cholera outbreak in 1854 (contradicting the foul air hypothesis)

- The example of chimney sweeps being 200x more likely to suffer from scrotal cancer (R. Doll, 1964)
- Smokers suffering 10 to 30x the incidence of lung cancer of non-smokers (A.B. Hill, 1965)

In all these cases we did not know the precise agent, but the source of the problem was demonstrated

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The option of applying the Precautionary Principle exists

Where some evidence exists for a chemical or chemicals to cause significant harm, we should act early to control or reduce that risk

But what evidence do we need, will anything do?

How do we typically build a case?

+

+

= **Overwhelming evidence?**

Lots of these studies

Quite a bit of these measurements

One or two examples of these

This is the typical assembling of evidence we might use in Europe in building a case against a chemical or chemicals

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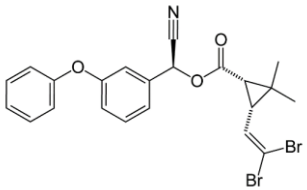
There are some different approaches to researching chemicals issues in the environment (not all start with the chemical)



Unique location concern

What is killing wildlife in my lake?

Retrospective

Will use mixed evidence but long-term wildlife monitoring essential

Specific chemical(s) of concern

Maybe this chemical is bad for wildlife?

Prospective or retrospective

Laboratory ecotoxicity led alongside chemical monitoring of the environment

Unique wildlife failure

Why are my vultures dying?

Retrospective

Field evidence with forensic ecotoxicology

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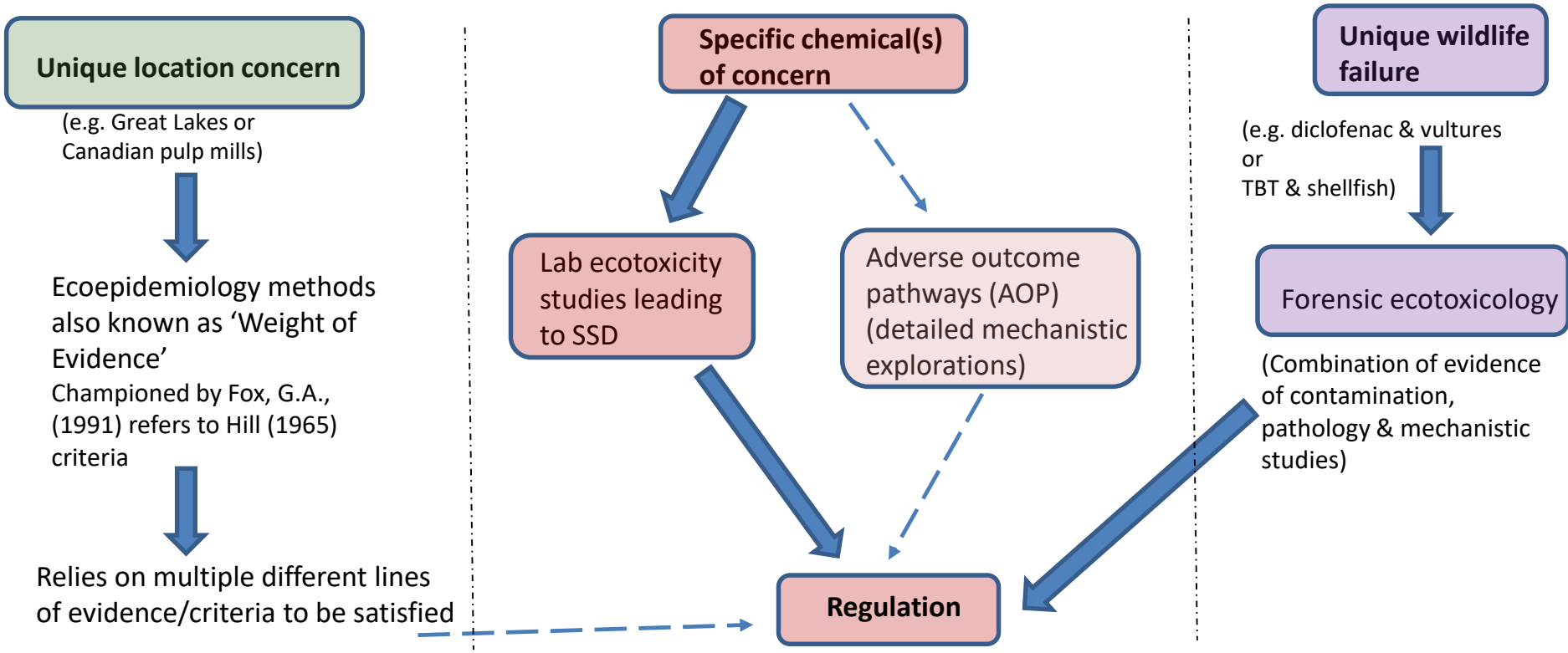
The language of researching chemical issues in the environment



Wildlife population impact is key part of the investigation

Wildlife population impacts not known or considered necessarily relevant

Wildlife population impact is central to the investigation

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The ability of laboratory ecotoxicity studies to predict what happens to an organism in the field may be limited…..

Might under-predict risks in the environment due to the actual presence of multiple stressors
Or over-predict risks due to lack of ecological realism of the study

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Adams S.M. Establishing causality between environmental stressors and effects on aquatic ecosystems. Human and Ecological Risk Assessment 2003; 9: 17-35.

Re-appraising Ecoepidemiology (weight of evidence)

Could that provide the structure we need to make those decisions?

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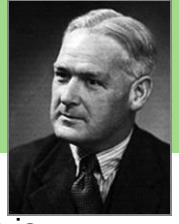
Austin Bradford Hill and his 1965 criteria



- **Strength**: this might be described as where the incidence of a disease (harmful population effect) and exposure is dramatic. He gives the examples of the incidence of lung cancer in smokers being 10 to 30 x that of non-smokers. So the differences here are eye-catching.
- **Consistency:** has the effect been observed to fit the same pattern by different people in different places, circumstances and times?
- **Specificity:** here damage is linked to the most exposed and vulnerable part of the population. Bradford Hill was considering unique exposures, such as for people working in a particular industry having adverse problems to a degree not seen in those in other environments.
- **Temporality:** Here harm is linked to a moment in time when exposure to a harmful agent started (and similarly would decline in time once the agent is withdrawn).
- **Biological gradient:** This is the well-known dose-response relationship and in our case greater exposure might reasonably be expected to cause more damage.
- **Plausibility:** This is where the relationship is biologically plausible. This is an interesting one as Bradford Hill said we cannot demand it (as biological knowledge may be different tomorrow from what it is today)
- **Coherence:** This is where different sources of evidence are brought together to complement the argument. Thus, linking the deaths to cholera to drinking water from a contaminated source is strengthened by also detecting Vibrio bacteria in the water.
- **Experiment:** In this case Bradford Hill appears to be asking does an intervention, such as reducing exposure through changing industrial practice, reduce the incidence of the linked disease?
- **Analogy:** Bradford Hill suggests that we might be able to refer to similar agents (perhaps in molecular structure) which we know have caused serious problems in the past to warn us of danger.

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Hill, A. B., The environment and disease: association or causation? Proc. R. Soc. Med. ***1965,*** *58, 295-300.*

Origins and use of epidemiological criteria in the sphere of chemicals in the environment

Description	Epidemiology	Epidemiology	Eco-epidemiology	Epidemiological criteria (Forensic ecotoxicology	Weight of Evidence	Lines of evidence (USEPA)	Forensic ecotoxicology		Ecological tiered assessment of risks
Topic	Cause of disease	Cause of disease			Is a chemical group causing any biological impairment	Cause of biological impairment	Environmental toxicology	Establishing causality	Supporting local or regional risk assessments
Reference	(Hill, 1965)	(Susser, 1986)	(Fox, 1991)	(Gilbertson, 1997)	(Ankley and Giesy, 1998)	(Cormier et al., 2003)	(Collier, 2003)	(Adams, 2003)	(Moraes and Molander, 2004)
1	Strength	Strength of association (greater the exposure the greater the effect and vice versa)	Strength of association	Strength of association	Adverse effects in populations		Strength of association	Strength of association	Strength of association
2	consistency	Consistency (across time, place and people we see the same outcome if exposure the same)	Consistency	Consistency in replication		Spatial fidelity	Consistency of association	Consistency of association	Consistency of association
3	Specificity	Specificity (ideally the outcome is uniquely linked to one cause eg polio and polio virus)	Specificity	Specificity		Specificity	Specificity of association	Specificity of association	Specificity of association
4	Temporality	Time order (chronology)	Time order (chronology)	Time order (chronology)	Recovery of populations when contaminant removed	Temporality	Time order/ temporality	Time order/ temporality	Time order/ temporality
5	Biological gradient	Susser puts this under coherence	Dose response relationship (coherence)			Biological gradient	Biological gradient	Biological gradient	Biological gradient
6	Plausibility	Susser puts this also under coherence	Biological plausibility (coherence)		Plausible mechanism	Plausible mechanism	Biological plausibility	Biological plausibility	Biological plausibility
7	Coherence	See above		Coherence	Coherence between observations	Coherence amongst			

The US EPA has formally adopted a form of Ecoepidemiology (weight of evidence) to find causation in locations of environmental damage

Why are they so keen and what does it involve?

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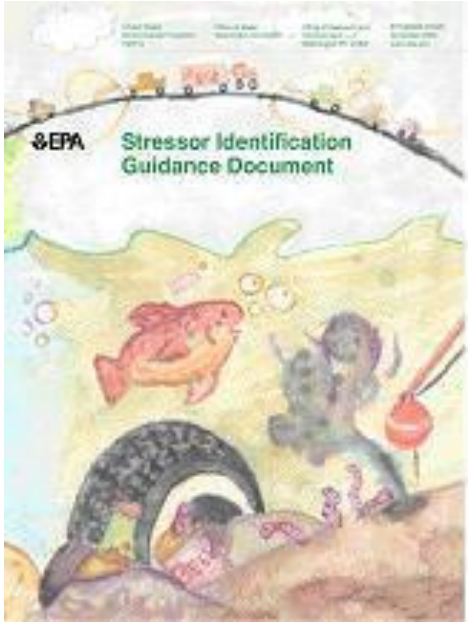
Why the US EPA thinks we need a formal structure to evidence gathering

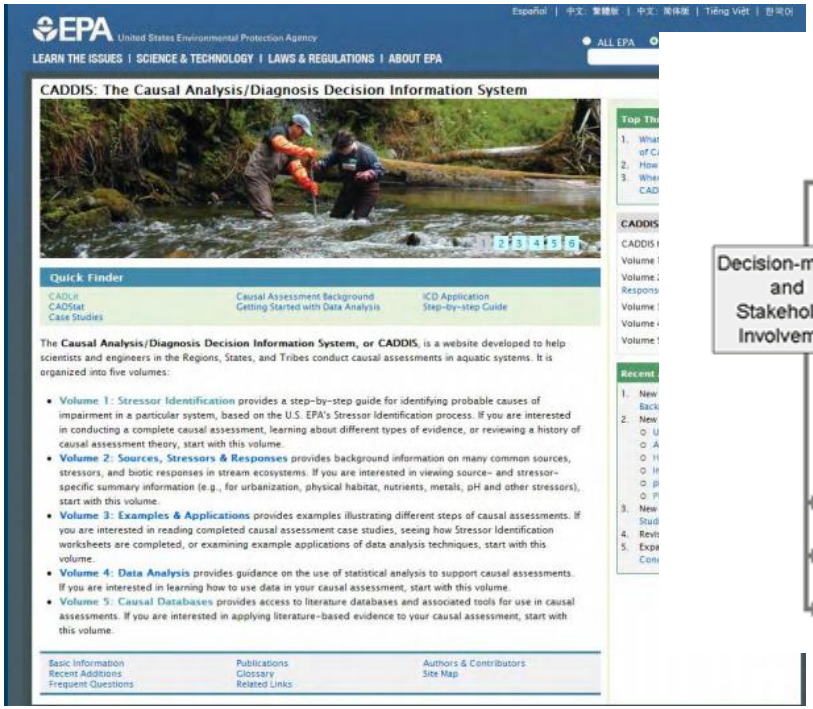
Why use a formal method?

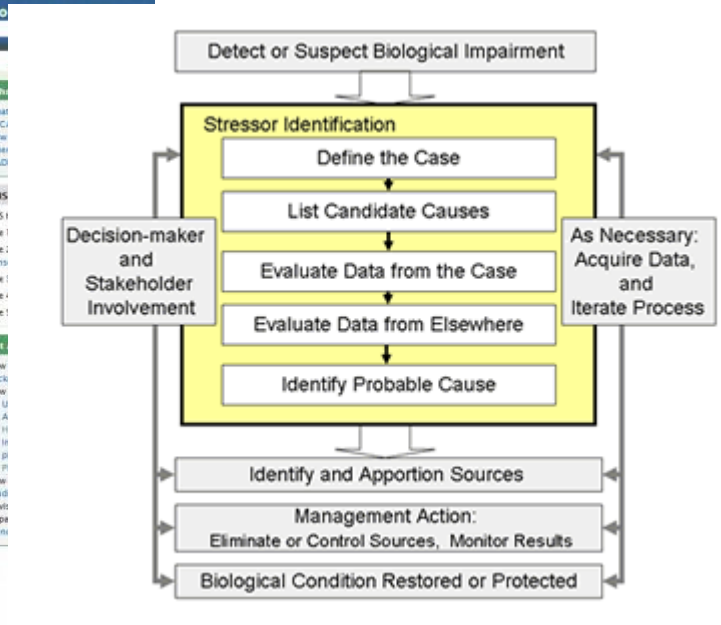
- To convince stakeholders
- To increase confidence that remedial or restoration efforts can improve biological condition
- To identify causal relationships that are not immediately apparent
- To prevent biases and other lapses of logic

"Science is a way of trying not to fool yourself. The first principle is that you must not fool yourself – and you are the easiest person to fool." [Feynman 1964]

The US EPA has formalised an approach to helping to establish causation 'caddis'







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http://www.epa.gov/caddis

The US EPA system was set up largely with the aim of establishing local causation

It is admirable in proposing the assembly of evidence using a framework

Keen to avoid presumptions on causation

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We can take the best from this to tackle chemical questions in the UK….

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A suggested evidence sequence

Observational/ecology

Observe a wildlife impact (consistent examples of population(s) in trouble)

Association (Are the examples of population(s) in trouble associated with putative chemical exposure)

Relationship (Are the population(s) most in trouble associated with highest putative chemical exposure)

Trends* (Are the population(s) continuing to decline over time)

Consistency (have these observations of harm been reported by many different people in different places)

Temporality (wildlife damage coincides when exposure to the harmful agent began and similarly impacts reduced when exposure reduced)

Trends* are not usually discussed in epidemiology

Chemistry

Contamination (wildlife in trouble have the chemical in their bodies

Coherence (wildlife issues are coherent with laboratory derived evidence including dose/response and mechanism

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But it won’t work if we ditch monitoring!

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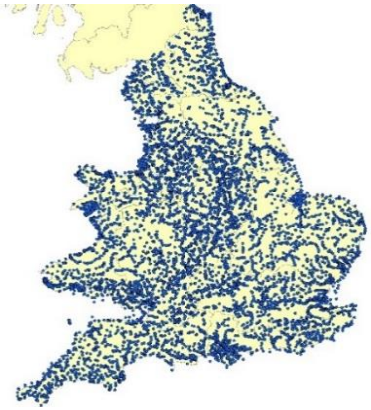
The UK is blessed with an excellent record

There are some 15,000 freshwater locations where macroinvertebrates and 3,500 locations where fish have been recorded over the past 10-30 years. This provides us with Millions of records!

BUT…..



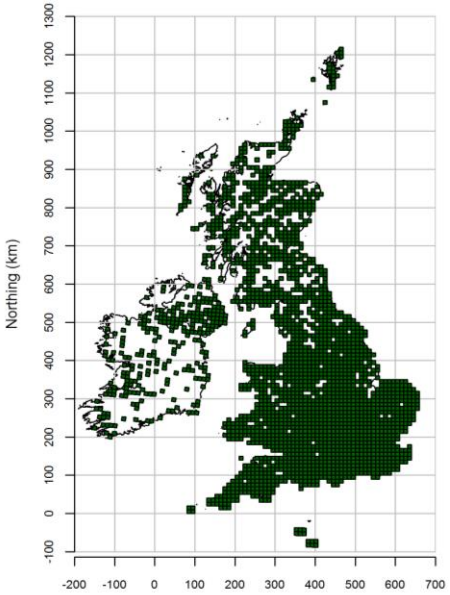
Location of macro-invertebrate long-term monitoring sites

Of these, there about 2000 macroinvertebrate sites and about 1500 fish (NFPD) have an unbroken and continuous 25 year monitoring record. **These are the crown jewels!**

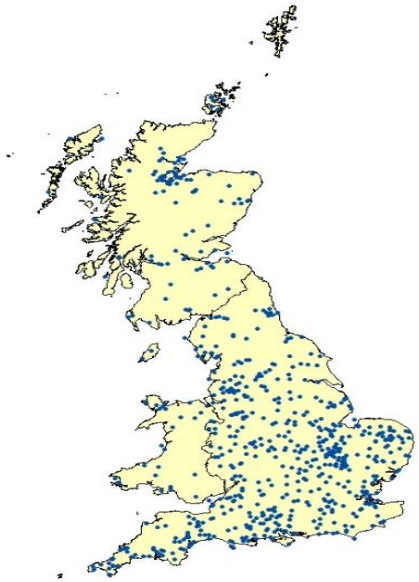
However, the EA planned SENTINEL network envisages an integrated network of 500 sites of which **only 100** will receive continuous monitoring. The site selection is weighted in favour of geographic spread, meaning 1st and 2nd order streams will be strongly represented (not necessarily lowland rivers which face the big human pressures)

TERRESTRIAL WILDLIFE RECORDS ALSO COVER A WIDE AREA AND GO BACK DECADES

Including..
Predatory beetles (natural pest control)
Arable area spiders (natural pest control)
Hover flies (pollination & natural pest control)
Butterflies & moths (pollination & cultural services)



Sparrowhawk



But these are dependent on recording by amateur naturalists and held at the Biological Records Centre

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Why we should have our own weight of evidence/ecoepidemiology criteria in the UK……

- There will always be ‘left field’ chemicals and substances which will catch us out where the public might demand dramatic action
- Having an established requirement of an evidence criteria/structure being satisfied could really help scientists, regulators and policy makers respond proportionately
- Not so much how much evidence you have, more about bringing together multiple lines of different evidence to provide a coherent picture

Why has ecoepidemiology not been taken up in Europe/fallen our of favour?

- Those working in the field tend to be specialists either in ecotoxicology or environmental chemistry
- Strong relationships with ecologists don't exist and retrospective analysis of monitoring undertaken by 3rd parties doesn't seem novel or exciting to research funders.
- Tradition and the WFD has funnelled the thinking of regulators around laboratory ecotoxicity, SSDs and risk quotients. Evidence of chemical impacts on populations is not needed by the EA
- The terms ecoepidemiology and weight of evidence cause confusion
- **Using a formal structure for evidence gathering, centring on wildlife monitoring, to support our analysis of chemical challenges has much to recommend it!**

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How is this different from what we do now?

- We tend to propose an agent as being harmful and then we scrabble around to find evidence later to support this e.g. microplastics
- The alternative approach is to examine our environment and in particular the health of wild populations
- We ramp up our investigations where harm has been identified
- We would include chemicals as one of the stressors