CONTEXT, CAUSALITY AND CONSEQUENCES
From robust evidence to timely action in biology, ecology, law and public policy

Friday 26 and Saturday 27 November 2021
Bern and online

Registration:
Online participation:
https://us02web.zoom.us/webinar/register/WN_X9hhCNv9QLCFACw8-QiGGA

Please contact us for onsite participation: office@ensser.org

"In what circumstances can we pass from this observed association to a verdict of causation?"
"The 'cause' of illness may be immediate and direct, it may be remote and indirect underlying the observed association."
"…the decisive question is whether the frequency of the undesirable event B will be influenced by a change in the environmental feature A."
"What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?"
"In occupational medicine our object is usually to take action. (...) we shall wish to intervene to abolish or reduce death or disease."
"All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time."

A. Bradford Hill: "The Environment and Disease: Association or Causation?",
Proc. Royal Soc. Med., p. 295 – 300, 1965,
https://doi.org/10.1177/003591576505800503

Keywords: agnotology, ambiguity, causality, cause, co-causal, control, determination, direct cause, effect, evidence, hubris, ignorance, indeterminacy, indeterminism, indirect causes, multi-causality, non-target effect, precaution, Precautionary Principle, precision, probability, risk assessment, target effect, uncertainty

Content of the conference

The understanding of "causes" is at the core of scientific investigations. It underlies all current efforts to face ecosystemic challenges. Physics has framed causality within robust and unifying "conservation principles" (energy, momentum etc.). But how can we understand and verify supposed causal relations within the historicity and diversity of life in a unified manner? How can we scientifically describe the effects which appear to be caused by a chemical or physical event on an ecosystem governed by reproduction and variation and on environments enabling diversity?

A special influence on our notions of causality in biology and ecology is exerted by our advanced reliance on automation, digitization, computers, in-silico models, computational surrogates, artificial intelligence and machine learning. If life-science phenomena are viewed as caused by one or a few selected agents, how does this view align with what can be obtained

from a computerized output, with less and less reliance on hands-on experience and human senses for verification?

A paradoxical situation seems to emerge, where this approach appears to enable to flip cause and effect, as exemplified in synthetic biology. Traditionally, say, cloning relied on the appropriate functioning of an entire framework system (environment), such as E. coli, to express a specific gene insert. With the advent of reverse genetics, the same technique is now publicized as being "under the control of the gene" to be expressed. It seems that modeling, and focusing on what one wants to obtain, has shifted the comprehension and understanding of what is cause and effect, demonstrating that the entire framework of causality is not only poorly understood, but little investigated.

When approaching this question from another level, one may ask what the biological influencers of causality are. Is it about material entities (e.g. genes), or also about interactions, relationships, and context (from sub-cellular to ecological)? A unique ability of life is to be able to adapt to a changing environment. So one may ask: what causes habituation and learning, and in which way do these influence the appearance of causality (in gene expression, disease and other phenomena)?

"Science is the organised attempt to discover how things work as causal systems."[11] But in current biology and ecology every cause has multiple effects and every effect has multiple causes. So at this conference we study notions of multi-causality within complex systems that encompass many variations, in time and space, of the "known" co-causal factors, whilst acknowledging the existence of many other unknown, yet possibly influential variables.

There are some current attempts to re-introduce simple causality, for example, in searches for the particular gene or environmental circumstances that are relevant to a specific biological trait, via genetic modification which is claimed to be "*precise*".

However, other approaches are attempting to identify modifiable co-causal factors within context and time dependent complex biological and ecological systems, factors which enable both life and harms to emerge and which, if removed or reduced in a timely manner, would result in less harm to people and environments.

Such approaches to identifying robust causal inferences in biology, ecology, tort law and public policy on health and environment eschew simplistic dichotomies (e.g. nature/nurture; gene/environment; acute/chronic; effects/adverse effects; statistically significant/insignificant effects; association/causation; valid/invalid arguments) in favour of continuums, or spectrums, along which choices need to be made as to where and when positions on such continuums justify robust causal inferences.

Current approaches to coaxing causality from complexity also include systematic review methodologies; systematic approaches to integrating different evidence streams; and "*inferences to the best explanation*" used in tort law. There are also approaches that render explicit and transparent the often implicit sources of divergent evaluations of the "same" evidence, which can be illustrated historically with: leaded petrol; low dose ionising radiations; and antibiotics as animal growth promoters; and currently with: per- and polyfluoroalkyl substances (PFAS); glyphosate; neonicotinoid insecticides; and electromagnetic fields (EMFs).

These sources of divergent evaluations include (1) different types of paradigms, assumptions and argumentation used; (2) the diverse disciplinary conventions of toxicologists, endocrinologists, epidemiologists, biologists, ecologists, and lawyers; and (3) their value based choices.

Public and stakeholder involvement in helping to identify robust causal inferences for public policy interventions have also been used, sometimes including communities affected by harms. The value of these approaches will be reviewed.

1 Waddington, C.H. (1943), "The Scientific Attitude", second revised edition, Penguin

Programme

Friday 26 November 2021

8:30 Registration
9:00 Welcome / Introduction – Diederick Sprangers, ENSSER

9:30 **Prof. Giuseppe Longo**, Centre Cavaillès (République des Savoirs), CNRS and Ecole Normale Supérieure, Paris
Causality and novelty production in biology

10:15 Discussion
11:00 Coffee break

11:30 **Prof. Denis Noble**, University of Oxford
20th century biology got causation in living systems the wrong way round

12:15 Discussion
13:00 Lunch

Moderator: Prof. Giuseppe Longo

14:30 **Dr. Siguna Müller**, Kärnten, Austria
Causality and SARS-CoV-2 – new findings that mandate a broader perspective

15:15 Discussion
16:00 Tea break

16:30 **Dr. Thierry Paul**, National Centre for Scientific Research (CNRS) and Sorbonne University, Paris
Causality, indeterminism and all that: a quantum entanglement

17:15 Discussion
18:00 End

Saturday 27 November 2021

Moderator: Dr. Ricarda Steinbrecher, EcoNexus, Oxford

9:00 **Dr. Maël Montévil**, French National Centre for Scientific Research (CNRS)
Organization, historicity and causality

9:45 Discussion
10:30 Coffee break

11:00 **Dr. Elena Rocca**, Centre for Applied Philosophy of Science, Norwegian University of Life Sciences
Difference-maker, mechanism or disposition? How explicating and critically discussing basic assumptions about causality improves scientific evaluations of risk

11:45 Discussion
12:30 Lunch

Moderator: Dr. Angelika Hilbeck, Swiss Federal Institute of Technology

14:00 **Prof. Ana Soto**, Tufts University School of Medicine (Boston), Centre Cavaillès, Ecole Normale Supérieure (Paris) and Institute for Advanced Studies of Nantes
Towards the understanding of biological causality and its application to endocrine disruption

14:45 Discussion
15:30 Tea break

16:00 **Prof. Thomas Vondriska**, David Geffen School of Medicine, University of California, Los Angeles
Genomes, epigenomes and causal inference in multicellularity and human disease

16:45 Discussion
17:30 Final discussion
18:00 Closing words: NN

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