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An Example from Oil Contamination.

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Abstract

Measurements of environmental toxicity from long-term exposure to oil contamination have delivered inaccurate and contradictory results regarding the potential harms for humans and ecosystems. This has led to a methodological discourse, in which orthodox approaches to risk assessment of oil toxicity are questioned. We argue that methodological stances towards risk assessment in cases of oil contamination hide deeper commitments to basic assumptions concerning the nature of causation. Explication and examination of such assumptions ought to be the starting point for a deeper methodological discussion, to identify the source of disagreement. This paper is an attempt to contribute to this groundwork.

Introduction: challenges and controversies in toxicology of oil contamination

After more than 50 years from the beginning of oil extracting activities, and despite of estimated 638 millions of people living close to oil facilities or contaminated spots in the developing countries, the health related risks of residents exposed to oil contamination remain uncertain (O'Callaghan-Gordo et al., 2016). Research on toxicity from exposure to oil contamination has delivered contradictory results, and it has encountered some challenging nodes of complexity. One problem is that affected areas are mostly in remote regions of developing countries. In such areas, there have been serious obstacles to carrying out population studies: scarcity of demographic information, lack of health registries with under-reporting of sickness and death and difficulty of getting precise data about exposure to contamination. Moreover, the task of investigating the long-term toxicity of a specific pollutant is complicated by the scarcity of health care and prevention, and probable exposure to several contaminants because of loose environmental regulation. The majority of the epidemiological studies carried out to investigate the exposure effects of oil contamination on residents, shows some acute symptoms and irritation similar to the symptoms previously described in oil workers. However, the incidence of long-term sickness, such as cancer, is inconclusive and the evidence contradictory (O'Callaghan-Gordo et al., 2016).

The situation has spawned debates about which scientific methodology (if any) can legitimately ground policy measures in this specific case of uncertainty. In a correspondence among environmental epidemiologists, Hurtig and Sebastian, supported by Terracini (2004), argued that evaluations of risk cannot "be reduced to a discussion of […] p-values and potential confounders"

(Hurtig and Sebastian, 2005: 1171). In order to infer risk from weak statistical studies in the contaminated oil basins of Ecuador, the authors invoked instead an "element of plausibility". Such elements could include the existence of a known mechanism of harm of oil contaminants, and consideration of some contextual circumstances, such as the high level of oil contamination in drinking water and heavy metal levels in the resident's blood. In contrast, Semyiatycki contests that epidemiology, being a rigorous science, can only infer risk by establishing reliable correlations, through an unbiased experimental design (Semyiatycki, 2002). He argues therefore that an epidemiologist can infer no risk regarding exposure to oil contamination from existent studies, because of the absence of proper statistically sound correlation data.

This is one of the debates among scientists in the last 30 years that demonstrate a split in methodological stand about how oil toxicity ought to be measured and understood. Sawyer and Loja (2015) highlight the indeterminacy of measurements of oil toxicity by pointing out how, in legal litigations between corporations and residents, different teams of experts inferred different levels of risk from similar measures of soil contamination. This is a phenomenon of big proportions. Globally, an increasing number of court cases take years and millions of dollars due to the ambiguity of evidence and methodological disagreement, which make it extremely difficult to prove harmful outcomes of extractive activities in a scientifically robust way (Li, 2015; Kirsch, 2014; Sawyer, 2004). The faith on a reliable, objective toxicological method, able to produce convincing evidence on the impacts of oil contamination in humans and environment, has been fading out in the last decades. In a 2003 *Science* review, for instance, Peterson et al. (2003) argue that, in light of studies on the ecological consequences of major oil spills, it is time to abandon

the orthodox approach to oil toxicology, based on chemical measurements, mathematical models and short-term exposure studies in lab animals.

Two observations are relevant for our purpose here. One is that scientists disagree over which methodologies are best suited to assess risk in cases of oil contamination. The other is that different methodologies provide different results. We will show how a split in methodological stance towards risk assessment can be motivated by disagreement over basic assumptions concerning ontology and the nature of causation and complexity. Our take here is that a fructuous methodological discussion ought to include an explication and examination of this fundamental disagreement.

A disagreement over basic assumptions

How can the same evidence possibly lead to different risk evaluations if the process of assessment is scientifically sound? One answer, which we support here, is that it can do so if the two evaluations are made under different basic scientific assumptions (Douglas, 2000; Rocca and Andersen, 2017). More specifically, we argue that different ontological intuitions about the nature of things result in different priorities about the most reliable methodologies.

Our argument is supported by recent literature proposing that the problem of evidencing potential harm from environmental contamination is not only an empirical one (Douglas, 2000; Longino, 1990; Hermasson, 2012). Sawyer and Loja (2015), for instance, gives a lucid diagnosis of the importance of extra-evidential elements for evaluation of oil toxicity. "Toxicity is far from natural", they write. Instead, it has often been "an imbricated technical, chemical and legal work that allowed toxins to matter, or not" (Sawyer and Loja 2015: 129). Within this discourse, risk assessment is not seen as merely a matter of measurements and methodology. Yet, protest some authors, the role of social dynamics, value judgements, basic philosophical assumptions, and the way in which they relate to each other, are not subject to public or governmental scrutiny (Hartley et al., 2016; Wickson & Wynne, 2012).

Starting from this position, we aim to contribute by clarifying the role of philosophical assumptions, particularly concerning the nature of causation. Although there is extensive literature about how socio-economic interests and value judgements drive risk evaluations, little has been said about the identity and role of philosophical basic assumptions in specific cases of scientific disagreement on environmental risk (Rocca and Andersen, 2017). We take this to be a dangerous omission, since basic assumptions are acknowledged as the door through which extra-scientific values make their way into the internal processes of science (Longino, 1990; Douglas, 2000). In the case of harm detection from oil contamination, which is heavily entangled with socio-economic interests, this issue is particularly relevant. Ours is mainly a philosophical argument, therefore, but one that has support from science, as we aim to show in the following.

One complication concerning our topic of interest is that basic philosophical intuitions are often adopted tacitly and uncritically, along with general methodology, evidence evaluations, and rules of inference that they generate (Kuhn, 1962). In effect, scientists who acquire different basic assumptions, for instance from their educational or professional background, might disagree on the overall rules of scientific inquiry as well as its content, without being aware of the fundamental foundations of such disagreement (see for instance Kvakkestad et al., 2007; Stoltz et al., 2004). If we accept that scientific disagreement is rooted beyond simple analysis of notions and evidence, and that philosophical basic assumptions are not always chosen with awareness, we are left with the challenge of correctly identifying different philosophical premises behind conflicting scientific methodologies.

In the following, we will look at some basic philosophical assumptions in a case concerning toxicology of oil contamination, with a particular focus on the notion of causation. For this, we first need to describe two different scientific approaches to risk assessment of oil contamination. The first approach has been in use since the early '90s, but big contamination events, such as oil spills, showed that its capacity of predicting toxicity to living organism might fall short. The second has been suggested as an alternative[1].

Disclosing oil toxicity: two different scientific approaches

[1] For an excellent analysis of the value-choices and socio-economic interests underlying this case, see Sawyer and Loja (2015).

The task of characterizing the toxicity of oil residues for living organisms is particularly challenging, because petroleum is not a single chemical. Indeed, it is a complex composition of different types of chemicals, mainly hydrocarbons, which interact differently with each other and with diverse environments. This is why oil is usually designated with the umbrella definition of 'total petroleum hydrocarbon' (TPS). Equal amounts of TPS, therefore, might have different composition and interactions. We describe here two different scientific approaches, which have been used to study this complex system.

The first approach is based on the postulation that the chemical composition of the specific TPS residue, and a series of initial environmental conditions of the polluted site, will determine to a big extent how the chemicals are going to behave. This would make it possible to predict with an acceptable degree of confidence which of the hydrocarbons will reach living organisms, and in which amount. The second approach, instead, is skeptical of the predictive capacity of closed systems and mathematical calculations, and professes that toxicity studies should start with thorough ecological inspections.

The first approach was largely promoted by the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG), with the aim of providing standardized, scientifically based criteria to guide the clean-up requirements of contaminated sites (Twerdok, 1999). The group elaborated a structured, pre-defined experimental procedure, intended to be applied to specific polluted sites to establish the level of risk posed to living beings and environment (Claff, 1999).

Following TPHCWG methodology, the safety limit (and therefore clean-up goals) of a certain contaminated area where evaluated in a three step process.

The first step is the analysis of the chemical composition of the specific TPH residue, by grouping its components in fractions. Each fraction is constituted by hydrocarbons with similar number of carbons, similar structure or the carbon chain (aliphatic or aromatic), similar physical properties (boiling point, vapour pressure, solubility), and similar aptness to bind to solid organic structures (partition factors for soil to water and soil to vapour concentration at equilibrium). The idea is that these characteristics determine whether groups of compounds will be more prone to evaporate, leach, or remain in the soil, which gives an indication of their bioavailability (McMillen, Magaw and Carovillano, 2001; TPHCWG, 1998). By determining the relative amount of each fraction, therefore, the total behaviour of the residue can be predicted with acceptable confidence.

The second step consists in calculating 'Risk Based Screening Levels' for each fraction component. These are mathematical calculations, in which the risk levels are functions not only of the fraction concentration, but also of its 'reference dose'. This is the maximum dose considered safe, and it is based on literature reviews of available studies, which are mainly exposure studies in lab animals or other models (Vorhees and Butler, 2008). Notably, to date there are available studies on a small amount of TPS components. TPHCWG therefore needs to approximate that components of each fraction have not only similar structure and physical properties, but also similar toxicology (ATSDR 1999, 13–14). The third step is the evaluation of the geology of the contaminated environment, which is needed to evaluate the possibility for the chemical to reach living organisms.

This procedure was described as 'risk-based': for every component, or fraction, it weights the chemicals' capacity of becoming available against its intrinsic toxicity (Vorhees and Butler, 1999). Its application throughout the years favoured the reiteration and establishment of the idea that oil toxicity is mainly acute, and diminishes exponentially with weathering. Indeed, the smaller aromatic compounds (benzene, toluene, ethylbenzene, xylene, and light polycyclic aromatic hydrocarbons), which are known to be carcinogenic, mutagenic or teratogenic because of their physical properties, are also the easiest to weather, and dissolve in a short time, provoking only acute toxicity. Heavier compounds, instead, are inert and therefore not biologically available. TPHCWG predictions often judged their bioavailable concentrations far below the risk levels.

Based on a process of causal separation, analysis and re-composition, experimentation and model predictions, which has been regarded as successful scientific approaches since the scientific revolution, the conclusions have been widely accepted. Because of this reasoning, indeed, many US states lowered their cleaning-up demands in contaminated areas (Sawyer and Loja, 2015).

Many of these risk evaluations, however, were challenged by different scientific approaches, initiated in the field of ecology. 'Injury to a species resulting from long –term exposure to low concentrations of pollutants is seldom noted or even tested', write Rice et al. (2001). After disastrous contamination episodes, such as oil spills, ecologists had for the first time the opportunity to observe the long-term effects of exposure to low concentrations of oil, in its real-life complexity within an open system. Unprecedented findings challenged the established paradigms of oil toxicity. For instance, fish mortality was observed at constant rate four years

after the Exxon Valdez oil spill, suggesting high long-term oil toxicity (Incadona et al., 2005). Even more surprisingly, toxicity in fish embryos was higher in areas situated further away from the contamination source, suggesting that it increases at lower doses of chemical (Incadona, 2012). The lowering amounts of fish eggs in polluted ecosystems and molecular characterisation of embryos suggested that developing fish have high sensibility to hydrocarbons (Rice et al., 2001). This was later tested by a series long-term exposures of larvae to low concentration of petroleum, showing that embryos are sensitive to part per billions (ppb) concentration of crude oil, including genetic malformations expressed often long after the exposure ended (Incadona et al., 2005; Rice et al., 2001). Ecologists thus suggested that weathering of oil does indeed tend to diminish its toxicity, but at an unpredictable pace that depends on the specific environmental conditions. Another surprising finding was that oil itself did not weather at the expected pace. Instead, rates of biodegradation diminished through time, since the most persistent residuals were trapped in environments (such as mussel beads or gravel shores), where dispersion was hindered by physical barriers (Peterson et al., 2003; Paine et al., 1996). Finally, ecological observation of coastal communities suggested that in the long term toxic effects in the population are amplified rather than diminished through environmental interactions (e.g. in the trophic chain), and therefore become substantial (Peterson et al., 2003).

Two main criticisms were moved by ecologists to orthodox risk assessment frameworks. The first is that the critical importance of analyzing post-contamination scenarios and ecosystems has been overlooked: ‘Oil spills, while tragic, represent opportunities to gain insight into the dynamics of marine ecosystems and should not be wasted’ (Paine et al., 1996). The second is that

the use of short-time exposure studies and the reliance on closed-system models hinder the predictive power of risk assessment frameworks. An ecosystem-based toxicology instead is needed, since 'ecologists have long acknowledged the potential importance of interaction cascades of indirect effects' (Peterson et al., 2003). Predictions for toxic impacts must start from ecological observations of contaminated sites. These ought not only to provide hypotheses of harm, which would otherwise be unpredictable. They should also guide the design of experiments aimed to test such hypotheses, for instance by prolonging the length of exposure to sub-lethal doses.

We thus see that, although the orthodox, predictive approach to oil toxicity adopts a number of procedures that can be fully defended as 'scientifically sound', opponents from the field of ecology claim that something is nevertheless wrong or unsatisfactory about this way of studying and establishing causal relationships. More seriously, they argue that traditional methodologies for causal prediction about oil toxicity are detrimental, because they perpetrate certain orthodoxies that contribute to hold back knowledge in the field. Although evidence emerging from post-oil spill research motivated partially this disagreement, the controversy is not purely a matter of this specific evidence. The rejection of acute laboratory tests as sufficient indicators of toxicity has long been a well- stablished principle of ecotoxicology (Peterson et al., 2003).

We now move on to discuss how this particular methodological disagreement relates with basic philosophical assumptions about causation.

Scientific methodology and the ontology of causation

Scientific norms and methods are not ontologically neutral, but carry with them a number of philosophical assumptions, especially concerning the nature of causation (see Kerry et al., 2012; Anjum, 2016; Rocca and Andersen, 2017; Anjum and Mumford, forthcoming). What exactly causation consists in has been an ongoing philosophical discussion at least since Aristotle. Some think of causation in strict empiricist terms, such as regularity (Hume, 1739; Psillos, 2002), probability raising (Reichenbach, 1956; Suppes, 1970) or difference-making (Lewis, 1973; Collins, Hall and Paul, 2004). These theories analyze causation into something perfectly observable, which could motivate epidemiological approaches where epistemic priority is given to statistical and comparative methods. Within this approach, causal conclusions are drawn on the basis of data, using various tools for analysis and calculation. Other theories focus on the interventionist side of causation, for instance causal mechanisms (Glennan, 1996), manipulability (Woodward, 2003), processes (Salmon, 1984; 1998) or energy transference (Fair, 1979; Dowe, 2000; Kistler, 2006). From this type of perspective, experimental methods might be better for teasing out causation than statistical methods. Typical for such methods is that they are more dependent on theoretical assumptions, so would not be the preferred approach for a strict empiricist or positivist.

These are some of the available theories of causation, and although they go in different directions, there are of course some degree of overlap between them, as is natural, since they all attempt to define the same phenomenon. While philosophers disagree over what causation is, it is nevertheless possible to detect some form of generally accepted orthodoxy or 'folk notion' of

causation (Norton, 2007; Kutach, 2007) in traditional scientific methodology (Anjum and Mumford, forthcoming, chs 4-6). As we will show here, the established, predictive approach to oil toxicity, described above ('the TPHCWG approach'), carries most of the elements of the orthodox view of causation. This view includes at least the following elements: (i) under specific conditions, the same cause always gives the same effect; (ii) causes necessitate their effects, meaning that if one has the complete cause, the effect will necessarily follow; (iii) causation brings with it determinism, thus predictability; (iv) causal complexity is best understood by composition and de-composition and; (v) causes make a difference. Let us consider these points one by one, look for their philosophical roots and relate them to our case.

(i) This commitment, of same cause, same effect, can be traced back to the empiricist philosophy of David Hume (1739). Hume, in his famous analysis of causation, motivated the orthodox view that has later dominated scientific methodology. On his view, causation is an extrinsic relation between two separate events that follow each other regularly and repeatedly. Repeatability is a central criterion for regularity theory, since no regularity can be observed from a single case. On Hume's theory, therefore, there can be no one-off, unique case of causation.

(ii, iii) Although Hume argued that there was nothing more to causation than a particular type of correlation, namely perfect regularity, most of his opponents have argued that causes produce their effects by necessitating them (which was the feature that Hume denied) (e.g. Harré and Madden, 1975; Mackie, 1980; Skyrms, 1980; Bird, 2007; Marmodoro, 2016; see Mumford and Anjum, 2011, ch. 3 for a detailed discussion). The reason why causation manifests itself in perfect regularity is then exactly because of this necessity. This view, that causes necessitate or

guarantee their effects, has also motivated the idea of causal determinism; that given one set of initial conditions, there can be only one possible outcome (for a criticism of this view, see Anscombe, 1971; Suárez and San Pedro, 2011). Causal determinism would make effects perfectly predictable, at least in principle, as long as the initial conditions are known. This is a standard assumption in scientific approaches, including the TPHCWG approach, designed to acquire as much information as possible about the oil sediment's composition and soils conditions at a certain time point, with the expectation that the more we know about such conditions the more accurately we will predict oil's environmental fate.

(iv) This aspect of causation does not primarily concern how the cause is linked to the effect (for instance by necessity, determinism or counterfactual dependence), but tells us something about the nature of the cause itself, and the type of effect it can produce. In philosophy, this is related to the debate on reductionism versus holism or emergence. Are causes found on the lowest level in nature, as the reductionist argue, or can there be higher-level causal processes, or even top-down causation (Dupré, 1993; Dupre, 2001; Skaftnesmo, 2009)? Can causal factor interact to create novel phenomena, known as cases of emergence, or is any novelty only due to our epistemic limitations about how their parts compose (Kim, 2006; Wilson, 2016)? The idea of reductionism and additive composition of causes goes back to the Scientific Revolution, with Descartes (1637) as a central figure. His analytic-synthetic method was an important element of the mechanistic philosophy, from which scientific methodology is still largely influenced. With this method, Descartes suggested a way to approach a complex problem: first by dividing it into smaller parts and treat them separately, and then putting them back together again into the

complex whole. From this perspective, complexity is best understood through identification of single causal factors and elements, and the observation of their behaviour in isolation. Crucial for this method, however, is the basic assumption that the world is composed of (in principle) isolated components. Complexity is then only a matter of additive and linear composition, where the whole is nothing but the sum of its parts. Descartes' mechanistic philosophy was thus an important inspiration for Modern deterministic and reductionist thinking of nature, depicting living things as clock-like machines. In standard scientific risk assessment, such as the TPHCWG approach, this assumption can be clearly detected. By dividing petroleum into fractions, and understanding how each fraction behaves in isolation, one can at least in principle 'recompose' the total behaviour of petroleum. This implies the assumptions that the total mix is nothing more than the sum of its parts, a straightforward Cartesian assumption.

(v) To disentangle complexity is considered an important scientific task. One reason for this is that complexity makes it difficult to see which element is causally responsible for which part of the effect. On a difference-making account of causation (Lewis, 1973; Collins, Hall and Paul, 2004), identifying the cause is a problem if the effect is overdetermined, that is, that there is more than one thing that could have produced the effect. If we remove something that we think is causally responsible for the effect, but the effect nevertheless occurs, then we cannot draw a causal conclusion from the intervention, since it didn't make a difference. If we want to know the causal potential of some complex situation or substance, therefore, the best way to do this is to separate it into smaller parts isolate them from their complex context, and see what they do on their own. This philosophical intuition motivates a scientific approach favouring causal isolation

as the best way to understand causation (for instance, in experimentation). While this does not rule out that simple observation of 'real-world' single cases might give us evidence of difference making in certain cases, it nevertheless implies that the best way to identify something's causal role(s) is to start from isolating each causal factor from their confounders. In the TPHCWG approach to risk assessment, the concept of 'Risk Based Screening Levels', described above, is heavily relying on toxicological experiments, where chemicals are tested in close environment and isolation, using experimental models. Moreover, as noted in (i), Hume's regularity theory of causation requires repeatability. A one-time experiment has little value. In order to see causation, we need to see it many times.

The TPHCWG approach to risk assessment thus shows a clear commitment to the orthodox view of causation (the 'folk view'), which contains elements of regularity theory, causal determinism, causal complexity as composition and difference-making.

An alternative philosophical approach

There are reasons for a life scientist, probably for an ecologist in particular, to doubt the orthodox, or 'folk view' of causation. First of all, it relies on causation to produce, at least in specific conditions, perfect regularities, which are difficult to find outside of theoretical physics. Only through theoretical models or abstractions, isolation of interferers and perfectly controlled experimentations, could anything close to a perfect regularity be produced. However, ideal or de-contextualised models can fall short in understanding causal relationships concerning living

organisms in an ecosystem, as we saw in the case presented above. Second, the orthodox view of causation tends to give little space to emergent phenomena and higher-level causal processes, since they usually start explanations and predictions from the micro-level. By initiating causal explanations from the molecular characterisation of petroleum and organic matrixes, and excluding higher-level mechanisms, we are led to believe that oil toxicity can only diminish, or at worse remain unchanged, in time. However, higher-level causal mechanisms, such as trophic chain and other environmental interaction, have the opposite effect of increasing toxicity, as shown by Peterson and colleagues. Third, ecologists have little use of the concept of causal determinism (the idea that, given the same causal conditions, only one outcome is possible). This is primarily because ecology only deals with open systems, where it is at no time possible to know all the initial conditions, or the 'total cause'. Moreover, no two identical initial conditions exist in an ecosystem. There is never a case of exactly the same causal set-up, and if two are similar, then this might make a vast difference to the outcome. The causal necessitarian can only offer a thought experiment or a theoretical model in which one did have identical conditions (Mumford and Anjum, 2011: ch. 3). For instance, one might reason that if an organism had an identical twin, with the same conditions, lifestyle, environment, and so on, then if both met the same stressor, the same toxicity would be guaranteed. This is exactly the kind of situation that one tries to produce in the lab setting, in order to accommodate repeatability and replicability. The expectation of perfect regularity, or even causal necessity, is thus philosophically motivated and based on an abstraction from our experience, rather than being an accurate report of it. Again, abstractions might be helpful, but they can become counterproductive as well, as described by the case.

There are some philosophical views of causation that stand in opposition to the presented orthodoxy, and we suggest that they are better suited to ground an ecological approach to oil toxicity, as the one described in the case. Cartwright (1999) famously argues against the absolute utility of isolated experimental set-ups, calling them nomological machines (since they are designed to produce causal laws), and pointing out that they are very different from the natural context to which the results are supposed to apply (Cartwright, 2007). External validity of the results from the isolated, idealised setting to the real life complexity then becomes a problem.

Starting from a different set of basic assumptions, about the nature of causation and of complexity, we could motivate a different methodological approach. Here, we suggest that a better philosophical starting-point is to understand causation as dispositions, or causal powers (Cartwright, 1989; Mumford, 1998; Mellor, 2000; Ellis, 2001; Molnar, 2003; Heil, 2004; Martin, 2008; Marmodoro, 2010; Groff, 2013; Groff and Greco, 2013; Jacobs, 2017). In particular, we will briefly present a version of dispositionalism called causal dispositionalism, developed by Mumford and Anjum (2011), since this theory explicitly challenges the basic assumptions from Hume and Descartes, discussed above. In addition, causal dispositionalism is currently the only causal theory that refuses both Humean regularity theory *and* causal necessity. It embraces instead a dispositional modality of causation, the idea that causation involves irreducible tendencies (Anjum and Mumford, 2018).

On causal dispositionalism, causation is not an extrinsic relation between two separated events, but rather a process of change. This process involves the manifestation of intrinsic, dispositional properties of things, such as when a fragile glass breaks or a poisonous substance kills.

Dispositions will tend to manifest under certain conditions. A glass tends to break when struck, but it is a tendency only, and no guarantee, that the causal disposition is manifested. Tendencies come in different degrees of strength. Glass can be more or less fragile, and new windows don't break as easily as old ones. Something can have many dispositions, and to various degrees. Oral contraception, for instance, is causally linked to both prevention of ovulation, in more than 99% of cases, and produce venous thromboembolism, in around 15 in 100.000 cases. Weak tendencies are more difficult to detect, but can nevertheless be a matter of life and death. In this sense, to uncover causal dispositions is vital for risk assessment.

If no effect is observed following the cause, then for Hume and other empiricists, no causation has happened. The regularity theory presupposes that both the cause and the effect occur, since we need to observe their correlation to infer that there is causation between them. For a dispositionalist, however, there could be causal dispositions in place, doing their causal work, but without an immediate observable effect. Being a mutual process between many dispositional properties, causation in the dispositionalist account has a dilated temporal dimension. The effect can be manifested at a much later stage, when a threshold effect is reached, or when some further factor is added. For dispositionalists, causal production is *essentially* complex. This means that causal factors are not merely an intricate web of extrinsic instances, in which case, it would be possible to get the complete causal picture by disentangling each causal factor and studying it in isolation. Rather, causation happens when different dispositional properties come together and start interacting. It is *because* of such interaction that the causal process begins. It is, in other words, a mutual manifestation (Martin, 2008; Anjum and Mumford, 2017).

We can explain causal complexity with a simple example: I strike a match and it lights. Although this can only happen under the presence of oxygen, we would not normally say that oxygen was the cause of the lighting. However, on a dispositionalist view, the match, the striking and the oxygen are all causes. Any distinction between them is thus only a matter of pragmatics, not ontology. The effect depends not only on one of the factors being present, but on their joint interaction. There is therefore no need to draw a strict distinction between causes and background conditions. Many of the background conditions are considered exactly because they contribute causally to the outcome, as mutual manifestation partners. Ontologically, the causal contribution of oxygen is exactly comparable to the striking, level of humidity, the sulphur on the match tip, the wood, etc. Often, what we take to be the cause is simply the factor that was last added, which could even be the oxygen, as in the *Apollo 1* fire in 1967. In this historical case, the fire was reputed to have started precisely because of the pure oxygen atmosphere in the cabin, which is not a natural condition. Other more orthodox theories of causation can account for the essential role of context, without the use of dispositions (see for instance Mackie, 1980). These theories, however, also assume causal necessity, since the total cause is sufficient to produce the effect. Context is essential, they profess, but if we knew everything about it, then we could make our prediction with total confidence. Causal dispositionalism, in contrast, denies this intuition and adheres to everyday experience. The total cause only tends to produce its effect, which means that all causal predictions are fallible. This aspect of causal dispositionalism is critical to explain the disagreement in the case of petroleum risk-assessment methodology, as described above.

This mutual interaction of causal factors can be linear, as assumed in the analytic-synthetic approach, but the default expectation should be that it is nonlinear. Dispositionalism acknowledges this as an important feature of causation: what something does in one context does not automatically transfer to can do in another context, together with different manifestation partners. This has practical consequences for scientific methodology. To study the various components of oil in isolation might not accurately represent what they can do together, for instance, which is why researchers promote cumulative risk assessment as a better alternative to traditional approaches (e.g. Callahan and Sexton, 2007; Checker, 2007; Løkke, 2010; Meek et al., 2011; Williams et al., 2012; Gallagher et al., 2015). Chemical interaction is a typical example of nonlinear composition, where the dispositional properties of a chemical mixture can be very different from the dispositional property of each component (a lump of sodium tossed in water for instance has a very strong disposition to explode, which water and sodium alone do not have).

This is why we say that causation is *essentially* context dependent, meaning that what effect is produced from a certain exposure will depend on what else is present. When making predictions we might acknowledge such context-sensitivity, but scientific methodology often includes attempts to minimise any difference in context, in order to guarantee that same exposure gives same outcome under otherwise like circumstances. Causal predictions are thus made under certain pragmatic assumptions, considering a model of a closed and deterministic system with limited complexity and additive composition of the factors. In recent years, with the progress of science and new emerging technologies, however, there is an increasing awareness of how the theoretical assumptions of the model differs from the real life contexts for which predictions are

made: indeterministic, nonlinear and open systems, with a number of unknown factors. A dispositionalist would take the latter case as the default situation, while a positivist would typically start from the theoretical model of abstraction and treat the latter as the exceptional or at least derivable from what we can learn within the model.

Discussion

The aim of this paper was to show that a scientific disagreement about which approach is the most appropriate for assessing health risks in oil-contaminated sites can be motivated, at least partially, by different philosophical basic assumptions about the nature of causation. Some clarifications are needed at this point. First, we do not mean to claim here that ecological observational approaches and experimental predictive approaches are mutually exclusive. On the contrary, risk assessment frameworks usually include both approaches, which should ideally be complementary (Callahan and Sexton, 2007). But how?

We have tried to show that the choice of methodological approach at least partly depends on one's basic assumptions concerning the nature of causation. From the perspective, of what we called 'orthodox concept of causation' (regularity theory, causal determinism, causal complexity as composition and difference-making), one might think that causal complexity and context sensitivity are obstacles that hinder causal understanding. Hence, the ideal scientific enquiry must begin by shielding off the cause from any potential interfering factors, in order to identify its true causal role(s). Having established this, one might go on to look at its interactions in more

and more complex settings. From the perspective of dispositionalism, in particular of causal dispositionalism, instead, complexity and context sensitivity are not problems for understanding causation, but rather something that can be used to our advantage. Only in interaction with other factors can we observe what causal work something is capable of doing, and different interactions will reveal different causal powers. But then it is crucial to identify also those contextual factors and interactions that contribute to the effect, either by aggregation or counteraction. Such knowledge will inform further experimentation, for instance by isolating parts of the causal process with only a few causal factors. We see, then, that these two different stances concerning causation can lead to rather different processes of assessment.

Secondly, one might object that it is hard to think about scientists as tacitly holding perfectly definite philosophical intuitions, for instance in respect to the modality of causation. With our terminology, we indicate different relative positions in an argumentative spectrum, rather than extreme positions. We hope to have shown, however, that the difference can be significant, as in the case of oil contamination, presented here.

Suppose one accepts our argument. Is there any practical significance to it? What exactly is the relevance of this analysis for the debate about risk assessment of oil contamination? An example might help illustrate the point. Going back to the beginning of the paper, let's consider the argument about epidemiological assessment of oil contamination in areas where population registries and resources are limited, as presented in the introduction. The well-known case of industrial oil residues spread by Chevron Corporation in the Ecuadorian Amazon is an emblematic one (Hurtig and Sebastian, 2005). Neither TPHCWG predictive approaches nor population studies

could demonstrate robustly the level of risk to which Ecuadorian population and environment are exposed (Sawyer and Loja, 2015; O'Reilly and Thorsen, 2010; O'Callaghan-Gordo et al., 2016). Seen from an orthodox, or 'folk view' of causation, the way out of this situation would be to improve population registries and wait until better data become available, so that reliable correlation can be found and regularities be uncovered in a large-population scale. Slow but steady movements in that direction are happening (see Kelsh et al., 2009). This could lead to more robust studies, although it has not yet and could still take generations. Although the urgency of the matter is clear, if we adopt the orthodox assumptions about causation we have to agree with Siemiatycki that science must follow its norms, rather than being 'on the side of the angels' (Siemiatycki, 2002). To this, however, Ecuadorian epidemiologists answer that, rather than for the angels, science need to be 'for the people', and therefore statistics, p-values and confounders should never be evaluated without 'an element of plausibility' (Hurtig and Sebastian, 2005: 1171). Are Hurtig and Sebastian proposing that we stop following norms of science in order to help the cause of their people? Not necessarily, according to our argument.

If our reasoning is sound, then we must accept that norms of science can change depending on which basic conceptual and ontological assumptions one adopts. By assuming that causation is *irreducibly* tendential, complex and context sensitive, for instance, the best chance we have to understand a causal process is to start by looking for causal dispositions and mutual manifestation partners in specific contexts. As explained above, repetitions, regularity and isolation are not necessarily detectors of dispositions. From this perspective, a scientifically sound enquiry should never discard or underestimate the following elements:

(i) Detailed assessment of post-harm case reports and case series, including not only biomarkers of exposure and effect (O'Callaghan-Gordo et al., 2016), but also non-traditional biomonitoring practices for exposure assessment (i.e. 'exposome', see Dennis et al., 2017), since these are all essential for generation of hypotheses in a non-targeted way.

(ii) Evidence of a possible mechanism of harm, including relevant mutual manifestation partners for linear and nonlinear causal interactions.

(iii) In-situ observation of the higher-level mechanisms that could interfere with chemical toxicity, requiring a participatory, rather than purely observational, approach to research, taking into account insights from anthropology and social science in the process of risk assessment (Checker, 2007).

(iv) A locally adapted experimental design, to accommodate causal heterogeneity or even uniqueness, rather than a rigid approach, applied universally across different contexts.

(v) A precautionary attitude: if we think of causation as irreducibly tendential, complex and context-sensitive, the precautionary principle becomes an essential epistemic value. If one takes this to be Hurtig and Sebastian's background assumption, it follows that prioritising evidence from population studies while dismissing contextual circumstances (such as the high level of oil contamination in drinking water, the heavy metal levels in the resident's blood, and the existence of evidence for a possible mechanism of toxicity), does not obey the norms of science.

We see, therefore, how the debate over risk assessment methodologies can be sometimes re-framed in terms of a more fundamental disagreement. Importantly, once basic assumptions are made explicit, they are also exposed to public examination. They can for instance be tested in relation to current scientific knowledge, which historically has been essential for paradigm shifts (see for instance Einstein, 1905). As suggested by feminist epistemologists, '[T]he objectivity of science demands that the background assumptions of research programs be exposed to criticism. A scientific community composed of inquirers who share the same background assumptions is unlikely to be aware of the roles these assumptions play […], and even less likely to examine these assumptions critically' (Anderson, 1995: 79). One can object that biological knowledge or empirical evidence might not be sufficient to ground a unanimous choice toward one or the other assumption about causation. Many philosophical basic assumptions, after all, are equally scientifically justifiable (see also Rocca and Andersen, 2017). In that case, however, it is still possible to use rational arguments of an extra-scientific nature to guide the choice. As Heather Douglas influentially postulated, 'In making a choice between these positions, scientists must consider the consequences of their choice, particularly if they are wrong' (Douglas, 2000: 576). Framing the scientific disagreement, therefore, and spotting where the real divergence lies, help scientist and decision-makers to make conscious and informed value judgement within the process of risk assessment.

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