"The Book of Why" For Epidemiologists
Prof. George Davey Smith and Prof. Judea Pearl
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Thanks again for the invite to UCLA. I would first like to ask, and you've got to not be influenced by the fact that Judea is here... I want to see who have got 'The Book of Why'? So that is the majority of people. Keep those hands up, people are holding them up .. they want to get it signed. Who has read any of it? Who has read it all the way through? Okay so, some hands have gone down.

Okay, so I have structured this around talking about 'The Book of Why' from the perspective of an empirical researcher. Also I would imagine some of you who haven't read 'The Book of Why', students might also know the primer that Judea and colleagues wrote as well. So if you have read either of them then this is fine.

(Slide 2) I'm going to outline as I see it the value of DAG theory to epidemiology. I'm also going to say a bit about the reality of use of DAGs in epidemiology, some of which has come from my experience of having to read a very large number of papers as the editor of the International Journal of Epidemiology. We were getting 1000 papers a year by the time I was worn out, well Shah Ebrahim and I (there were two of us co-editing it, with a team including Karen Michaels as associate editors).

I'm going to say something about Sewall Wright here, which I'll call "getting Wright wrong". Judea can come back on his view of Sewall Wright. I'm going to talk about where background knowledge comes from and the consequences of believing the DAGs. (Slide 3) Those of you who have read 'The Book of Why' will know, I think, the really useful framing of this at the beginning in terms of the ladder of causation, starting off with association (slide 4) in the statistics of Karl Pearson and his correlation coefficients, with a debatable interpretation of Pearson's apparent rejection of the concept of causality. Such quotes can be given, but it is very clear that Pearson was making causal statements in a very large amount of his actually applied work, and my reading of "The Grammar of Science" is that Pearson denied that individual-event causation was a meaningful concept. It is clear that at the group level he made statements that were very clear. As a eugenicist, he obviously meant you could do something through manipulation, so he certainly believed in group level causation.

(Slide 5) Then of course R.A Fisher appears at the second level of the ladder - intervention - with Fisher's development of randomized control trials, but importantly also Fisher's very clear notion of causation in the development of the analysis of variance in 1918, in its application to partitioning genetic and environmental causation of traits, again at an aggregate level. Then at the top of the ladder, (Slide 6) Judea appears with the counterfactual reasoning which is a form of abstraction of what you have learnt from the levels below, and allows transportability of what you have learnt. (Slide 7)

Also you will have seen this really helpful diagram as well at the beginning of the "engine of inference", with the key issue of background knowledge in the dotted box on the left, which allows making quantifiable assumptions and building a model which allows you to have a recipe for answering the query at a model level, and then to actually generate estimates with actual data. This is the inference engine you'll have seen.

(Slide 8) Now for what from my point of view are absolutely unequivocal gains to epidemiology from employing directed acyclic graphs. The first is learning about the structure of biases. (Slide 9) Here is

an illustration of one such bias which I think really helps with the structure. As I always do as an empirical researcher - I can't think of things that are at an abstract level really – is talk about an actual paper. One in the New England Journal of Medicine, 15-20 years ago purported to show that heavy alcohol consumption reduced the risk of stroke, which just seems implausible because we know alcohol increases blood pressure, which increases stroke, and also has some anti-coagulant effects which certainly increase haemorrhagic stroke. And we now know from other methods such as Mendelian randomization alcohol actually increases the risk of stroke rather substantially, as in the recent Lancet paper from the Kadoorie China Biobank group for example.

So why did the NEJM study get the answer wrong? The most likely reason is that the study was based on the American Cancer Society Volunteer Cohort. A tiny proportion of people who could have participated in this cohort actually did participate, these are volunteers. If you are a heavy alcohol user you are less likely to volunteer; and if you are in poor health you are less likely to volunteer. Depending on how those two things act together this could lead to generating a negative association in the volunteers between alcohol consumption and poor health. i.e. the heavy alcohol consumers would be less likely also to have poor health and maybe other characteristics which would increase the risk of stroke. Now that structure of bias - the idea that you automatically condition on a variable (participation) when you have volunteer samples, now this was recognised before put into DAGs and labelled collider bias. For example at a time when I certainly was not using DAGs, in 2004, we explain this bias here (Slide 10). But when you explain it in words, it is a cumbersome explanation to give. It is considerably easier to explain with the diagram. Interestingly the first example that I know of - also referenced in 'The Book of Why' - is Arthur Cecil Pigou's discussion, again interestingly of alcohol (slide 11). This related to heavy alcohol consumption and the quality of offspring, with Karl Pearson suggesting from various secondary data analyses that heavy alcohol consumption in the parents didn't actually adversely affect the offspring. When I say Pigou was not the 4th man it is because in Britain he became famous after he was dead as possibly being the 4th man who had be one of the spies for the Soviet Union. But he wasn't the 4th man, and what he is best known for are Pigouvian taxes which are taxes on negative externalities. So for example if climate change reducing taxes were introduced, they would be Pigouvian taxes. But he explained in words here (slide 12) from the Westminster Gazette of 1911 the structure of collier bias. We've reprinted it in the IJE (slide 13); it's great being an editor because you could just say "we will reprint this" when we wanted to (Slide 14). So an unequivocal gain from employing DAGs is you allow the structure of biases to become transportable, because it's not just alcohol and volunteering into studies, or alcohol and other qualities of the parents that Pigou was talking about, but a huge number of potential biases introduced into epidemiology by this sort of conditioning on an outcome (or "collider bias"). So you can make understanding of these biases transportable through DAGs, and explicable. And that I think is an unequivocal gain.

Judea, in a piece he wrote a while ago about why didn't economists pick up on path diagrams said the explanation was (Slide 15)

> *"... early econometricians were extremely careful mathematicians, they thought they could keep the mathematics in purely equational-statistical form and just reason about structure in their heads. Indeed, they managed to do so surprisingly well, because they were truly remarkable individuals and could do it in their heads."*

And the same is definitely the case of some of our heroes in epidemiology (slide 16). Here's Brian MacMahon in 1965. Who has heard on the "smoking and birthweight paradox"? OK few of you have heard of it. It is the paradox that, if you're looking at your own mortality (as all of us do) and you were born with low birth weight (as I actually was), then you actually want your mother to be a heavy smoker, as your mortality is actually better if your mother was a heavy smoker, at an given level of birth weight, which seems counterintuitive and some people used it to support the notion that

maternal smoking wasn't bad. But I'm thankful you know my mother was a heavy smoker, not because that was good for me, but because it means that my low birth weight is less likely to portend bad things. Some people have suggested this paradox was solved through understanding the causal structures through causal diagrams. (Slide 17) But Brian MacMahon said this in words before such diagrams were generally used. There are many examples of such things being solved - because some of these people are clever, and they can solve them in their heads - but as most of us aren't, and can't do that, it's very useful to have DAGS for transportability of the structure of biases.

The second advantage of DAGS is providing an explicit rationale for constructing adjustment sets, you know you read epidemiological papers, and often things happened to correlate with your exposure of interest, and everything's adjusted for. Actually having to provide a rationale for that is good; you don't want to adjust for concomitant variables which are influenced by the exposure and might be influenced by other things. At least you don't want to in your main analysis; you might want to do that in sensitivity analyses etc. (slide 18) And related to that is that DAGS lead to an explicit presentation of some of the assumptions the researcher holds. In presenting a DAG you have to make your assumptions clear, some of those assumptions may be wrong, and it's good if they've been put into a diagram (slide 19).

Thinking about the DAG for a particular study design or particular study situation has been or can be helpful with methodological developments. Did anyone come yesterday to the causal inference workshop? You did … people aren't admitting it .. (laugh) God it was that bad! (slide 20) Anyway, yesterday I talked about sibling studies and how actually trying to talk in words about the possible issues of doing between sibling comparison, which we may just think "adjust" for lots of things we want to control for, can introduce problems as well, and again, waving one's hands about, one could talk about it without diagrams, or with schematics which weren't causal diagrams, but it's difficult (slide 22). So these things are unequivocal gains for epidemiology. The issues I have are, as I say from the experience of being an editor amongst other things, with the reality of the use of DAGs in actually published epidemiology. So there are programs such as Johannes Textor's DAGitty programme - this now has approaching 700 citations. When I was writing a paper in 2016 with Nancy Krieger, it had only about 150 at that time, and I went through the papers that cited it and constructed DAGs supposedly using it. It was not a great experience. The authors themselves are apparently carrying out a full systematic review of users of the program (slide 23). But in lots of cases one saw that even though the program was used to help people construct the DAGS often it appeared without necessary thought. This just happens to be the most recent citation when Nancy and I wrote the paper in which we discovered this, that you

> *"Directed acyclic graphs and 10% change ... (*10% change in estimate procedures are not ones I hope taught in UCLA as ways of variable selection) ... *in estimate procedures were used to identify covariates for inclusion in multivariable models; these included age, education, living with a partner, parity … (*which *co*uld be an outcome of breast feeding, which was the exposure)*, … and history of preterm birth"*. (Slide 23)

And this is repeated over and over and over again, the actual reality of what's done is one doesn't see the benefits that one sees in the abstract use of the DAGS for thinking about biases in transportability. (slide 23) I won't go through this, given time, but when you actually look at any DAGS in papers, there are always many issues with it with every particular one. Because you've got to remember that as important - or possibly more important to the paths the actual arrows show – are the arrows in a DAG that aren't there. When you have nodes or vertices that are not joined by arrows you are making the assumption that there is no causal effect between those two things. So the absence of arrows is as important as the presence of arrows (slide 25). Or an example which I think is a really interesting example because it's one of the classic teaching examples comes from Maria

Glymour's chapter on "Using causal diagrams to understand common problems in social epidemiology". She is talking about looking at the effects of education on diabetes, and she draws a DAG where she thinks that family income during childhood will affect their education, which it will do. And in return mother's genetic diabetes risk will affect the fact the mother had diabetes, and of course your mother's genetic diabetes risk influences your risk of diabetes, so if you are trying to look at low education and diabetes you shouldn't adjust for mother's having diabetes. (slide 26) This is didactically said

> "*Under the graphical criteria, one should not include mother's diabetes status as a covariant"*.

I hear these things parroted back because people have learnt them from these pedagogic approaches. The point is that mothers glucose level during pregnancy may well effect cognitive development in utero, there's a whole raft of studies where that is the case and of course it effects diabetes risk. Thus this could be viewed as a straightforward and probably important confounder. But you're told you shouldn't adjust for an M-bias collider. But it's also a potential confounder. You get these rules and we saw these all the time in papers that were submitted to the IJE: people will say we didn't adjust for these factors because of a data structure like this. Sander Greenland wrote a paper which I highly recommend people go back and read in 1993, when he discussed the likely magnitude of bias through not adjusting for confounders due to being worried about collider adjustment. This is seen just both in didactic teaching and in what you actually get submitted, which is what my experience is based on.

(slide 27) This is a highly recommended paper, if people haven't read it by Peterson – 'Causal Models and learning from data'. And there they say:

> *"A structural causal model provides a tool for understanding whether background knowledge, combined with the observed data, is sufficient to allow a causal question to be translated into a statistical estimand, and, if not, what additional data or assumptions are needed".*

And then

> *"In many cases, rigorous application of a formal causal framework forces us to conclude the existing knowledge and data are insufficient to claim identifiability - in itself a useful contribution."*

And I very much agree with this, that this really would be a useful contribution, but again my experience is I don't think there are situations where people actually do not go ahead if they've got a study they're doing. I don't know of situations where people say 'okay you know we've looked at the assumptions that would be required to make this estimand sensible and we won't do it. I've not seen that, I'd be interested to hear people's experience of having seen that.

(slide 28) And what about the

> "*... assumptions of causal DAGS and causal modelling approaches?"*

when you're actually looking at data, as opposed to thinking about abstract issues of bias and data structure, etc.

(slide 29) Well you know there are obviously many assumptions, but to discuss two important ones. First no measurement error. A remarkable thing is in a good chapter on 'Probabilstic

Computational Causal Discovery for Systems Biology' using DAGs (and applying it to settings which they call "Mendelian randomization" - but I don't think that's what they're doing", but I suppose I don't have a patent). What they say though is that

> *"No measurement error: the variables are measured with measurement error. This is a subtle assumption that is required to learn Causal Bayesian Network (CBNs), often not realized by practitioners who apply these techniques."*

This is from people in the field: measurement error being a subtle assumption. Again how often have you actually seen DAGs which actually include measurement error? Of course you say you can put measurement error in. But when have you seen that done, and when have you seen people who move to the next stage and say that means you can't do that estimation?

Were any people here yesterday for my "Post-modern epidemiology" talk? So at that talk you will know that in 1991 without the aid of any DAGs, and thinking about measurement error in the set of variables I published a paper in 1991 saying that the HDL cholesterol - triglycerides question could not be answered with data. So (slide 27) DAGs aren't required to make that sort of statement. I've seen very few examples of that which have followed on from DAG modelling.

(slide 30) Oh yeah … and there's "no unmeasured confounding" too …

That's an assumption you can't make estimates if there's unmeasured confounding. The equation is that (slide 31)

> "*No measurement error*" plus "*No unmeasured confounding*" equals "*Not epidemiological data*"

Judea and I agree about quite a lot of things I think, (slide 32) and one thing we do agree about is the genius of Sewall Wright. I'm going to say just a few things about Sewall Wright. It's very difficult not to like him. He wasn't a very sociable man, quiet, but a genius. (slide 33) James Crow's National Academy of Sciences Biographical Memoir of him says

> *"He read his father's math books and learned to extract cube roots before entering school, a skill that he said brought him instant, lasting unpopularity with the other students".*

Now that's something that probably some of the people here can equate with.

(slide 34). Here in an admiring review of 'The Book of Why', picking up on things which are stated in it,

> "*Wright's work was vigorously and explicitly discouraged by the thousand-pound gorillas of Pearson and Fisher" ...*

This isn't true. I mean Pearson never commented on Wright's path diagrams at all to my knowledge. Fisher didn't disparage path diagrams as such, and Fisher and Wright came to similar "analysis of variance" conclusions. Fisher in 1918, in a paper which Wright was ignorant of when writing his paper shortly after, Wright certainly came independently to this, but Wright came to it with his path diagrams, Fisher did it with no diagrams at all. And they came to the same model. I think Sewall Wright is a genius, but Wright is the one who made a mistake with his path diagrams, with respect to dominance, and not formalizing the fact that parent-offspring and sibling offspring quantitative

associations will be different because siblings can share dominance to a greater extent than parent-offspring.

(slide 35) So, now in 'The Book of Why', Judea talking about Wright's work says that

> *"A rebuttal published in 1921 by one Henry Niles, a student of American Statistician Raymond Pearl (no relation), who in turn was a student of Karl Pearson, the godfather of statistics".*

So the suggestion here is that that Niles' pathetic paper with an attempted rubbishing of Wright was due to Pearl, with Pearl being a student of Karl Pearson. I'm fortunate enough to be a Member the National Academy of Medicine which contains docs and is attached to the National Academy of Sciences, so I know the process of how things used to get published in PNAS. And if you look at the paper that Judea discusses by Wight (slide 36) what you notice is it was communicated by Raymond Pearl. It is impossible that Pearl had anything but the highest admiration for a paper that he communicates. I'll tell you, you do not communicate a paper at the National Academy of Sciences by someone who you want to squash or someone whose work you don't agree with.

(slide 37) Anyway, I'm going to skip this, I'd love to talk to Judea about Barbara Burks, it's a really very interesting story, but I have disagreements with how its presented. I bought my copy of a 1945 posthumously published pamphlet on heredity to share.

(slide 38) This is from Judea, talking about

> *"A prominent SEM researcher once asked me, "Under what conditions can we give causal interpretation to identified structural coefficients?" I thought this colleague was joking. As a faithful reader of Wright (1921) and Haavelmo (1943), I had come to believe that the answer is simply, "Always!..."*

Well I think that would not be Sewall Wright's answer. (Slide 39) Because Wright developed path analysis in this context of genetic analysis, when you have the causal anchoring of either arranged cross-matings. You can also have causal anchoring from measured molecular genetic variants. This is Wright's writing on the genetical structure of populations in 1949,

> *"The rate of decrease of heterozygosis in systems of mating more complicated than self-fertilization was first worked out from the recurrence relation between successive generations independently by Jennings (1914) and Fish (1914) for brother-sister mating and by Jennings (1916) for some others. The present writer, who had assisted Fish in his calculations, found a simpler way of finding this quantity, the method of path coefficients, based on the correlation between uniting gametes (Wright, 1921)."*

Sadly the notes that Wright took when he was doing his work for Fish aren't available, but he clearly developed path analysis based precisely on systems of mating. (Slide 40) And of course the famous figure introducing path analysis have genetic anchoring.

(slide 41) And what he says so often, - there are too many of them to show

> *"It was of course realized that the "concrete, phenomenal actuality" of the results was not proved by the analysis by path coefficients" ...*

It was absolutely explicit that path coefficient analysis just produced standardised regression coefficients as we call them now, which are just about associations.

> *"... This rests on the validity of the premises, i.e. on the evidence for Mendelian heredity. The paper began with a quotation from East and Jones on the universality of Mendelian inheritance on the sexual reproduction, as a justification for the analysis".*

Or a couple more cases

> *"The hypothesis that heredity is Mendelian may usually be used safely as information external to a system of correlations among relatives"*

This is the "background knowledge"

> *"... external information of a most precise sort is provided by the pedigree and the practical universality of Mendelian heredity".*

Virtually all of Wright's path analyses were genetic, in the papers where he reviewed path analysis he included I think three or four non-genetic examples, but they were ones when you had strong biological knowledge. If you're interested in Wright and his development probably the best place to start is the long exchange and two papers by Wright and by John Tukey in 1954 in 'Statistics and Mathematics in Biology' a compilation. (Slide 43) And interestingly, Tukey by the way thought of the three step ladder of causation, he talked about three steps in the ladder *"… from description to tangential ..."* which was local causation as it were, and then *"to functional"* when you could extend beyond that. And Tukey also pointed out that one of Wright's very few non-genetic path analyses - when he was looking at birth weight in guinea pigs, litter size and gestational age - Tukey actually drew a path diagram. There was what we would now call a potential collider in that situation. He drew a path of what he was talking about but he then in the text describes that as a reason why you might not trust the findings.

(slide 34) So Wright's path analyses were almost entirely based on you having genetic anchors. And in a paper which I disagree with quite a lot, but it's a fun paper to read – Gutman's 'What is not what in Statistics' he says

> *"Path analysis does not analyse non-genetic paths"* – I love this!

> *"Genetics has but one modest framework for paths. In contrast according to current journals sociologists keep discovering new fundamental path frameworks every month; and sociological graduate students are required routinely to hand in, as individual class exercises, new discoveries equalling Gregor Mendel's."*

So it was recognised a long time ago that this validity entirely depended on Mendelian heredity.

(slide 45) And here's a letter from Egon Pearson to Jerzy Neyman, and I am sure all of you will know of these Neyman-Pearson inference, which is certainly not what we think of as Bayesian. Egon Pearson writes

> *"However, by 1929 I had come down firmly to agree with Fisher that prior distributions should not be used, except in cases where they were based on real knowledge, e.g. in some Mendelian problems."*

So Wright, Neyman, Pearson, Fisher even would agree that in those genetic situations you have some foundational knowledge to base things on. (Slide 46) So: for the inference engine, where does the

"background knowledge" come from?" (slide 47) Now as Judea explicitly says in this paper, background knowledge is a set (slide 48)

> *"of qualitative causal assumptions that the investigator is prepared to defend on scientific grounds"*

Our aim in causal inference is to produce knowledge that we can then move forward with. You need to remember that DAGs are recursive graphs, i.e. you know these are graphs where the output of one can become the input of another. They are recursive systems. (slide 49) and here with respect to causal diagrams in Judea's 1995 paper - which I very highly recommend, I find you always learn things from going back to early papers when things are being actually spelt out -

> *"The basic limitation of the methods proposed in this paper is that the results must rest on the causal assumption shown in the graph, and that these cannot usually be tested in observational studies."*

I strongly agree with this

> *"In related papers (Pearl, 1994a, 1995) we show that some of assumptions, most notably those associated with instrumental variables, (see figure 5B), are subject to falsification tests."*

Which I entirely agree with.

And here (slide 50) from Sander Greenland's very highly recommended paper 'Overthrowing the Tyranny of Null Hypotheses Hidden in Causal Diagrams':

> *"As with regression models, causal models in observational health and social science (OHSS) are always false..."*

So all causal models are false. There's always something to think of that could go in, but the question is are they false to the degree where you are going to get the wrong answer and the wrong inference?

> "*Because we can never know we have a correct model (and in fact in OHSS we can't even know if we are very close), to say G is causal if unconfounded is a scientifically vacuous definition: It is saying the graph is causal if the causal model it represents is correct."*

In 'The Book of Why' there's a blunt statement that there isn't circularity in its reasoning I don't think this is so; it's not a fatal flaw, but I think there is circularity and if that is acknowledged, this is more useful for health scientists trying to use DAGs.

(Slide 51) There's a sense in "The Book of Why" that there is a recent causal revolution. In the preface Judea refers to "returning the Causal Revolution to its womb in artificial intelligence" which essentially dates its origin to around the time he entered the field. When I was at University College London in the mid 1980s the one book that there were three copies of (slide 52) - I nicked one of them – from which I learnt about path diagrams was Asher's 1976 book on 'Causal Modelling'. (slide 53) And in fact path diagrams were tried out in epidemiology. I went back and flicked through the early International Journal of Epidemiology issues and there was a rise and fall, so as they entered sociology from the 1950s and 60s they were tried in epidemiology, and I think their use abated because people found that without causal anchors they weren't producing what one wanted.

(Slide 54) I'm now going to get onto the one issue I know that Judea and I quite strongly disagree. And this is about the position of randomized controlled trials. Hence "consequences of believing the DAGs", my last section, you'll be very pleased to hear.

(slide 55) Judea introduced the "front-door criteria" which I think is a very clever notion. Regarding the model on the slide, as Judea says David Freedman and others said years ago it was unlikely to be veridical. I've spent a lot of time trying to think of an example where the front door criteria will be useful and could work, and whilst I think it's very clever, I haven't come up with one. There are, it has to be said, very limited examples of the front door criteria being applied and demonstrating effects that one believes. But in 'The Book of Why' Judea says (slide 56)

> *"RCTs are considered the "gold-standard" of causal effect estimation for exactly the same reason. Because front-door estimates do the same thing, with the additional virtue of observing people's behaviour in their own natural habitat instead of a laboratory, I would not be surprised is this method eventually becomes a serious competitor through randomised controlled trials".*

So this is about a method for which there are currently no robust demonstrations - even in proof of principal settings - of it working. (slide 57) in the Turing Award Winners speech Judea says

> *"The most significant practical impact of the Causal Revolution would probably be a continuous erosion of the supremacy of randomized clinical trials (RCT) in the development and evaluation of drugs, therapeutical procedures, and social and educational policies.*

And this is something that I find concerning. (slide 58)

> *"Under the 21st Century Cures Act, the Food and Drug Administration is tasked with developing a programme to evaluate the use of Real World Evidence (RWE) to support approval of new indications for approved drugs or to satisfy post approval study requirements. RWE can be defined as the clinical evidence regarding the usage and potential benefits or risks of a medical product derived from analysis of Real World Data (RWD). A framework for this program will be published by the end of 2018."*

The issue is, as I'm sure people here know much better than me because I'm not from the US, there is a huge push towards deregulation. The Trump administration is appointing ex-marketing guys of medical devices into serious regulatory positions, and the absolutely last thing I think we would want to do as health scientists is in any way suggest that is acceptable. This is probably the only serious point - well a few things are serious - but I really think that anything which feeds into the notion that you can get cheap RWE evidence which, nicely, won't find out detrimental effects of metal on metal hips isn't good.

(slide 59) And here in the paper I was recommending from 1995, Judea says in regard to a comment by Paul Rosenbaum that

> *"Rosenbaum mistakenly perceives path analysis as a competitor to randomized experiments".*

I agree with the 1995 version. I was going to skip this because I've been going on more than half an hour. (slide 60) Here is Judea commenting on Deaton and Cartwright, for example, writing a critique of randomized control trials, which I think will be damaging. I think sometimes their statements make it sound as though this RWE framework can do more than it possibly can do.

(slide 61) From the primer,

> *"It proves the enormous, even regulatory, power that causal graphs have in not merely representing, but actually discovering causal information."*

is ambiguous, suggesting that the causal graphs can discover causal information. Something which I think is not the case except when you've got very clear causal anchors, like genes.

(Slide 62) Regarding this language, Levine writing in Epidemiology in 2009 says

> *"George Orwell wrote that language could be used to give the ". . . appearance of solidity to pure wind." It is disturbing that the language of "causal modelling" is being used to bestow the solidity of the complex process of causal inference upon mere statistical analysis of observational data."*

I agree with the Levine, in terms of the language. Even though it's repeatedly stated that these are causal models only if your assumptions hold, that gets lost and as we saw sometimes statements are made which go way beyond that.

So I have to give my conflict of interest before I am going to hand over to Judea: my conflict of interest is that I'm old and time expired. (Slide 63) This is a letter Judea wrote in response to a paper I wrote with Nancy Krieger. He said that it was the

> *"dazzling speed with which epidemiology has modernised its tools which lies behind the authors' discomfort, and that it will subside as soon as researchers gain greater familiarity with capabilities and flexibilities of the new tools."*

So here's my conflict of interest from published version of my talk yesterday, this is the beginning of the last paragraph, citing Judea, I say

> *"As has been suggested, the views I express here may well reflect the last spasms emitted by a reluctant and diminishing group refusing to recognise it's superfluousness".*

Thank you.