

**LECTURE****Social inequality: The embedding of adversity across the life course**

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We know a lot about social inequalities in health. These inequalities are surprisingly persistent, are surprisingly large and their failure to disappear over the last 50 years is a major topic of concern. As far as socioeconomic inequalities is concerned, there is nothing special about health. We can look at trust, we can look at certain kinds of social behavior, we can look at participation in democratic institutions, we can look at a whole load of things, and you will see of much the same picture.

If you board a metro train at Newcastle Airport, you are getting on amongst people who live in the surrounding area who have an expectancy of healthy life of about 75 years. If you travel the four kilometers to a place called Fawdon, that has already dropped by eight years. And if you go on a few kilometres through the city center to a place called Byker, you have lost a whole decade of healthy life. This is just an observational finding, clearly there are issues of selection and causation and what causes people to live in the neighborhood they live in. We cannot say that neighborhoods are causal, but for whatever reason, or whatever combination of reasons, the people in Byker are going to be sick by the time they reach statutory retirement age on average. The people who live out in Ponteland, south of the airport, they are going to be going to the tennis club for another 15 years. So why? This is a city with free health care, this is a city with – by historical standards – high absolute levels of income, these people all come under the same municipal authorities, they – in principle – have access to the same public services, and yet there is this huge discrepancy in the outcome.

In the briefing material for this symposium, we had the stimulus question: “which research questions concerning social inequalities need to be addressed?”. We think there is something to do with the social conditions, that there is something about the social conditions of Byker versus Ponteland, that is causal here. And of course teasing out causality from other kinds of processes, like reverse causality and so on, is very hard. If we use these things like composite socioeconomic neighborhood score or household level socioeconomic position variables, that do not really tell us anything about causality. If there is a causal relationship there, what is it in the organisms’ experience that actually is doing the work? What is it about being poor or from a poor neighborhood or of low education that is actually making a causal impact? And there you need to put yourself in the place of the individual human organism. What do they see? What do they hear? What do they care about? What are their emotional reactions? And that is hard.

How do social determinants accumulate over the life course? George Davey Smith has mentioned that we need to take a life course perspective on this. Correlations between socioeconomic position and health may not be to do with exposures acting entirely in adulthood, but may also involve the accumulation of exposures that act in early life. But if there is an accumulation of impacts across the course of life, how do they accumulate? Is there something special about childhood? Can we measure the difference in sensitivity between early life and adulthood? And where there are both adult and childhood exposures, how do they interact? Is your final health expectancy

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the simple sum of everything that has happened to you over the course of your life, or are there non-additive interactions between adversities occurring at different times? These are important questions that are not yet entirely solved.

What is the somatic signature of social conditions? If we think that there is a causal link from social conditions to physical health, then you got to talk about physiological mechanisms. Things that we conceive of as fundamentally social, psychological or economic in character, somehow have to be cashed out in physiological terms. That is how you can die from them. So there must be a pathway that links the stuff on the outside to the physiology on the inside. And I do not think environment or physiology or social structure and physiological determinants are either/or choices. These are just different parts of the same problem.

What I will mainly talk about today is the role of behavior. It is really clear that one of the striking differences between the people who live in Ponteland and the people who live in Byker, is that they behave in different ways. Their physical activity is different, their diets are different, their smoking – above all – is very different. This is perplexing, because the people in Byker do not have a lot of disposable income to spend. Smoking is an expensive habit, and yet they smoke more and they smoke their cigarettes more heavily than the people in Ponteland South. So they behave in a different way.

A lot of people have argued that a key mediating variable between socioeconomic position and health outcomes could be what I am going to call “impulsivity”. Impulsivity has a number of synonyms; it is known as “delay discounting” in economics or “time preference” in psychology. This is basically to do with how the individual weighs up behavioral consequences that may materialize over different time courses. Something you do may have an effect today, but other consequences may only materialize in a year’s time. What is the relative weighting you give to those immediate versus deferred effects? That is what this variable “impulsivity” or “time preference” measures. What we do, for example, is we give people a battery of choices: Would you prefer five pounds today or a hundred pounds in a year’s time. Most people say they will have hundred pounds in a year’s time, so then we ask what about ten pounds today? What about 15 pounds? 25? 50? 75? 80? 90? And gradually we find that there is a point where people flip and say “I do not want the larger later reward anymore, I’ll just take the smaller sooner one”.

If you take the deprivation score of people’s postcode and give them a battery of these choices between a smaller and sooner or larger and later reward, you

find that the more deprived their environment, the more smaller and sooner choices they make, in other words, the bigger the payoff you have to give them to make them wait for a deferred reward. They have a preference, a discounting of reward with time, that increases quite consistently with the deprivation of the environment they live in or with household measures of deprivation. This is an interesting finding, because it causes us to collide two views of the problem that are often posed as alternatives to one another, although I do not think they are.

On the one hand, you can say well, these behavioral proclivities, this tendency to prioritize the immediate payoff over the long-term one, might be the independent variable, and health inequalities the outcome. So why do people in Byker live less long, well, they do not care about the future so much, they do not take so much care of themselves, they smoke, they do not eat well, they do not promote health advice. So you can see the behavioral proclivities, wherever they come from, as driving the health inequalities. But on the other hand, from a more structural or social-science point of view, we might say, well, the structural inequalities have a primacy here: the reason why people in Byker do not care so much about the distant future is because they face massive structural adversities. These define the field on which they make their decision. So here, you treat structural inequalities as the independent variable, as the starting-point, and the behavioral proclivities would be the dependent variable or the intermediate variable, then the health inequalities will be further downstream in the causal pathway.

People have strong intuitions that the one or the other of these views is right, often depending on their disciplinary background. It really makes a difference which way we end up thinking about the problem. If you think the behavioral proclivities are the primary thing, and the health inequalities are a consequence, then you tend to favor various health interventions based on giving people information, trying to persuade them and give them incentives to think about the future, or you compel them to opt for the behavior that is in their long-term interest. So changing individuals’ behavior becomes the focus. Arguably, this does not work very well, or at least unevenly. In the case of smoking, for example, we have seen huge changes in the way you are allowed to advertise and market tobacco products. What have done very successfully is reduce the rate of smoking among people whose rate of smoking was reducing anyway, but it has had less impact on the people who live in Byker. The individual behavior change approach tends to open the door that was already opening anyway, and

accelerate the opening of that door. It does not do so much for the populations that are hard to reach.

If, on the other hand, you think structural inequalities are causally primal, then reducing health inequalities becomes an agenda for more thoroughgoing social reform. In other words the question is: why should there be the kinds of structural inequalities that cause people in Byker to care about the distant future less? And, politically, what can be done about them. We need nuanced positions that take into account both individual behaviour and larger structural inequalities.

I will now talk about the theoretical model that underlies my work. As a small aside, in my view, one of the things I think is really missing or perhaps is underplayed in health inequalities research, is theory. In science, what we need more than anything else, is theory, and I think, occasionally – my esteemed co-speakers today are certainly not guilty of this charge – what happens in epidemiological research is the idea that data will haul us over the line, if we just collect more data, more populations, more variables, and, ideally, adjust for more things in our vast regressions, that will somehow haul us all the way to explanation. But it does not. You have explanations in science, these must be based on theories: theories about human nature; theories about human biology; theories about how the individual organism actually works. Because that is how you decide which variables should be control variables and which variables are actually the variables you should be looking at. This is just an old-fashioned plea, really, that in our excitement for all the things that we can measure, we do not forget what are the theories we are trying to test, because the notion of theory is very closely related to causality, and if you are going to design better policies, it is theory that is going to get you there. Only theory can motivate investigation of which policies you should try.

I will now present a possible way to conceptualise the problem¹. First, we have structural disadvantages: these are unmodifiable exposures that just come with your socioeconomic position. For example, if you are very poor, you cannot choose the air quality of the neighborhood you live in. If you are rich, you can do that. I argue that these lead to important attitudinal shifts. If stuff has happened to you that you fundamentally cannot control, exposures that are going to be bad for you physiologically that you cannot opt out of, that leads to what we are going to call “contextually appropriate responses”. That is, responses that make sense, given your inability to control your exposure to disadvantage. Unfortunately, these “contextually appropriate responses” have a secondary side effect that, while they may make a certain

amount of sense given the conditions under which you have to live, they actually you make the disadvantage worse in the long term. Smoking is a classic case. If there are uncontrollable disadvantages in the environment to which you which you are exposed, cutting down on something that really only kills you after you are 65, might quite rationally not be a high priority. So you are more likely to continue to smoke, because you simply do not see the benefit of the behavior of quitting. However, that does of course feedback and make your health worse. And it is very expensive and has all these other unfortunate side effects, which actually, via this feedback loop, means that you overall burden of disadvantage is even greater than the disadvantage with which you started out. So you end up with feedforward cycles where small initial disadvantages in exposures get exacerbated by people’s shifts in behavior. That is the way I try to have my cake and eat it about which of the two causal priorities I alluded to earlier is important. But I give a primacy to structural factors, because without structural disadvantage, there is nothing to be exacerbated by behavior.

I published a simple model of this process a number of years ago². In this model, I say that “look, you are an organism, you have to decide how much to invest in life-extending health behaviors that are effortful in the short term?” In the short term, they detract from other things you may be doing, but in the long-term, they are going to make you live longer. What is the rational level of investment in such behaviors? You are trying to maximize some kind of summation over your life-course of all your various pleasures and pains, so if you are going to do something that has a short-term pain and a long-term gain, the long-term gain must be bigger over the life-course than the short-term pain. Crucially to this model, if you invest maximally in healthy behaviors, if you do everything you possibly can to not smoke, have a good diet and so on, there is still some bad stuff that can happen to you, and it happens to you with a certain probability m in any period. These are the structural things you cannot control: you might get hit by a tram or struck by lightning. The question we want to ask, is how does variation in m , the probability of bad stuff happening to you over which you have no control, affect your rational investment in things over which you do have control? The finding is that as m – the probability of uncontrollable bad events happening to you – increases, obviously your overall life expectancy goes down. But the optimal level of health behavior that you should adopt rationally, also decreases. Why is that? Well, because what we think of as healthy behavior, are investments in very long-term outcomes. Typically, they are benefits

that are deferred into the distant future. And in an environment where there are uncontrollable shocks to your life that you can do nothing about, the probability of that benefit ever materializing is necessarily reduced, simply because that long-term future, for reasons entirely beyond your control, is less likely to come about. So there is a greater likelihood that your investments in keeping yourself healthy will be wasted because something bad will happen to you anyway before you cash in on those investments. Basically, we show that the optimal level of health behavior declined as this rate of extrinsic mortality increases. And this means that, if you think about a population that has inequality in exposure to those extrinsic shocks, there will be a primary effect of that, in that those individuals who live in those neighborhoods or cities that have greater exposures will have a higher total mortality rate because precisely because they are exposed to these shocks. But in addition to that, there will be a secondary effect, that they will disinvest in very long-term outcomes, precisely because it makes less sense to do so given that what they are exposed to, and that will further exacerbate the inequality. To put in another way, if you could reduce these extrinsic shocks, the broader social structural exposures to uncontrollable bad stuff, you would reap a double dividend. Firstly, it is good to do that anyway because it reduces people's exposure. But secondly, people would respond to that by looking after themselves better, because they have now got a greater asset to protect. Thus, we predict a double dividend you get by actually addressing structural inequalities.

In a modest way, we have done an empirical test of this model³. We studied a cohort of 500 adult Americans. We were interested in understanding how peoples' perception of their future might play into their effort in looking after their health. We asked them a slightly different variant of the conventional "how-much-effort-do-you-make-looking-after-your-health" kind of questionnaire. We asked what they thought their chances were of surviving to 75. We asked them to answer in a number of different ways. We asked: "If you make the maximum effort you possibly can in looking after your health, what do you think your chances are to survive to the age of 75?" And we also asked: "If you do not bother, if you do not really make an effort in looking after your health, what do you think your chances are?". So this gives us a couple of interesting variables. It gives us the difference between the maximum and the minimum answer: this is peoples' perception of how much difference is made by trying to look after their health. That can be differently patterned across socioeconomic positions: maybe poorer people think it will

make less difference, and that is why they make less effort. We can also look at the difference between a hundred percent and your chances of surviving to 75 if you really try. That gap tells us what you think the chance is of bad stuff happening to you in a totally uncontrollable way, beyond the scope of behavioral control.

We use people's socioeconomic position to predict the gap between 100% and their expected survival if they really try; and the difference between their expected survival if they do and do not try. First of all, not terribly surprisingly, the higher peoples' socioeconomic position, the greater self-reported effort they had in looking after their health. But interestingly, the other thing that varies strongly with socioeconomic position, is not how much difference it would make to look after your health—that was about the same—but your chances of dying anyway, even you really tried to look after yourself. That is the big difference between socioeconomic positions: Poor people think that even if they do really try, they are more likely to die anyway before they are 75. As you go up the socioeconomic hierarchy, more and more of your behavior is perceived to be within the scope of your behavioral control, in the bit that is up to you, according to how much effort you put in. The relationship between socioeconomic position and health effort is fully mediated by your perceived chances of dying anyway, even if you really try to look after yourself. Thus, if people think they are going to die anyway, they do not try so hard. And that makes perfect sense: why would you try if you thought that despite your best efforts, bad stuff would happen to you anyway?

We do not know whether these perceptions reflect reality, but certainly it seems that on the perceptual level, what characterizes people from lower socioeconomic positions is that they think that even if they really try, there are overwhelming obstacles beyond their control. There's a famous cartoon that nicely sums up this idea: two soldiers in a warzone with bombs flying over them, one saying "don't you listen? Sarge says drugs are dangerous!" We describe this as a "contextually appropriate response" perspective, because we are emphasizing that if it were the case that a lot of bad stuff beyond your control, it would actually make a lot of sense not to worry about the temporarily distant outcomes so much. This perceptive really differs from a widespread view out there that basically says that "the poor get stuff wrong, they get stuff wrong because it is stressful and horrible being poor, and as consequence poor people make a lot of mistakes". Ours is a very different view from that. I am not suggesting that stress does not negatively impact our cognition; it may do. However, we

are making a rather different argument. Our argument relies on the idea that adverse social conditions entail unavoidable harms and damages, and people respond to that in their decision-making.

I believe this view has implications for policy and for interventions. It correctly predicts that many health interventions have the biggest effect in the people who need them the least. There are people who are looking for ever-better ways of looking after themselves. When given a little more information, they really seize on it. There are groups of people who just feel that an only slightly better way of looking after themselves is of limited interest: our contextually appropriate response perspective sheds some light on why this might be the case.

I also believe this way of thinking can inform our causal interpretation of epidemiological patterns. When we observe correlations between health behaviors and biomarkers of health, we rather readily assume that poor behavior leads to poor health. Our perspective also suggests there can be causal arrows in the other direction: if your environment or your state is poor anyway, then there is little incentive to make an effort in looking after health. Thus, the damage may come first, and be caused by the environment: the behavior could be a downstream consequence. We should remember this, particularly when interpreting cross-sectional associations.

If we really want to sort out cause and effect in the domain of society and health, then randomized control trials, at scale, represent our best hope and best opportunity. They have to be done as scale because a lot of things work at community level, or at least at neighborhood level. If what is causally important is being around a lot of other people who are not poor, then individual-level random income uplift will not be effective. What is needed is a cash transfer intervention randomized at the level of the neighborhood, or city, or province. Then you can begin to ask about causality in a way that is uniquely powerful.

For anyone who wants to read up on this, the Great Smoky Mountains study is worth following up⁴. It is a natural experiment rather than a randomized control trial. The natural experiment was that a casino opened on a Native American reservation and because of the agreement that was made between that Native American group and the casino owners, an unconditional cash transfer every year began to happen to people who were of Cherokee descent in that community. It became quite a large cash transfer because the casino was very profitable. This basically introduced, if you like, a randomized intervention in half of the kids in the study: their families started getting a substantial unconditional cash transfer.

The results are very remarkable, particularly on mental health and behavior. The prejudice that people have about unconditional cash transfers is that if you give people money, they will spend it on drugs or alcohol. What is really clear in the Cherokee study is that many people were spending on drugs already, and they spent *less* on them when they had *more* money. This is a really nice example of how you change peoples' behavioral priorities by making them richer. They just did not consume more of the stuff they wanted anyway, they actually changed their preferences in an interesting way.

Finally, I wanted to say a little on the accumulation of influences over the life course. It is becoming increasingly clear that things that happen in early life matter and that that may affect the modifiability of social determinants in adulthood. This is a question that needs to be studied longitudinally and in an intergenerational way. Here too, we need some theoretical understanding. The best theories we have in life-course epidemiology basically say that all the stuff that happens to you over the course of your life matters. This is true, but hardly constitutes a theory. We need casual models of why things accumulate over the life-course in a particular way. The general finding seems to be that bad stuff that happens early in life and bad stuff that happens in later are not additive, but super-additive. In other words, the effect of being poor in adult life is worse if you were poor as a child. It is not that you somehow adapt to poverty because you got these early life experiences that weather forecasted your eventual poverty and you managed to develop a resilience; just the opposite. If you have a bad start, a bad continuation is even worse. The best explanations of why this might be are based on the idea of somatic redundancy. If we think of a structure like a suspension bridge, the roadway is bound to the towers by using many cables. The consequence is that you can come along early in the suspension bridge's life and cut away half of the cables and nothing bad would happen: it would not fall down. So you can have two bridges, one where you cut half of the cables and one where you do not, who for years will behave exactly the same. But you wait a hundred years, until the cables start to wear thin and break spontaneously. Suddenly, there will start to be a massive difference between your two bridges, because what early life has done is eat into the engineering redundancy of one of these bridges but not the other. This is how we have to think about bad exposures early in life: they eat into a lot of our psychological and physiological redundancy. You do not initially see any difference in the phenotype, but then you add the effect of adult adversity or age – which is a kind of adversity really, just the accumulation of bad stuff over time – and then you

suddenly begin to see these big differences according to early exposures. This unfortunately means that you can draw the wrong conclusions from short-term follow-up studies about childhood social policies. You can say: “There is a no real difference between my groups”. Well, you wait 50 years until these people start to age or wait for an economic recession when these people might get differently exposed to bad stuff, and then you might start to see that the childhood exposures were enormously important, but it was latent. When you are young and vigorous, you have enough engineering redundancy for vulnerabilities to not immediately surface in patterns of ill-health.

As a parting thought, we can now measure internal aspects of the organism beautifully in a way that we never could before. We can measure genetics, we can measure all kinds of bio-markers. But let us bear a thought for the fact that we are not measuring the environment nearly as well. If you actually care about explaining causal impacts of societal and environmental exposures or disadvantages on people, we need to measure the social environment better, and we need to measure it at the scale of the individual organism. We use summary variables like socioeconomic position, social class and so on, but what do they actually mean in terms of the lived experience of the individuals? Because for them to have a long-term physiological effect, they must get internalized by something. There are imaginative ways of doing much better than a once-in-every-fourth-year questionnaire to your participants: we should actually study, increasingly in a way that we could automate and that we could mine using big data techniques, the temporally fluctuating lived experiences of participants. For example, what may matter is not your

average level of hunger or pollution exposure, but the peaks of it. With a conventional static questionnaire, we never see that. But if people were reporting many times and we were capturing their experience in more imaginative ways, which there are all kinds of technologies that let us do now, we could see the temporal fluctuations in experiences, the peaks and troughs, the variances as well as the classic static measure in one time interval.

With that, I will stop and thank you for your attention.

Notes

1. Pepper, G.V. and D. Nettle (2017). The behavioural constellation of deprivation: Causes and consequences. *Behavioral and Brain Sciences* 40: e314.
2. Nettle, D. (2010). Why are there social gradients in preventative health behavior? A perspective from behavioral ecology. *PLoS ONE* 5: e13371.
3. Pepper, G.V. and D. Nettle (2014). Perceived extrinsic mortality risk and reported effort in looking after health: testing a behavioral ecological prediction. *Human Nature* 25: 378-392.
4. Costello, E. J., Compton, S. N., Keeler, G., & Angold, A. (2003). Relationships between poverty and psychopathology: A natural experiment. *Journal of the American Medical Association* 290: 2023–2029; Costello, E. J., Erkanli, A., Copeland, W., & Angold, A. (2010). Association of family income supplements in adolescence with development of psychiatric and substance use disorders in adulthood among an American Indian population. *J Journal of the American Medical Association* 303: 1954–1959.