>> Welcome. You are listening to the UC Davis Center for Poverty Research seminar series. I'm the Center's director, Ann Stevens. This series brings scholars and policy experts from around the country to discuss their work on poverty and poverty research. In October 2012, we hosted Douglas Almond, the Center's Fall visiting scholar.

He is an Associate Professor of International and Public Affairs and Economics at Columbia and previously served as Staff Economist of the Council of Economic Advisors during the Clinton administration. As a Fulbright scholar, he studied the health effects of air pollution in China. Here's Almond presenting his seminar, Fetal Origins and Anti-poverty Policies.

>> Thank you. So, so, thank you for that very nice introduction and thank you for hosting me Ann and Marianne this week. It's been a great visit so far, and, you know, it compares well with all my other visits, and the one I think of is the one to Beijing ten years ago to NVR conference, where I felt I was treated particularly well.

And your hosting, I think, is, is, is at that, at that high levels. Beijing, Beijing ten years ago. And, and, and just to frighten Hilary a little bit, I'll also add to my sort of CV that I was Hilary's student at at Berkley x years ago, so apologies in advance for this talk Then, so in econ, you, you know the rules of engagement are you're, you're welcome to interrupt me at any time, so please feel free to do that.

I won't, take it as rude or anything like that. And as suggested, I'm gonna sort of do a, an, sort of Ã¼ber discussion of some of the, the recent and important work on fetal origins. Okay. So by way of motivation, obviously, I think this is an audience where we, we think that childhood is very important.

And there are lots of nine month periods in our life that are very important. So, a month ago, our daughter Abigail started kindergarten at the Columbia school and she has an awesome teacher. And she's learning a lot, actually she's learning much more than I thought she would. I wasn't sure if she was gonna buy into it, but she's really coming home every day, with, with, with something new she's learned that she wants to tell me, so this, this is gonna be a very important nine months.

What I think the Fetal Origins hypothesis has asked us to do, is to think about five years ago, because she's actually starting kindergarten, age four when she was in utero. And how important was that nine month period relative to this, very important neni month period, right now. And so this is the picture of, of her in, in utero.

okay. With consent of them, I'll tell you So the Fetal Origins hypothesis basically says that the prenatal foundation lays the foundation on which the rest of childhood is built. And I saw this definition recently. which, which I liked from Seco, which is, referring to, fetal programming as the action of the factor during sensitive period or window, of development to exert, organizational effects that persist throughout life.

Okay, so something, so, fundamental architecture is being affected. It has the next point repeats these outcomes persist throughout the rest of life. So, with you know, many other interventions people are, are concerned about fade out over time. And fetal origins is, is a place where, from the get go.

Where is thinking less about fade out, partly, for, just the practical reason is the early analysis are looking at stuff that happened very early on and then looking at adult outcomes. So,sort of while there may be fade out, it's not going to be complete fade because we're still finding effects in adulthood.

In epidemiology and we discussed sort of the, the role of the epidemiologist, epidemiological literature more in this JEP article that Ann nicely mentioned. It's often, often associated with a physician named DJ Barker and he did a lot of sort of original data analysis in England looking at a correlations and regression adjust to correlations between measures of early life health and later life chronic health conditions.

This work by Barker some 20 years ago was actually a reaction against an earlier literature which viewed the fetus as the perfect parasite. So, basically, if there was adversity during the prenatal period, the mom was a very good buffer,

and she sort of absorbed the bad event and, and protected the fetus.

So you know smoking rates into the 1950s and 1960s in the US were something like, you know, going up to around 50% during pregnancy. And it was, it was by many people's reckonings, the thalidomide episode which sort of shattered this notion that the mom is protecting the, the fetus from, from, from, from every insult.

So a natural question is what matters, to the fetus and its long run outcomes in utero, and the original hypothesis that came to us again, from epidemiology focused on prenatal nutrition, and how much and what the mother eats. The most studied famine, in, in poet book economics and epidemiology is the Dutch Famine of 1944 to 1945, and there are a couple of reasons why this famine was sort of particularly appealing to analyse.

One is there was a fairly short famine and, and fairly unexpected. So, you know, the Netherlands did pretty well, despite being occupied until the Nazis cut off food shipments. And then there was a sudden, sudden and unexpected famine. And furthermore, it happened in a county that actually had good data.

Usually we had famines in places where the data are pretty bad. So, those are couple of reasons why these cohorts have been particularly studied, and what do they find, they find a whole host of, chronic health conditions in adulthood that are compromised. The initial study is looked at cognitive outcomes among military recruits and that was back in 1975 and didn't find much.

But as these cohorts have aged they've found heart disease, hypertension, obesity, all these things responding to the prenatal environment. One sort of piece of subtext here that I"m probably more interested in then, than other people is this literature has been actually pretty contentious when in epidemiology. And one of the reasons why I think it's been contentious in epidemiology is the role of birth weight.

Okay? So the cohort that seems to be most, negatively affected by the Dutch famine tends to be those exposed during the first half of pregnancy, whereas when you look at the birth outcomes of the Dutch famine. The ones with the largest decrease in birth weight are those exposed in the second half of pregnancy, so there are a lot of people who really thought that the dutch famine, sort of the, the, exclusion restriction should hold for birth weight, that birth weight should pick up.

The long run effect of famine, and that wasn't being found, so granted, they have a good natural experiment, but people are sort of kind of confused at why birth rate, wasn't the, wasn't the, the, the metric to capture that. And I think now over, just over the last couple of years, people have said, well, you know, this sort of says, there's a need for other metrics, and that's actually going to be.

A point that I come back to a couple times later. Is, a real challenge in this literature is, is the absence of a good early life metric. Because we're forced to look at these you know, if we're gonna, if we're really gonna analyze long-run effects, we have to go back in time to see, look at the historical event or treatment, back in the past.

If we had that metric, we wouldn't have to be doing economic history all the time. Okay? But, in the absence of such a metric, we're, we're, sort of, forced to do that. Oka, so, now I'm gonna, sort of, wave the Economics flag a little bit, here, What are the contributions economists have made to to this field?

So I'll, I'll sort of try and distill these into, to three and then talk about more recent work. One is to say it's not just about famines. It's more general than thinking about famine exposures. Not to say famines aren't important, and ongoing. And then, you know, obviously they're very important for the history of human evolution, et cetera.

but, but economist have sort of, in some sense been a little bit lit-, liberated from the, from the Biology, to consider other things, that, that the epidiem-, epidemiologist hadn't really posited mechanisms for. Secondly economists are very interested in things other than health, so actually the original incarnation of my job market paper was looking at health outcomes, and then I sort of thought, you know it would actually be even more interesting to consider things like wages and that sort of thing.

and, and in some sense the data in the census were particularly well suited to that. So, expanding beyond health to consider a whole manner of other things. And then the third thing which, you know, probably I'll talk about more than I should is about the convincingness of, of the evidence.

So I'll, I'll briefly develop these three points before proceeding to sort of some of the more recent literature. So. Famines effects, you know, famines are a very severe event and in some sense, if you do something sufficiently bad to a mom while she's pregnant it's kind of plausible that there would be an injury, okay?

And so what economists have done is considered things that aren't quite as severe as famines and so one of the things economists have looked at is the disease environment. And so looking at you know pandemic influenza and malaria exposure a war time stress, ionizing radiation, a whole gamete of early life exposures beyond famine have been studied.

So it seems to be more general than, than just about famine. Secondly as mentioned, people have looked at a lot of other outcomes besides just health outcomes. Not to say health isn't important, but things like test scores. So,. A, a whole literature, you know, obviously is concerned with educational inputs and, and impacts on test scores.

Well, you know we can sort of engage that literature a little bit by saying, you know, what are the effects of income, earned during an early childhood on, on test scores, or that sort of thing. And the thing that gets many education kind of people, kind of upset.

Maybe not upset, upset is not quite, quite the right word but is that the impacts tend to be often as large or larger than conventional educational interventions, okay? So, despite the brevity of the prenatal period, you can really have some outsized effects on things like test score. Growing beyond test scores to education attainment wages and incomes.

And then other sort of more demographic stuff like, whether you get married, the characteristics of your spouse, and the neighborhood you live in, welfare dependency. Just, it, it actually, in, in some sense it becomes a problem, this big range of things that are effected because it sort of restricts the set of falsification exercises we can perform because it's not so obvious what should not be effected when you sort of when you, when you trigger something, during the prenatal period.

Okay, then the third thing is, like why should you believe this? This sort of sounds like astrology in the sense you're born at this particular time under a certain star and then this is how your life is going to be. And. It, it's not just that. I think there is a lot of astrology caliber research out there on the fetal origins hypothesis.

And and, you know, people have heard this before, but I would say, you know, the modal study in fetal origins is equating correlation with causation. And you know, part, you know, that, I think that generated some of the controversy within epidemiology and you know, it, at the risk of being just pedantic, you know.

It, it's not just correlation is not the same as causation. You can do regression now, it says, with a ton of control variables. And still I'm not very convinced that we're getting close, closer to sort of causal inference there. So the paper that I would like to plug here just randomly.

And I feel kind of comfortable doing this, because this is not actually by an economist. Just by what the title is. Is, you know, if you're in a situation where you think you might have admitted variables bias, does including more regressers help reduce that bias? And I think you know, the, the, the, the, the usual prior on that is if I control for more stuff I reduce bias.

And this, this, this paper makes the point that that's that's really not the case, okay? You can actually often increase bias by sort of kitchen sinking your regression. And what they point to and what, what I'm going to turn to next. Is, you know, you like to be in a situation where what you control for really doesn't matter much to the effect you're estimating.

And so research design is sort of an alternative to sort of you know, kitchen sinking our regression. Okay. So, example

one and I apologize for taking these examples that are close to home, is the, is the 1918 influenza in the United States. So, one of the things that that is notable about the 1918 influenza is it really appeared out of nowhere.

This is September 1918, number of deaths per month and then we, you know go up by more then an order magnitude in October of 1918. So this is just something that, that hit suddenly and out of the blue and after, you know, it was a really scary four or five months and you know, hundreds of thousands of people died, but after four or five months you get back down to sort of fairly normal death rates in the US.

Okay, So, if you think about the cohort that was, you know, born, say around here. The, sorry not born around here. Born around here, they were conceived prior to the arrival of the pandemic. So we don't think that they're, they're, they're conception decision was affected by expectation of this flu but but they were very likely to have a mom who got infected with the 1918 flu.

So while it is the case that millions died from the 1918 flu that's actually not the most common thing that happened. The most common thing that happened is you got infected, you were sick for a little while and then you. You got better, and you lived. so, something like one third of pregnant mothers got sick whereas fewer than 1% of mothers died.

To the extent that mothers did die or, or, or weak fetuses died, that's actually gonna bias against us finding effects. So there is a calling effect that we need to have sort of floating around particularly for famine episodes where mortality rates are even higher. But that's, you know, to, you know, at least at first pass, that's gonna bias us against finding anything.

So the question the the flu paper asks is, if you happen to be in utero in the U.S. during the 1918 flu and conceived prior how are you doing later in life? Well, so, this is where the census data in, comes in. And so, if you look at the 1960 census data, so this is 42 years after the flu pandemic this plots the average educational attainment by the year of birth, okay.

And there's this upward trend. So, people in the US were getting more education over time. Part of that, I think, is due to the compulsory school laws. They're inducing you know, later-born cohorts to have more schooling. But the striking thing about this is, you can sort of do the, the visual de-trending; you see that to cohort born in 1919 is substantially off that trend, okay?

And we can do a couple of additional comparisons. One is we can sort of dummy out this, this average drop for the 1919 cohort and look using the information on where people were born to see if in a difference-in-differences analysis if being in a, being born in a state that had a worse pandemic leads to a larger decrease here, so a bigger drop here than if you were in a, a state with a milder pandemic.

So, you know, that, that helps to address concerns about oh gosh, it was the end of the World War II, World, sorry, World War I and maybe there were other changes. So those changes would have to be correlated with the severity of the pandemic around the US, which fortunately for analysis purposes was pretty, pretty idiosyncratic.

We can also carry this forward to 1980 when these guys are in their early sixties, and we see about, you know, 20% more likely to be disabled if the mom was infected with flu and greater than 5% reduction in wages, okay. Example number two is looking at Chernobyl fallout in Sweden.

So, again we sort of have a similar figure here that this is the gamma radiation in sort of, central eastern Sweden plotted daily 1985, and 1986. So what you see again, is this huge spike that's, you know, shorter than the spike for the, for the 1918 flu. And we can think about those cohorts that were, happen to be in utero at the time.

Now Sweden wasn't considered a Chernobyl hotspot. Yeah, sorry.
>> You know, you, when you first introduced the flu, you know, you, you, you were careful to say that, the, the, you know, many folks would many of the affected kids would already, the fertility choice would already have been made.

>> Yep.
>> And that's real helpful.
>> Yep.
>> For getting the convincing part right.
>> Yep.
>> But, and I'm, you, you know, you may be talking about this later, but of course, you also need to worry a bit about survival to birth.
>> Yep.
>> and, how that might play in, in any, and, you know, it's a challenge in, in most any of these kinds of empirical settings that you're looking at.

>> Yep.
>> I wonder whether you might wanna just talk about that challenge or whether you're gonna talk about it later.
>> Right so that's exactly right. So, so I think the, the more plausible bias, as opposed to people anticipating it-
>> Right.
>> Is that there's a selection effect.

Fetuses are very vulnerable.
>> Right.
>> And so, you just have part of the distribution that's getting sucked out by the event itself, and then we have sort of a survivor bias-
>> Right.
>> Later on in life. Okay, so the, let me, let me give sort of a, a, a half answer to that.

The, depending on the thing considered, we may not have the data to look at fetal mortality and that and, and particularly during 1918, that's tough. And so what people have done and what we've done is to look at the number of births. So basically, if you account for sorta smooth trends in, in, in number of births we should see a smaller cohort.

You know, if, if say fetal mortality peaks, you know, in the first trimester or something like that, then, then we should see a smaller cohar, cohort. It turns out these things seem to be, this in particular, seems to be sufficiently mild where we don't get fetuses vanishing. Where, where that will change, and I'll come to that in a second, kinda surprisingly is Ramadan.

You see, you see a lot of, of, of missings there. So maybe, maybe I can come back to that a little bit, but that, that, that's exactly right. I think that's, that's much more in particular, fetal mortality because you know, obviously childhood mortality can increase as well, but that, that base level is so low that you're really stretched to generate, you know, much of an effect with, with childhood mortality.

It would really have, I, I think the thing that I usually worry about is the fetal mortality cuz those rates are just much higher.
>> Right.
>> Yeah, yeah.
>> Exactly, and a lot of people talk about the fertility piece
>> Yeah.
>> Which I think, for the most part, time and time again.

>> Yep.
>> We reject that that's what's changing.
>> Exactly, I, I totally agree. And yes, so, so, so that's right. And we'll, we'll come back to this a little bit with Ramadan. With, with, with, particularly with this, we're pretty confident we don't, we don't have to worry too much about that.

We, we, we, I think the intuition still generally goes through-
>> Right.
>> That those that are most likely to die off are the coming from the bottom of the distribution. And so that's gonna, that's gonna lead to attenuated effects in general, yeah. But, but we'd like to know what those are, right?

Like, cause we'd. That's interesting in and of itself.
>> Before you switched to the slide at the end of last night, you mentioned you said, if the mothers had the flu. You don't know if-
>> We absolutely do not, so, so, tha, very nice catch, yeah.
>> So one thing that wouldn't have been differenced out in a state-by-state or region-by-region difference-in-difference, mod research design is stress on on medical resources because, so it might be that the resources are moved away from prenatal care or that people stay away from hospitals because they don't want to risk the infection.

So that would, so I'm just wondering if
>> No, well, you're, you're exactly right and, and, so a related point is, can we say that this was because the mom got influenza? Not really, like it could be just in places where there was high influenza infection rate, it was very stressful to be pregnant.

And it could be that it, that it literally was that. It, had we the data to observe which influ, influenza wasn't a reportable disease. We cannot observe the thing that, which you very smartly caught, which is that we don't observe the individual and to, to see whether it was really those people that were infected that are showing the long-term damage.

That's, that's absolutely right. So we cannot reject those things which mimic the variation on pandemic. Now, prenatal care I'm not so worried about at this time as a confounder because births tend to be outside of hospitals. And and there, I guess I have more questions about, sorta, what prenatal care does.

But stress, I, I, you know, I, I can't, I can't rule out stress for, for 1918 flu. I really can't yeah. okay. Here we have sort of an analygist type of comparison. And what we have in Sweden in which we didn't have in the U.S. is we basically have a lot more data to throw at, throw at the problem.

Okay, so what we can do here is look at things like, is there a heterogeneity by family background. Or what does it look like within the family. Okay, so we can, we can do the comparisons in Sweden of, were you a sibling that was exposed prenatally to Chernobyl fallout while the other sibling was not.

So, that's sort of a departure from the previous work in fetal origins which yes did sibling fixed effects but in the absence of an identification strategy, this is sort of sibling fix fix with identification strategy. And, and an interesting thing, which I'm gonna come back to in a little bit, is those estimates with the fixed effects and without the fa, fixed effects I, going into this, would've thought they'd be the same.

Okay? But that was just sort of just like an extra control variable. And you know, it, it was robust to inclusion of some additional controls. It's actually not the case. If you put in, if you, if you, so, I, I didn't say you find large defects on, on math scores for these cohorts, which lines up to an earlier literature on on the A-bomb drops on Japan and IQ of cohorts sort of around the hypocenter of the A-bomb drops.

And they find the largest effects on, sort of, measures of IQ. Math, math works closely to IQ. We find it at these, these radiation doses that people thought should be should be harmless. And we, we don't see things on, sort of, more English or Swedish or language type things.

It seems to be sort of keeping mainly onto the math. What I was gonna say about the, the sibling fix effects is, I was surprised to see that when we do the sibling effects we actually find much stronger within family estimates than when we do comparisons that also include across families.

And I'll come back to that point in a little bit cuz that was a big surprise, and it was a real puzzle. And what I think it

motivates. and, and, Sweden's not the pla, best place to do this cuz we don't have good data on parental investments that I know of is to think about how parental investments respond to fetal origins.

Okay, so policy implications, Avi Ebenstein, who's another Berkeley PhD. I'm not sure which of my papers he was ripping on. I think it was probably a famine paper. And he said, so what's the policy implication of this? That we shouldn't have famines? Like-
>> Like, we, we kind of, we kind of already knew that one Which, which, this is a pretty fair point, right?

Like, I'm not gonna convince anyone to not have a famine or a flu pandemic because, you know, in 50 years people aren't gonna make as much money. You know, like we already, we already know this is like a policy failure of some kind So-
>> This is sort of the making the rubber bounce.

Like, who, you know, it's not gonna be pivotal for policy, that, that pandemics have long-term effects. so, what that has motivated is the consideration of, you know, less extreme events. So things, things are less extreme and more commonly experienced. so, in a way, this is, sort of, circling back to the early literature.

So, the earlier literature did look at less extreme events. So, like, there were studies of this 1958 British cohort. And, what's its birth weight, and how are these guys doing later in life. The variation in birth weight wasn't that, you know, famine-induced birth weight. This was just, like, cross-sectional variation in birth weight.

So you could think that was the less extreme variation. The problem was, you know, we really, or I at least, really think that, that those types of comparisons really set up affirmative variables bias that birthrates correlate with all sorts of other stuff that's important that, you know, we're not sure we're doing a great job of, of, of sweeping out absent in a, an experiment.

So, I think at this point this, this literature that did use less extreme events, you know, fifteen or twenty years ago and in, was in the absence of the, sort of identification strategy revolution, is viewed, kind of, as descriptive, which isn't to say it's like lousy. It's to say, you know, it motivated peo, a lot of people to work on this.

And so, it sort of, it documents some interesting associations. But to sort of really put the knife in, people have gone to the natural experiments. And the challenge here is, can we sort of maintain these the, the generality of the exposures considered and still have some some identification strategy.

That's the tension, I think. and, and I think that, you know, from my perspective, the, the literature has kinda succeeded in making this jump. It will require some, some, some larger data sets but, but we'll come to that in a little bit. So again sort of being narcissistic and, and perhaps lazy, prenatal fasting during Ramadan.

Okay, the Ramadan fast is determined by daylight hours. And there are various studies that say the, the, total number of calories doesn't change. It's just the timing of nutrition, okay? And the night, you know, I, I'll come to this in a second, but they're, they're, they're nice reasons for considering the Ramadan fast from an identification standpoint.

There's recent work by Courtney Ward. She has a number of papers looking at seasonal influenza, so again, something that's not as traumatic as pandemic. And there's also stuff on weather and, sort of, a host of, sort of, milder, more commonly experienced exposure. I get a, I guess I would say, you know the, the Chernobyl thing might tell us something about radon.

So, like, a lot of people are exposed to radon, and that's pretty low-level, ionizing radiation. Maybe we think that there is some journal to that. So this is, I would say, still a developing literature. But there are there are these, sort of, well-identified studies that consider things that are mild.

They might actually be pivot, pivotal for, sort of, whether we decide to sort of, participate or not participate in you know this type of mild exposure as opposed to something more, more serious. So this is a paper from, I guess like a year ago where we used Ramadan use Ramadan as a natural experiment.

And the, the, the nice thing about Ramadan for analysis purposes is that it moves around the calendar, okay. So I could never remember when Ramadan was, and it turns out the reason why I could never remember when Ramadan was, was cuz it was like, moving 11 days every year.

So we, we know that there's seasonal effects of you know, seasonal effects on health. So birth outcomes, and even long-run outcomes, are differing, differing depending on. When you were born. Well, we can sort of sop out those those seasonal differences and focus on sort of the idiosyncratic movement of Ramadan around the calendar.

And, furthermore, we can also use variations stemming from the definition of the fast as being during daylight hours. So you know, if you're off the equator, unfortunately, Uganda's on the equator, so we don't get that variation, and we do analyze Uganda, but if you're in Michigan or, you know, somewhere else, then you get that daylight variation as well.

We can sort of take an additional difference, and see the defects are indeed larger when you're supposed to be fasting for longer. So we, we do two things here. One is we look at birth outcomes in Michigan. And then we sort of make the jump of looking to other countries that-

And we're looking at Iraq and Uganda purely for data reasons. So maybe this sort of drives home the point of the data challenges with fetal origins research, that you know, you- To implement identification strategy like this, you wanna know, you'd ideally like to know people's religion, you'd like to know exactly when they were born.

Like, you're- Often you just have age. Age isn't gonna cut it. Year of birth isn't going to cut it. You want at least a quarter of birth, and preferably month of birth. And the reason why we're in Uganda and Iraq is cuz they actually report the month of birth on the on the census.

And, and, Uganda reports the religion. So, so, so then, then, then, we can, sort of, move forward from there. You can ignore the bottom. So Esther asked us to do this, sort of, I think, in the spirit of randomized control trials. It's basically, just conduct a balance test.

So if you look over here, the nice thing about about Ramadan is, if you compare those births, where there is exposure so, the effect of Ramadan occurring at any time during pregnancy, on maternal education. It doesn't look like there's any difference. You can look just at Arabs in Michigan, or, or the non-Arabs.

It doesn't look like there's any differential likelihood by maternal education of having a birth during Ramadan. So it doesn't seem like, which may be a sur, actually this was a surprise when I presented at Chicago. People, I don't believe that people don't time their births with regard to Ramadan, well, it doesn't look like they, time their births with regard to Ramadan.

So we get fat zeros here. And so, we're, sort of, we're breaking that link, you know, which is very robust, between SES and health with this natural experiment. So, it does- So, in the end, it's not gonna, it's not gonna matter whether we control for education or not.

Okay? That's sort of that's the game, is just sort of find the situation where you know, what you control for is, is- You know, once you've identified that variation, or isolated that variation that's important, then what you control for it doesn't really matter that much. Nevertheless, we find impacts on birth weight.

Okay? So, you know, these arenâ€™t like honking big noticeable effects on birth weight, if this was your baby or something. But theyâ€™re, theyâ€™re significant. So it suggests that there is, indeed, a first stage that moms are observing the fast. Now one thing to mention, and I actually didn't quite know where this fits in the presentation but I want to flag it nonetheless, is pregnancy recognition.

So maybe its actually good time to mention this, right after having said, what do they think at the University of Chicago. So University of Chicago, I don't think they'd be thinking so much about what are the policies, you know, we

can do to sort of improve fetal health, as much as, you know, how can we give moms the information to make better informed decisions.

And there is evidence from epidemiology that once moms know they're pregnant, their behavior changes. Okay? And the big effects we find for Ramadan are actually, you know, in that, basically in that first month or two of pregnancy. So most of these moms don't know they're pregnant, and they're fasting.

Okay? If they knew they were pregnant a little earlier then, you know, maybe they wouldn't fast, or maybe they wouldn't do other things. But if you look at, you know, the host of, sort of, lifestyle things. Dieting you know, drinking, smoking. These things you know, change when people recognize they're pregnant.

But, that first little window before people learn they're pregnant can still be really important. So I think that's, like, that's something that, like, we could agree with with the Chicago people that fostering earlier pregnancy recognition would cause individual behavioral change, but you know, might also be something we can target with policy as well.

Okay. So, yes, we find long run effects of that.
>> How is that evident in those results? You get the same
>> Yes, yeah. So, so that's for birth weight. Where, where you really see the, the, the-
>> Is in the long run effects. They, those totally heap up

>> Okay, so here's the case where there's not a bunch of mapping.
>> Yeah. Here's- Exactly. So this-
>> Birth weight.
>> I see this as showing that there was a first stage, but again, like, I never, I never see a very-
>> That's not the channel.
>> Yeah, that's not the channel.

>> But that's not the only one.
>> Doesn't and not the only one, exactly, exactly.
>> Do you update on gestational age? Cuz I wonder how you know whether Ramadan occurs
>> Yeah, right. So we do know gestational age not in Uganda or Iraq, but we do know it in Michigan.

And so we can assign Ramadan one of two ways; the dumb way which is just to sort of count back and assume a, you know, normal pregnancy, or we can use that exact, well, depending on who you ask, but date of last menses is the usual thing that people use to, to do it.

Or they can do a clinical estimate of gestation. It turns out whether we do that, use that information or forget about that information, it doesn't really matter. We, we throw out about, I think, like 15% of our sample when we do that. It does look like there is a small effect on the duration of gestation.

But most of this effect is conditional on gestation length so, intrauterine and growth retardation. The the point I want to make with this next slide here, is, essentially this is this is a study from Chicago that has nothing to do with Ramadan, per se. It's just about breakfast skipping.

Okay? So what they did is, they they had pregnant moms and they they they let them have dinner the night before. And then they brought 'em in, you know, like, at 6 a.m the following morning, 7 a.m. Okay? And then they monitored their blood biochemistry over time. Okay?

And what, what was the really starting, startling finding if you look at glucose or free fatty acids, all this stuff, you see that as the fast, you know, extends from 12 hours, 14 hours, 16 hours, 18 hours, the, the blood biochemistry stays pretty stable in the non-pregnant population.

But it goes pretty haywire in the non-pregnant population. And this, this sort of haywire of the blood biochemistry has, has a name from the diabetes literature which is called accelerated starvation. Cuz this, this behavior.
>> I'm sorry?
>> We're just remembering.
>> We're just saying it wasn't my imagination.

>> Okay. Yeah. Yeah. All hell is breaking loose. Yeah. yeah. This has been called accelerated starvation and there have been a number of recent studies on Ramadan that show basically exactly this for Ramadan, that when you extend the you know when the Mom is fasting for Ramadan then the blood biochemistry goes crazy.

I think actually Bosch working on this, but I think, you know, the, the leap from Ramadan to thinking about other, sort of more commonly experienced, I mean, Ramadan is very commonly experienced, so I think the internal validity question's still really interesting. But I think it's less of a leap to go from Ramadan to things like, breakfast skipping, than it is to go from, you know, famine to, to, to other things.

So I think we're sort of, we're, I'm not saying it's complete, or it's, or it's, it's a perfect thing. But I think we're sort of, we're, we're sort of filling in that, a little bit. And so if you can think of a good design in breakfast skipping or this sort of thing, dieting, whatever, like, that, that's gonna be, that's gonna be great to look at.

okay. So now I wanna sort of segue a little bit to poverty. Okay? So, and, and I had trouble making a slide just because it's, it, I think it's something that is, like, so obvious to everybody. So and I, so I, apologize for this. It's, it's probably super, super obvious.

Poor people experience worse prenatal environments on average. Okay? So if you look at something like neonatal, neonatal mortality, which is mortality within the first 30 or 28 days of life that is thought to register the, pretty well the prenatal environment. As opposed to post neonatal mortality, which is more like infection and accidents, and stuff like that.

And so if you, if you stratify I'm very proud of using the word stratify,
>> The high SES from the bottom SES, well, indeed you find that there's a, there's a, there's a substantial gap in the neonatal mortality rates, which would suggest that there's differences in the prenatal environment.

Similarly, Anne Eisner has some nice work with the National Collaborative Perinatal Project. Data from the 1960s where they collected really detailed data over a sample of low SES households and measured things like cortisol. And cortisol is sort of a frequently invoked stress hormone that in laboratory studies where they can randomly do nasty things to pregnant mice or whatever, that you find effects on the next generation when you manipulate cortisol.

And so she, she has a table that shows that you know, those with normal cortisol levels during pregnancy have, on average, $42,000 in income. And those with high cortisol levels have lower income. So this is just to say so if we think the fetal environment is really important, and exerts these long term effects on both health and SES, well it quickly becomes very relevant the fact that you know, that, that poor people experience worse environments.

So that's one of, one of the drags on, on subsequent outcomes. So in terms of actual studies on fetal origins and policy there are relatively few existing studies that look at this. And one of the reasons, I think, is that, you know, you have the usual policy evaluation challenges, which everybody is really familiar with, plus this sort of, this hungry longitudinal data requirement to be able to follow these cohorts forward.

Okay? So that just makes it a tougher sort of thing, and again, it's, it's if we had that metric of early life that captured this, then we, then we could sort of- We could look at the birth rate as I was there and say, okay I think I have a good sense of what this policy is gonna do in 30 years.

But I don't think we have that metric. So, so there is this disconnect. I think that's a reason why we haven't has as many of these policy studies. That said, I think they're absolutely critical and useful. So I'm gonna highlight a few of

them and I, I probably omitted some and then, then I'll turn to to, to some other issues.

So food stamps roll out in the US. That's a paper that's been presented in here. I'm not gonna talk about that in much detail, just summarize the results. There's a fairly well know paper by Peter Nielsen that looks in Sweden at a couple of counties where in I think end of 1967 they liberalized strong beer sales.

And if you've been to Sweden, like, alcohol is really expensive. That's why they all go, like, out on boats and get drunk, because then they don't have to pay the taxes there. But in 67 they liberalized the sales outside of the, of the state liquor stores to sell strong beer in grocery stores.

And basically it was just like, you know, debauchery and drunkenness and such that they had to, had to stop the experiment eight months later. Okay? What he does is he looks at that, that cohort that happened to be in utero when suddenly, you know, a lot of people were drinking like crazy and again this exercise of looking at the, the ones that were conceived prior.

Ward, so the same Ward that I mentioned before, she has a paper which I'm gonna, I haven't read but looks really intriguing, on school immunization policy. So basically, when different states adopted different immunization requirements. She looks at the cohort that was in utero then. So they would have experienced sort of like a, a herd benefit of having the older kids immunized, and find long run effects on outcome of that.

I mentioned this land reform in China for two reasons. One is, I'm gonna talk about land reform in China tomorrow. So if you can stand another talk that'll be me. The other reason is land reform has been called the largest anti-poverty program in the history of the world, because it lifted hundreds of millions of people out of poverty.

So basically, they decollectivized agriculture, and people gained a lot of agricultural income by being able to farm for themselves. And they're, and, so, there are long term effects of that. Okay? I'm actually not talking about the long term effects tomorrow. I'm talking about short term effects. But that's another instance where we can map the policy to later life outcomes and I, I am guessing there is a WIC project in the works of long run effects of WIC.

Is that true?
>> It might or might not be.
>> Okay. Okay, okay. All right. but, but maybe this is to, to, to highlight the need for additional studies. And I've probably omitted some. I was thinking that the Nick Sanders study was a policy one. But then that's the recession one, not, not the long run effects of the Clean Air Act Amendment.

So I was sort of scratching my head for other stories, studies. But I'm, I'm, I'm, I'm hopeful that there will be more soon.
>> I'm Ann Stevens, the director of the Center for Poverty Research at UC Davis, and I want to thank you for listening. The Center is one of three federally designated poverty research centers in the United States.

Our mission is to facilitate non-partisan, academic research on domestic poverty, to disseminate this research, and to train the next generation of poverty scholars. Core funding comes from the U.S. Department of Health and Human Services. For more information about the center, visit us online at poverty.ucdavis.edu