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Comment

James P. Smith

In the last decade, economists have rediscovered health as a fundamental subject of research (Smith 1999). The subsequent research has been instructive and insightful. Not the least among these important contributions has involved documenting the importance of childhood health, not only on subsequent health outcomes during the adult years, but also, and in large part due to this life-course health linkage, on a series of economic outcomes as an adult. These outcomes have included final years of schooling, labor supply, income, and occupational status (Smith 2006). Anne Case with her colleagues has been among the most important contributors in this fast expanding literature with a series of papers documenting these linkages into the early and middle part of the adult years (see (Case, Lubotsky, and Paxson 2002) and (Case, Fertig, and Paxson 2005) for two examples). This chapter extends Anne Case's excellent recent work with Chris Paxson on height and childhood health into much older ages than she has done before by using the Health and Retirement Survey (HRS), which samples a population of Americans who are at least fifty years old (Case and Paxson 2006).

The central thesis of Case and Paxson is that very early childhood health is extremely important for subsequent adult health and SES outcomes. Measuring childhood health is difficult in itself, but combining it with data that measure these outcomes during the adult years is extremely challenging. The key insight of Case and Paxson is that adult height is a particularly

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good marker and summary statistic for these early childhood health and environment factors.

In this chapter, Anne Case extends this insight into an analysis of adults at much older ages—age fifty and beyond. Using the Health and Retirement Survey (HRS), Case relates adult height to several health outcomes, including prevalence of hypertension, general health status, measures of functional ability, and measures of muscular and motor skills. She mostly finds that additional height promotes better health in all these dimensions. In particular, and the one I will focus on in my comments, she finds that one is less likely to be hypertensive if you are tall. Generally speaking, Case also reports that these beneficial effects of height on adult health increase with age—that is, there is more of a health benefit to those extra vertical inches the older one is.

While these are important findings indeed, Anne Case is more scientifically ambitious than to leave it just as that. In the second part of her chapter, she takes it all back a step by asking what influences adult height in the first place (and by backward induction, what influences childhood health). She relates crop production in the state of birth around the time the mother was pregnant to these late life health outcomes to see if this helps explain the height to later life health pathway. She reports quite positive results—good crops in year before birth (that is, the time when the baby was in uterus and most sensitive to the nutritional environment of the mother) predicts additional height and also lowers the subsequent likelihood of hypertension.

This is a fine chapter with important new findings that represent a significant contribution to the literature. In my comments, I will raise three questions: (a) do these results generalize beyond hypertension and beyond the HRS; (b) why does the effect of height increase with age; and (c) how robust are the results on crop failures?

Do These Results Generalize Beyond Hypertension and the HRS?

In the original literature linking childhood and inter-uterine environments to subsequent childhood health, two types of diseases were prominent—heart disease (of which hypertension is an important precursor) and diabetes (see Barker 1997). While the HRS is an important data set and is in some ways ideal for what Anne is trying to test, it does have its limitations. In particular, HRS only measures diagnosed disease. A nontrivial segment of the population has a disease, but is not aware of it (Smith 2007). A data set that does measure both diagnosed as well as undiagnosed disease is the National Health and Nutrition Examination Survey (NHANES), where blood pressure is taken on respondents as well as blood samples so that undiagnosed diabetes can be detected. Heights are also measured more accurately than in HRS as respondents' heights (and weights) are actually measured directly in NHANES.

To answer these questions in this subsection of my comments, I apply the

Table 8C.1 **Estimated effect of height in a probit model of diagnosed hypertension: NHANES 4, 1999–2002**

	df/dx	*z*	df/dx	*z*
Overweight	.132	6.15	—	—
Obese	.296	13.9	—	—
Height	.003	0.88	.002	0.81

Note: Also controls for race, ethnicity, gender, age, education, smoking, and exercise.

type of analysis conducted by Anne Case to diabetes. Table 8C.1 contains estimates (partial derivates from a probit model) of the impact of height measured in centimeters on the prevalence of hypertension for a population aged twenty-five to seventy years old. These models were estimated using the combined NHANES for the years 1999 to 2002. The models also include the standard set of other covariates typically included in prevalence models (race, ethnicity, gender, age, smoking, exercise, being overweight, or being obese). But contrary to the thesis in Anne Case's chapter, the estimated effect of adult height is statistically insignificant. Height appears not to matter a whit.

My initial thought was that the contradiction between the results present in Anne Case's chapter and these results summarized in table 8C.1 was a consequence of the other controls included in the model. In particular, the diagnosed hypertensive prevalence model in table 8C.1 includes controls for excessive body mass index (BMI)—either being overweight (BMI between 25 and less than 30) or being obese (30 or more). The construction of BMI (weight in kilograms/[height in meters^2]) has height in the denominator so that the direct effects of height might be suppressed by these BMI controls. Moreover, there is a quite reasonable argument based on the perspective taken by Anne Case that controls for BMI are not appropriate. The reasoning might be that one of the extra benefits of additional height is that it makes one less likely to be obese as an adult.

The results summarized in the rightmost columns of table 8C.1, however, reject this conjecture. In those columns, I list the estimated impacts of height obtained when the overweight and obesity variables were removed from the model. The effect of height remains small and insignificantly different from zero.

But not all is lost. The models in table 8C.1 are prevalence models for diagnosed hypertension (as are the models in Anne Case's chapter). But what we really want to know are the predictors of total hypertension prevalence, including those with undiagnosed diabetes. Fortunately this is possible since the NHANES includes blood pressure tests on their respondents, allowing us to identify those with undiagnosed hypertension and thus total prevalence of hypertension. The models summarized in table 8C.2 are identical to those

in table 8C.1 except that the outcome variable is now total hypertension prevalence instead of just diagnosed hypertension as in table 8C.1. Once again the estimated effects of height do not depend on whether or not the overweight and obesity models are included in the models. But height now has, as Anne's chapter argues that it should have, a statistically significant negative impact on the probability of being hypertensive.

The reason is more readily apparent from the probit model listed in table 8C.3. This is a model of the probability that one is an undiagnosed hypertensive given that one is hypertensive (either diagnosed or undiagnosed). Additional height significantly reduces the probability of being undiagnosed so that the diagnosed hypertensive model alone gives a biased perspective on the true impact of height on the probability of being a hypertensive. One of the benefits of additional height, apparently, is that you do the right thing and make certain that any disease you might have is diagnosed and presumably treated.

But not all is regained either. Tables 8C.4, 8C.5, and 8C.6 perform an identical analysis but this time examining diagnosed diabetes, total diabetes, and the probability of undiagnosed diabetes, respectively. In addition to hypertension, diabetes represents an excellent additional test of the early health impacts during childhood (using height as a marker) as it was one of the diseases mentioned by Barker (1997) as a prime candidate for early childhood illness being a marker for later life onset of disease. Just as was the case for hypertension, the effect of height is statistically insignificant for diagnosed diabetes (whether or not the controls for excess weight are included in the model (see table 8C.4). But this time we are not rescued by examining total diabetes prevalence since height remains statistically insig-

Table 8C.2 **Estimated effect of height in a probit model of total hypertension prevalence: NHANES 4, 1999–2002**

	df/dx	*z*	df/dx	*z*
Overweight	.135	6.35	—	—
Obese	.273	13.0	—	—
Height	–.006	1.92	–.006	1.91

Note: Also controls for race, ethnicity, gender, age, education, smoking, and exercise.

Table 8C.3 **Estimated effect of height in a probit model of the probability of undiagnosed hypertension: NHANES 4, 1999–2002**

	df/dx	*z*
Height	–.008	4.28

Note: Also controls for race, ethnicity, gender, age, education, smoking, and exercise.

Table 8C.4 **Estimated effect of height in a probit model of diagnosed diabetes: NHANES 4, 1999–2002**

	df/dx	*z*	df/dx	*z*
Overweight	.025	1.90	—	—
Obese	.084	6.22	—	—
Height	–.001	0.86	–.001	0.82

Note: Also controls for race, ethnicity, gender, age, education, smoking, and exercise.

Table 8C.5 **Estimated effect of height in a probit model of total diabetes prevalence: NHANES 4, 1999–2002**

	df/dx	*z*	df/dx	*z*
Overweight	.047	3.06	—	—
Obese	.138	8.68	—	—
Height	–.002	1.02	–.002	0.93

Note: Also controls for race, ethnicity, gender, age, education, smoking, and exercise.

Table 8C.6 **Estimated effect of height in a probit model of probability of undiagnosed diabetes: NHANES 4, 1999–2002**

	df/dx	*z*
Height	.0006	0.69

Note: Also controls for race, ethnicity, gender, age, education, smoking, and exercise.

nificant in that model as well (see table 8C.5). This is, of course, no surprise since height apparently has no effect of the probability of being an undiagnosed diabetic (table 8C.6).

Thus in the end this supplementary evidence from the NHANES is mixed. On the one hand, the data on hypertension, the same disease used by Anne Case, is quite supportive of her hypothesis. On the other hand, the data on diabetes, a disease that in at least my view should yield similar results, does not support the notion that height (a marker for early childhood health) is related to diabetes prevalence. More data and more diseases should be brought to bear on this problem since (as I said earlier) my intellectual sympathies are with the view that Anne Case puts forth so well in her chapter.

Why Does the Effect of Adult Height Grow with Age?

One of the more intriguing findings in this chapter is that the positive association of height with better adult health at older ages appears to become stronger with age. At first reading this result surprised me and my puzzlement remains unresolved. I can think of no really good reason why the path-

ways that Anne Case is talking about with the delayed effects of better child nutrition on hypertension should show up with greater force at age eighty when the prevalence rates for hypertension are quite high, for example, than at age fifty, when the incidence curve of hypertension is quite steep. In fact, I would have thought that the opposite result was more likely.

Let me offer a potential explanation. Adult heights are not completely fixed after the teenage years, as is often assumed. This assumption is fine for most of the adult life span, but becomes less tenable at really old ages. People actually do "shrink" at older ages and the centimeters begin slowly to disappear. Moreover, it is reasonable to presume that those in poorer health and the frail shrink the most. If shrinkage is sufficiently important, then adult height becomes endogenous, and at least to some degree the causation now runs from poor later life adult health to adult height. In a panel context, we could use, say, height in your fifties or early sixties to control for this problem, but that is not yet an option open to Anne using the HRS. Many of her observations entered this sample when they were quite old and to some extent their adult height may have already reacted to their increasing poor health and frailty.

How Robust Are the Results on Crop Failures?

The second part of the chapter tries to identify the sources of the adult height to adult health relationship. In this part of the chapter, Anne Case relates corn production in the division of birth of respondent while mother was pregnant to these late life health outcomes to see if this helps explain the height to later life health pathway. The basic idea is that good crops in year before birth through better nutrition predicts additional height and thereby lowers hypertension as an older adult.

The results summarized well in Anne Case's chapter, generally speaking, lend good support to her hypothesis. For example, good crops in year before birth predict additional height and lower the prevalence of hypertension as an adult.

This is a very ambitious part of the research agenda and I want first before I raise any questions or doubts to congratulate Anne on her boldness and urge her to continue on this line. One concern that I do have is that this seems to be asking a lot of the data given the real possibility of age reporting problems, especially at very old ages. Isolating precisely the year before pregnancy is difficult given normal age reporting problems, which may be compounded by the fact that bias may also be operating in that older people may say that they are younger than they really are. One possibility does exist to help mitigate this problem. The HRS has matched to social security records where ages are far more precise. These Social Security (SS) match records can be used to get the "true" ages of these respondents. Finding the same results with these ages would say a ton about the robustness of the results.

The second concern I have is the regional basis of the analysis. Even dur-

ing the period when these HRS respondents were born, I would think that the food market was pretty national, produced in some places but consumed in all. Bad corn crops would show up as more scarce corn and higher prices of corn and their related products everywhere. Corn is also an important feeder crop for animals (like pigs) and these lags would seem longer than just during the term of pregnancy.

Conclusions

It should come as no surprise to those who have read Anne Case's prior work on this topic that she has written yet another fine chapter. I think that the evidence is growing and is quite persuasive about the fundamental importance of very early childhood health on our economic and health lives during the adult years. Anne Case provides some additional evidence in this chapter that these linkages are not just for the middle-aged but also follow us into our post-retirement years. The fact that I raise some questions about the details should not camouflage the fact that at its core I believe the perspective she advances in this chapter and in her prior and (I hope) subsequent work is dead-on right.

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