

Response to “Growth in Earnings and Health: Nothing is as Practical  
as a Good Theory”

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Avdic and Karlsson (2016) (AK hereafter) provide a thoughtful commentary on my recent work published in this issue of the *Review of Income and Wealth*. While it is true that Table 2 of AK does replicate my core findings (albeit with lower magnitudes), they do argue that these results come from a misspecified model; and that a correctly specified model that is informed by theory fails to replicate my findings and, therefore, provides no evidence of income-to-health causality.

Before I proceed, one issue that merits attention is that AK do not follow the sample selection criteria that I outlined in the appendix of my paper, which closely mimics the sample selection from Meghir and Pistaferri (2004). In addition, in a previous version that was published as an IZA working paper (see Halliday, 2007), I employed a less carefully selected sample from the PSID that is more in line with AK (and less in line with Meghir and Pistaferri, 2004), as it does not drop individuals who have implausible income movements while in the panel. With this sample, I obtain an estimate of the effect of income on self-rated health of 0.005 (in absolute value) for working-aged men in column 1 of Table 3 of that paper. This is consistent with the magnitudes in AK. I presume that failure to drop these individuals with highly erratic income dynamics increases the amount of measurement error in income, which increases the attenuation bias in the estimates of the parameter  $\beta$ .

I now respond to several of the critiques from AK. First, I will argue that dynamic panel methods, while not perfect, can and do provide important insights into causal relationships in panel settings when best practices are followed. Second, I will argue that the theoretical framework outlined in AK, contrary to what is asserted, does not inform researchers about what the true moment conditions are. The truth is that theoretical and statistical models both allow the researcher discretion over assumptions. Finally, a potential misspecification in their model may be resulting in their negative estimates of the effects of income on health due to the presence of negative serial correlations in earnings growth.

## 1 DYNAMIC PANEL METHODS

Recent work by Roodman (2009) provides excellent insights for practitioners who employ the dynamic panel methods discussed in Arellano and Bond (1991)

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and Blundell and Bond (1998). As pointed out in AK, such methods leave a lot of room for practitioners to make choices about appropriate moment conditions, lag structure, and the number of instruments employed. Because of this, many researchers think that these methods may be abused for the purposes of data mining. I would take the opposite position, namely, that *when used appropriately* they make data mining less likely.

In regard to choices about the lag structure of the model and which moments to use, researchers have a battery of specification tests at their disposal to inform them of the validity of these assumptions. While it is true that one can never test if any one instrument is valid for a given model, it is true to say that if all of the instruments are valid, then an over-identification test will reject the null with a small probability that is set by the researcher *a priori*. So, in this sense, these specifications tests can be viewed as providing necessary conditions for the model to be properly specified. While such a statement may not be surprising to many readers, it is the case that much of the literature on the SES/health nexus has ignored this, as I discuss in my paper.

Another important feature of best practices is to provide tests for whether or not the excluded instrumental variables are weak. There is an extensive literature on this topic that has shown, under conditions, distribution theory for test statistics that are used to test for weak instruments. While these conditions can be seen as restrictive, one can view these papers as providing rules of thumb governing the decision rules for these tests. Moreover, and in contrast to the tests of the exclusion restrictions discussed above, one can always test whether or not an excluded instrument is weak, whereas one can never test whether or not any *given* instrument is uncorrelated with the residual.

Finally, Roodman (2009) discusses the pernicious effects that having too many instruments can have in these models. As far as I know, there is no formal test for the “correct” number of instruments. However, given the potential negative effects of instrument proliferation on estimation and testing, it is critical for researchers to be mindful of this and to follow a few simple rules of thumb. The first is to cap the number of instrument lags at some (admittedly *ad hoc*) number. The second is to run robustness checks while varying this cap.<sup>1</sup>

Thus far, much of the focus in this literature has been on linear dynamic models, as in my paper and others such as Michaud and van Soest (2008). However, relatively recent work by Arellano and Carrasco (2003) has shown how to incorporate predetermined variables into non-linear dynamic models. Future work should employ these methods for unraveling causality between SES and health.

## 2 THEORY AND EMPIRICS

Researchers can either make assumptions in a theoretical model or in a statistical model. At least in this case, the former is no better nor no worse than the latter. Essentially, what is done in AK is to make assumptions about a particular specification of the Grossman model and then it is claimed that theory implies

<sup>1</sup>In unreported results, I did this and found that my results were robust.

that particular moment conditions are appropriate. In particular, AK write down the following specification for health investment:

$$h_{t+1} = h_t + [I(y_t) - \delta_t],$$

where  $h_t$  is the health stock at time  $t$ ,  $y_t$  is income at time  $t$ ,  $I(\cdot)$  is the health production function, and  $\delta_t$  is the depreciation rate.<sup>2</sup> In the model, it is *assumed* that income impacts health with a lag and that the only channel through which it can do so is investment, which we presume is medical consumption. This is not an implication of theory.

The truth is that, *a priori*, one can conceive of scenarios in which either contemporaneous or lagged income affect health. Making an *ad hoc* assumption in a theoretical model does not answer this question. Moreover, understanding the appropriate lag structure of income is largely an empirical question. The results in AK largely indicate that it is contemporaneous income and not lagged income that matters, since the results that use contemporaneous income are consistent and robust, whereas the other results are not. Notably, I found the same result while performing numerous analyses when I was responding to referee reports for this journal.

### 3 THE COVARIANCE STRUCTURE OF EARNINGS

Finally, a large literature has documented that income growth exhibits negative serial correlation (e.g. Abowd and Card, 1989; Meghir and Pistaferri, 2004). This empirical fact can result in a negative bias in a misspecified model. To fix ideas, let us consider the simple empirical model

$$h_{it} = \alpha_i + \beta_1 y_{it} + \beta_2 y_{i(t-1)} + u_{it},$$

where  $h_{it}$  is the health status and  $y_{it}$  is the income of individual  $i$  at time  $t$ . If we take first differences, then we obtain

$$\Delta h_{it} = \beta_1 \Delta y_{it} + \beta_2 \Delta y_{i(t-1)} + \Delta u_{it}.$$

For the sake of simplicity, suppose that we estimate this model via ordinary least squares while omitting either  $\Delta y_{it}$  or  $\Delta y_{i(t-1)}$ . Then the asymptotic biases of the estimates of  $\beta_1$  and  $\beta_2$  will be given by

$$p\lim \hat{\beta}_1 = \beta_1 + \beta_2 \frac{\gamma_1}{\sigma_{\Delta y_{it}}^2},$$

$$p\lim \hat{\beta}_2 = \beta_2 + \beta_1 \frac{\gamma_1}{\sigma_{\Delta y_{it}}^2},$$

<sup>2</sup>Perhaps, a better model would have been  $h_{t+1} = (1 - \delta_t)h_t + I(y_t)$ .

where  $\gamma_1$  is the first-order autocorrelation in  $\Delta y_{it}$  and  $\sigma_{\Delta y_{it}}^2$  is its variance.<sup>3</sup> As already discussed, a preponderance of evidence has shown that  $\gamma_1 < 0$ . Therefore, if  $\beta_1 > \beta_2 \geq 0$ , then it is possible to obtain that

$$plim \hat{\beta}_1 > 0 > plim \hat{\beta}_2.$$

Of course, one can also obtain these inequalities if  $\beta_2 < \beta_1 \leq 0$ . However, this indicates that positive income shocks lead to *worse* health outcomes and that they mostly affect health with a lag, but I do not find this story particularly compelling.

In summary, the results in Table 3 of AK are not necessarily at odds with my results when seen in this light. Given the fact that the estimates vacillate from positive and significant to insignificant to negative and significant, they do not yield a coherent picture and so indicate that there is no clear relationship between lagged income and health. In contrast, my results as well as those in Table 2 of AK indicate a stable pattern in which contemporaneous income has a positive effect on health for working-aged men. That should be the take-away point of this work.

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<sup>3</sup>We are assuming stationarity.