The Effect of Disability Insurance on Beneficiaries' Mortality

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1. Introduction

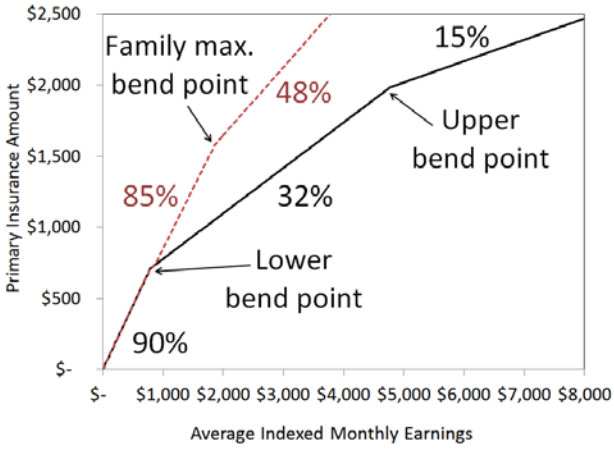
A sizeable body of research has established that Disability Insurance (DI) has substantial work disincentive effects (recent studies include Borghans, Gielen and Luttmer 2014, Gubits et al. 2014, Kostøl and Mogstad 2014, Autor, Maestas, Mullen and Strand 2015, Coile 2015, Moore 2015, and Gelber, Moore, and Strand 2015). This raises the possibility that DI has not only direct costs through transfer payments but also indirect costs via these disincentive effects.

Much less work has documented the potential benefits of DI, especially in terms of whether DI improves health outcomes. Weathers and Stegman (2012) use the Accelerated Benefits demonstration project to examine the effects of expanding the health insurance coverage of newly entitled DI beneficiaries, finding positive impacts on self-reported health and no impact on mortality. Garcia-Gomez and Gielen (2014) find that stricter eligibility criteria for DI in the Netherlands led to more frequent hospitalizations and higher mortality among women, but lower mortality among men. However, none of this work has examined the effects of the size of DI payments on health outcomes. One reason is the difficulty in identifying causal effects on health for a program that specifically targets people whose health is poor.

We estimate the effect of DI payments on mortality using the details of the formula that determines benefit amounts. Figure 1 summarizes the relationship between Average Indexed Monthly Earnings (AIME) and the Primary Insurance Amount (PIA). The rate at which monthly DI benefits (PIA) replace past earnings (AIME) changes around several "bend points." Below the first bend point, the marginal replacement rate is 90 percent; between the two bend points; the rate is 32 percent; and above the second bend point, the rate is 15 percent.

In addition, the rules for the maximum benefits a family can receive imply that the marginal replacement rate for a family's combined worker and dependent benefits changes from 85 percent to 48 percent at an AIME value that lies between the two bend points discussed above. We refer to this point as the "family maximum bend point" and show it using the dotted line in Figure 1. This bend point is different from the bend points in the family maximum formula for retirement benefits.

Figure 1 Primary insurance amount and average indexed monthly earnings



Using these bend points, we implement a "Regression Kink Design (RKD)" (Nielsen, Sorensen and Taber 2010, Card, Lee, Pei and Weber 2012). Intuitively, the technique is based on observing changes in the slope of the relationship between mortality and the AIME values that determine the size of DI payments around the bend points. Any abrupt change in the relationship between mortality and AIME that occurs at a bend point suggests that DI payments affect the health of beneficiaries. We focus on claimants that are close to the bend points, as they should be similar to each other. The RKD method requires that claimants are not easily able to control on which side of a bend point they fall. In this sense, each bend point creates an experiment that can be used to estimate a causal effect relevant to that group of beneficiaries (i.e., a Local Average Treatment Effect (LATE)).

We investigate whether the data support this quasi-experimental interpretation. First, we show that the population that is not affected by the bend points – that is, non-beneficiaries – does not experience a shift in mortality around the bend points (placebo tests). This suggests that there is not some pre-existing relationship between average earnings and mortality that changes at the bend points. Second, we show that the population characteristics and population counts do not shift around the bend points (covariate balance tests). This gives indirect support to the idea that claimants do not manipulate their position relative to the bend points. Third, we show that shifts in mortality of similar magnitude do not occur at other points in the distribution of AIME away from the bend points (placebo kink tests).

2. Data

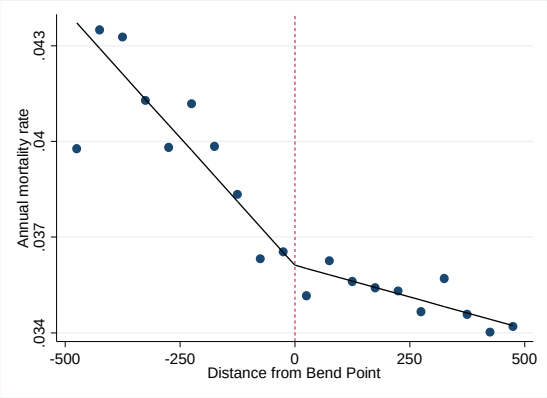
We use administrative data from the 2010 version of the Disability Analysis File (DAF) (previously called the Ticket Research File). The DAF is a compilation of multiple administrative data sources from the Social Security Administration, including the Master Beneficiary Record, Supplemental Security Record, 831 File, Numident File, and Disability Control File. We updated the mortality data up to the end of 2013. We choose a sample of individuals who entered DI between 1997 and 2009 and who were aged 21 to 61 years at the time of filing. The program rules were largely consistent throughout this time period, and we are able to observe whether these individuals died within four years of beginning to receive DI payments. The upper age restriction to those under 61 avoids interactions with rules associated with the Social Security Old Age and Survivors Insurance program. We also limit the sample to DI claimants who did not receive Supplemental Security Income at any point in the sample period. For comparisons to non-beneficiaries, we use the Continuous Work History Sample.

3. Results

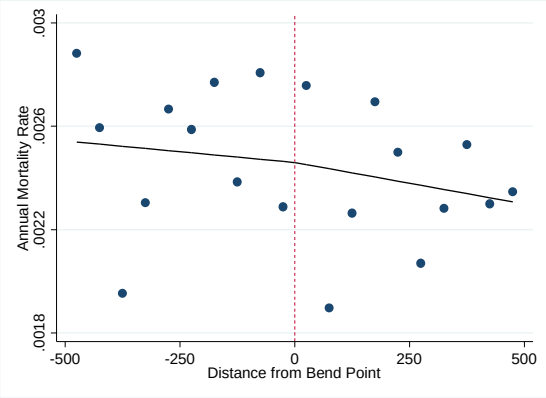
We begin by plotting the mean yearly mortality rate in the four years after DI allowance against AIME with claimants grouped in $50 bins. If higher income is protective against mortality, we would expect a positive change in slope in the region above a bend point relative to the region below it. We observe this for the lower bend point in Figure 2A, where the negative slope becomes flatter. Intuitively, as the marginal replacement rate decreases, additional income becomes less protective. Equivalently, a higher replacement rate is associated with lower mortality.

Figure 2 Mortality rates around the lower bend point

A: Analysis sample



B: Non-beneficiaries (placebo)



We estimate the magnitude of the effect using RKD methods. Because the functional form of the relationship of mortality and AIME is unknown, we estimate linear, quadratic and cubic forms. Also, we test specifications with and without discontinuities at the bend points, and with and without controlling for covariates.

At the lower bend point, we find consistent evidence across all these specifications that increased DI benefits leads to a substantial reduction in mortality. We scale the results to represent a $1,000 increase in annual DI benefits, compared to average annual DI benefits of $8,268 near the lower bend point. For our preferred specification, the corresponding decrease in annual mortality is 0.47 percentage points on a basis of a mortality rate of 3.61 percentage point. The resulting elasticity is -1.11.

At the upper and family maximum bend points, the results are not robust to different specifications. In our preferred specification at the family maximum bend point, the elasticity is -0.60. At the upper bend point, the estimate is not statistically or economically significant.

We test whether the data support interpreting the magnitudes as the effect of DI benefit amounts on mortality. First, we test whether the effect appears among the population that is not subject to bend points in the PIA formulas. In Figure 2B, we show the relationship between mortality and AIME for the population that is DI-insured but has not applied for benefits. There is no increase in slope at the lower bend point—in fact, if anything, the slope modestly decreases at the bend points, though not sharply. This suggests that no population-level phenomena cause the kink in mortality. Second, we show that a number of claimant characteristics that can be observed in the administrative data do not show kinks or discontinuities at the bend points. This suggests that individuals are not able to locate their AIME in relation to the bend points strategically. Third, we show that—at least for the lower bend point—the magnitude of the kink is statistically significant at the bend point and is not significant away from there.

These tests suggest that RKD methods are appropriate for estimating causal local treatment effects. We present strong evidence of an effect at the lower bend point and mixed evidence of an effect at both the family maximum and upper bend points.

4. References

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