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IS RECIPIENCY OF DISABILITY PENSION HEREDITARY?

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Is Recipiency of Disability Pension Hereditary?*

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Abstract

This paper addresses whether children's exposure to parents receiving disability benefits induces a higher probability of receiving such benefits themselves. Most OECD countries experience an increasing proportion of the working-age population receiving permanent disability benefits. Using data from Norway, a country where around 10% of the working-age population rely on disability benefits, we find that the amount of time that children are exposed to their fathers receiving disability benefits affects their own likelihood of receiving benefits positively. This finding is robust to a range of different specifications, including family fixed effects.

Keywords: disability, intergenerational correlations, siblings fixed effects
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1. Introduction

In the US and most of Western Europe, the share of the working-age population living on a disability pension (DP) is increasing (OECD 2010a, Autor and Duggan 2003, 2006). Together with an aging population, this is an increasingly important policy issue. Several aspects make this a concern. The rising number of disability pensioners itself puts a strain on public finances. Moreover, reduced fertility rates and increasing longevity combined with low average retirement ages adds to a worsened dependency ratio. Put together, these are factors that question the sustainability of the existing welfare states. To the extent that some people on disability benefits could have been working, there may also be individual costs in terms of foregone earnings and social exclusion that result from being outside the labor force.

The increasing DP trend is not reflected in deterioration in general health. On the contrary, standard health indicators suggest *improved* public health.[1] The combination of improved public health and rising disability rolls is encountered in many Western countries; see Autor (2011) for a recent discussion of the US case. This observation has lead researchers to test other mechanisms and routes to DPs. One obvious candidate stems from the innate moral hazard problems enforced by the generosity of the disability insurance systems. In the US, there is a long series of contributions discussing the incentives produced by US disability programs, such as Parsons (1980), Leonard (1986), Bound (1989), Chen and van der Klaauw (2008) and Autor (2011). Börsch-Supan (2007) compares the EU15 countries plus the US, and finds, after controlling for demographic structure and health status, a substantial cross-national variation in DP enrolment rates. As much as three quarters of the variation, he

[1] See for instance OECD (2010b).

claims, is due to country-specific disability insurance rules. Furthermore, Bratsberg *et al.* (2010) show that a large percentage of disability insurance claims can be directly attributed to job displacement and other adverse shocks to employment opportunities and therefore conclude that unemployment and disability insurance are close substitutes, at least in Norway. Rege *et al.* (2007) report substantial social interaction effects in DP participation among older workers after plant downsizing. Hence, DP, in addition to being a social insurance against severe health losses, also appears to be influenced by the labor market and social norms. Norms are established at home and transferred from parents to children. Our intension in this paper is to disentangle the different ways through which parental disability is transferred to the next generation.

Parents' influence on children's outcome has been studied within many different fields and from many different angles. As one would expect, economists have been focused on the transmission of *economic* status from one generation to the next. In particular, there is a vast literature on income mobility across generations, expressed by the estimation of intergenerational earnings elasticities; see Solon (1999) and Black and Devereux (2011) for overviews and Bratberg *et al.* (2005), Bratsberg *et al.* (2007), and Nilsen *et al.* (2012) for Norwegian assessments. In later years, however, we have witnessed a growing interest in other aspects of intergenerational transmission. Examples, surveyed in Black and Devereux (2011), are education, health, welfare participation, jobs and occupation, consumption, attitudes, etc. Moreover, it has become increasingly common to address the causal mechanisms underlying the intergenerational correlations.

As for DP, Kristensen *et al.* (2004) report a relatively strong positive association across generations. Note however, this association is to be interpreted as a *causal* link only if the child becomes a DP receiver because of his/her parents

receiving the same pension. This is a point that is made in several earlier papers, most of them based on analysis of intergenerational transmission of welfare benefits and/or welfare participation; see Duncan *et al*. (1988), Gottschalk (1990), Levine and Zimmerman (1996), Pepper (2000), Page and Stevens (2002), and Mitnik (2010) for US and Canadian analysis.[2] Stenberg (2000), Edmark and Hanspers (2011), and Lorentzen (2010) are recent analysis from Sweden and Norway, respectively. The Scandinavian welfare model differs somewhat from the other Western economies, notably the Anglo–American, in its relatively extensive use of unitarian, health-related social insurance, e.g., DP, at the expense of means-tested welfare benefits. Contrary to welfare benefits, DP is an absorbing state, meaning that once an individual enters this form of benefit scheme, he/she rarely returns to self-support. In an ideal world with perfect information, DP simply serves as insurance against health-related loss of the ability to work. In reality, however, DP may be used as an early retirement route even if ability to work has not changed.

There is obvious scope for parents to affect children's propensity to become disability benefit receivers. Children inherit their parents' propensity to become welfare receivers (partly) as a result of negative impacts from the welfare system. Such adverse influences can be the result of values, attitudes, and behaviors of parents and neighbors and/or a decrease in the stigma associated with the welfare system. It may also be because of the development of self-defeating work attitudes and poor work ethics.

On the other hand, a positive association might also reflect (observed and unobserved) characteristics of the family that might have been present *before* the parent became a DP receiver. In that case, the observed relationship between the DP

[2] For an overview of the sociological literature, see for instance Corcoran (1995)

behavior of parents and children could turn out to be spurious. For example, individuals at the lower end of the earnings distribution are far more likely to end up as DP receivers than those at the upper end. The income of a pension receiver is further reduced compared with the earnings before receiving DP. As in Levine and Zimmerman (1996), the correlation between DP status and their status as low-income earners makes it difficult to separate the intergenerational transmission of DP from the intergenerational transmission of earnings.[3] If disabled parents provide limited educational opportunities, live in substandard neighborhoods, etc., it may affect the children's education, their probability of living in the same neighborhood, receiving social insurance and, ultimately, the probability of ending up on public benefits. In such cases, we will observe a strong correlation between parents' and children's DP, but it will be misleading to argue that parents' DP is *causing* the children's receipt of a pension. An even more obvious example is health. Poor health is a necessary qualification for the eligibility of DP. At the same time, it is often the case that children inherit poor health from their parents. Hence, health is clearly a potential confounding factor in our attempt to isolate the causal mechanisms behind receiving DP.

In this paper we investigate whether children's probability of becoming DP receivers increases the longer they are exposed to parents whom themselves receive DP. This has the potential of taking our understanding one step further regarding the way DP is transmitted between generations: genetic susceptibility is congenital and will not be altered, while children are more likely to pick up their parents' attitudes and behavior the longer the duration of exposure. In the analysis we consider the probability of DP for the offspring before the age of 40; hence, we do not restrict the

[3] The authors study mother–daughter correlation for the receipt of American Aid to Families with Dependent Children (AFDC). In terms of Levine and Zimmerman (1996), "the poverty trap will confound the estimation of the welfare trap" (p.3).

transmission to take place while the children are living with their parents.[4],[5] The analyses are performed on a sample of Norwegian siblings. The ability to identify siblings is an important characteristic of our data, allowing us to control for fixed, unobserved heterogeneity within families. To illustrate the advantage of a family fixed-effect, it may be informative to consider specific sources of parents' state of disability, e.g. addiction. According to medical research addiction is to some degree hereditary (Kendler *et al.*, 2007). Moreover, being exposed to parents being DP receivers because of, for example, alcohol addiction obviously has adverse effects on children in the family; increasing, in turn, their own likelihood of becoming DP receivers. The focus of this paper is the latter effect controlling for the former, which is exactly the virtue of the family fixed-effect model. Genetic susceptibility is common for the siblings in a given family and, hence, is integrated out in the fixed-effect model, while the exposure varies according to the ages during which the siblings were confronted with their parents' addiction and DP status.[6]

By focusing on Norway, we have access to data that enables us to calculate parent–child correlation but also, more importantly, to explore the mechanisms underlying the intergenerational correlations in DP receipts. Norway has also experienced rising rates of DP payments for several decades, and currently has one of the world's highest disability rates at 9.5% of the population aged 18–67, according to the National Insurance Administration (persons on temporary disability benefits not

[4] It is possible that the younger children move out relatively earlier as a response to the DP state of their parents (this information is not available in our data). In that case, it may be argued that our measure of exposure will be underestimated for the youngest siblings.

[5] In the remainder of this paper we will use the terms child and offspring interchangeably independent of the age of the child/offspring.

[6] To the degree that the parents' receipt of DP is an indicator of poor health, their health condition might influence their opportunities and abilities to take care of their children. If so, this is a case where parents' poor health affects the children adversely and ultimately might influence their probability of becoming DP receivers. Furthermore, one must assume that this effect increases with duration of exposure.

included). No corresponding deterioration in general health has been documented. On the contrary, standard health indicators, objective as well as subjective, point in the direction of *improved* public health (see for instance Norwegian Institute of Public Health). The Norwegian data have several advantages. First, they are full population registry data with information on receivers as well as nonreceivers of social insurance benefits, and the representativeness of the sample is not an issue. Second, we have longitudinal information on social insurance, making it possible to infer the age of the offspring when a parent started receiving DP. Thus, we are able to construct a variable that measures children's exposure time to parents' receipt of DP. Third, because our data include siblings, we can control for unobserved family effects. Finally, with data on both parents, we are able to analyze differences in the correlations between son–father, son–mother, daughter–father, and daughter–mother.

Our analysis shows that there is a positive correlation in the probability of receiving disability benefits between children and parents. We also find that the amount of time a child is exposed to parents' receipt of DP benefits affects children's likelihood of receiving disability benefits. Finally, when separating the intergenerational transmission of DP from the transmission of family fixed effects, the negative and statistically significant effects of exposure to parents' disability benefits is still present. Several robustness checks are performed. First, one-child families are added to the sibling sample. Second, different age group variables are employed to measure the duration of exposure. Third, "stable" families are estimated separately. None of the robustness checks reject our main finding; the longer children are exposed to parents' receipt of disability benefits the higher their own likelihood of receiving disability benefits.

The rest of the paper is organized as follows. In Section 2 we describe the institutional background. Our data are presented in Section 3, while the empirical model is discussed in Section 4. Our results are discussed in Section 5, while some concluding remarks are given in Section 6.

2. Background and Institutional Details

The Norwegian Act of Disability was passed by the Parliament in 1960. In 1967 it was integrated with a comprehensive social insurance scheme called The National Insurance Scheme (NIS). The NIS encompasses the old age retirement scheme, sickness benefits, disability benefits, unemployment insurance, and health insurance. In principle the NIS gives full population coverage, with defined benefits based on earnings histories.

All employees who have been with the same employer for at least four weeks are covered by the mandatory sickness insurance scheme, which stands out as very generous compared with other countries. Sickness benefits are paid by the employer for the first two weeks, and then by the NIS for a maximum of 50 weeks. Individuals with permanent impairments may apply for disability benefits, roughly corresponding to old-age pensions. The application must be certified by a physician. Furthermore, the NIS supplies benefits for participants in medical and vocational rehabilitation. Old-age pension is a mandatory defined-benefit system, which includes an earnings-based supplementary benefit in addition to a fixed minimum pension benefit. Disability benefits are, roughly speaking, calculated as the old-age benefits the beneficiary would have been entitled to had he/she continued working until the age of 67, the ordinary retirement age. The average DP compensation ratio is 50–60%. To be eligible for

disability benefits, relevant rehabilitation should have been attempted. Rehabilitation benefits are roughly the same as disability benefits.

The sickness and disability insurance schemes both place heavy burdens on the Norwegian welfare state. Direct expenditures associated with the sick leave scheme are in the order of 2.5% of GDP and workdays lost constitute 6.5% of total working hours. The disability benefit recipiency rate, i.e. the number of DP recipients as a share of population aged 18–67, is very high in Norway compared with other OECD countries. Numbers reported by OECD (see OECD 2010a, Figure 2.9), state that in 2008 the recipiency rate was a little higher than 10% in Norway, while the corresponding number for the OECD on average was just below 6%. The evolution over time is shown in Figure 1, covering the period 1980–2011. The stock of disability pensioners rose steadily through the 1970s and 1980s, stabilized in the early 1990s following a stricter admission policy, increased again from the mid-1990s, and has decreased somewhat from the turn of the century. As of late 2011, 9.5% of the population aged 18–67 are disability pensioners. In 2004 a reform introduced temporary disability benefits that could be granted for a maximum of four years. In 2010, the temporary disability benefits were abolished and replaced with a new temporary benefits program that also includes previous rehabilitation benefits.

[Figure 1: Evolvement of the disability benefit recipiency rate in Norway]

[Figure 2: The disability benefit recipiency rate by age and sex]

Figure 2 shows that the probability of ending up on disability benefits increases exponentially with age. As for age 60, we see that approx. 35% of the female population are receiving disability benefits, while the corresponding numbers for men

is 24%. At age 66, one year prior to the standard pension age in Norway, the corresponding numbers are 49% and 40% (women and men, respectively). This is shown in Figure 2.

Unlike many other European countries, Norway had no general early retirement scheme until 1989, when a program was introduced, which covered employees in the public sector and about half the private sector from the age of 62. The substitution from disability to this scheme was moderate (Bratberg *et al*. 2004). In 2011 a major reform of the pension system was introduced, where flexible retirement from the age of 62 is an integral part. This reform does not affect our analysis, which covers DPs granted up to the 2004 reform.

3. Data and Sample

We use data from Norwegian registers covering the entire population, provided by Statistics Norway. Importantly, the data include parent–child links via personal identifiers. The database includes earnings data starting in 1967 and other background information with yearly updates from 1986. We also have longitudinal data from the Norwegian Labour and Welfare Administration (NAV) including information on DP receipts. The NAV data include two variables that are relevant for determining when a person started receiving DP: the first date for actual receipt of DP, and the date when the sickness spell leading to DP started, typically several years before receipt (but the individual benefits from other social insurance benefits in the meantime). The DP-start variable is censored in 1991, but not the other variable, which covers spells back to 1966. In the analysis, we base the outcome of children on actual DP starts (the

censored variable) while we use the other variable to assess the DP status of parents.[7] The NAV data are limited to individuals below the retirement age (67) in 1991, thus we do not include parents born before 1925.

In the analysis we consider the probability of DP for the child before the age of 40, using the cohorts born 1951–1963, with DP status measured in 1991–2003. The 1963/2003 restriction is because of the reform in 2004 that introduced temporary disability benefits, see the previous section. This reform implied that some individuals who would have been granted permanent DP in the old regime were now granted temporary benefits, but also some who were accepted into the new program might have been rejected permanent benefits. As we want to focus on the time children are exposed to parents' disability, we exclude observations for children whose father received disability benefits starting before the child was born. This gives us a sample of 334,995 father–child and 374,307 mother–child pairs. As we shall discuss in the next section, the main analyses are performed on a sample of siblings, i.e., single children are excluded. The siblings sample includes 257,705 and 289,032 father–child and mother–child pairs, respectively. Results using the full sample are reported in the appendix. To define the outcome variable (DP before the age of 40) we chose the year of entrance into the absorbing state of permanent DP. This is also consistent with how disability rates are reported in public statistics. For parents, it is reasonable to assume that a potential effect on children begins before DP is actually granted, thus the available variable is well suited for the purpose. The main explanatory variables are indicators for parental DP receipts, and an interaction term between this indicator and dummies for offspring age intervals at the beginning of the parental sickness spell

[7] The time span between the initial sickness spell and actual DP start varies greatly, with an average of about 4.5 years and a standard deviation of the same magnitude for 35–39 year olds.

leading to DP. We control for child and parental cohorts, birth order, parental education, parents' age at childbirth, and family income (average for offspring age 16–19 in 10,000 1989 NOK).

[Table 1 about here]

Table 1 shows the descriptive statistics for the siblings sample that is used in the main analysis. We note that 4% of male offspring and 5% of female offspring are DP receivers at age 40, while 44% of fathers and 45% of mothers become DP receivers during the observation period, indicating that despite the necessity of a medical diagnosis, DP to some extent may work as an early retirement. We also note that on average fathers are born in 1930 and mothers in 1933, and were respectively 28 and 25 years old when the children in the sample were born. Mothers on average are significantly less educated than fathers, reflecting the fact that they grew up in the 1930s and 1940s before the educational explosion.

[Table 2 about here]

Table 2 shows family characteristics by fathers' disability status, where DP = 1 indicates that the father became a disability recipient before the son/daughter was 40. We note that the groups are quite similar with regard to cohort and age at childbirth, but parents in the DP group are less educated. This latter group also has lower incomes, but as incomes are not necessarily measured before DP was granted, the difference is not only due to lower labor market earnings. The DP families are slightly larger, indicated by three or more children.

4. Econometric Model

A simple linear probability model for intergenerational correlation in DP, *dp*, is

$$(1) \qquad P(dp_{ij}^{c} = 1) = \alpha + \beta 1(dp_{j}^{p} = 1) + \gamma X_{ij} + f_{j} + u_{ij},$$

where 1(.) is the indicator operator, subscripts *ij* denote individual *i* from family *j*, and the superscripts *c* and *p* denote child and parent, respectively. For sons and daughters, *dp* is measured at age 40, for parents, $dp_j^p = 1$ indicates disability before the offspring is 40. X_{ij} is a vector of family (parent and child) characteristics, f_j is family-specific unobservables that may affect the outcome, and u_{ij} is a random error term. If there is a correlation in the probability of DP between the generations, we expect $\beta > 0$.

If f_j is correlated to dp_j^p, β will be biased by unobserved characteristics present in the family before the father became a DP recipient. Typically, this would be hereditary factors that affect health, or differences in family preferences. If we have several observations on each family (i.e. a couple of siblings), the model could be purged of f_j by treating it as an unobserved family fixed effect.[8] Note however, the way equation (1) specifies no variation in dp_j^p in family *j*. On the other hand, if there is a learning effect in the intergenerational transfer of disability, we would expect the child's disability probability to be increasing in the time he/she is exposed to parental disability. Exposure time will vary between siblings, thus a siblings fixed-effect estimator becomes available by interacting dp_j^p with exposure. To implement this, we let a_{ij}^c denote the age of the child when the parent became disabled and augment (1) to

[8] Ekhaugen (2009) exploits sibling variation in a recent paper on intergenerational correlation in unemployment.

$$
\begin{aligned}
P(dp_{ij}^{c}=1) = \alpha &+ \beta_1 1(dp_j^{p}=1)\times 1(\alpha_1 < \alpha_{ij}^{c} \le \alpha_2) \\
&+ \beta_2 1(dp_j^{p}=1)\times 1(\alpha_2 < \alpha_{ij}^{c} \le \alpha_3) \\
&+ \beta_3 1(dp_j^{p}=1)\times 1(\alpha_3 < \alpha_{ij}^{c} \le \alpha_4) \\
&+ \beta_4 1(dp_j^{p}=1)\times 1(\alpha_4 < \alpha_{ij}^{c} \le \alpha_5) + \gamma X_{ij} + f_j + u_{ij}
\end{aligned}
\qquad (2)
$$

where $a_1 < a_2$ etc. If exposure time matters, we expect $\beta_1 > \beta_2 > \beta_3 > \beta_4$. In the main analysis we use age intervals 1–15, 16–20, 21–30, and 31–40. The first disability spells in the data start in 1966 when the 1951 (child) cohort was aged 15, hence the upper limit in the first age interval. The next age interval, 16–20, covers the period before most children started to study, and therefore, moved out of their parents' home. In the appendix we also report results for the cohorts 1957–1963 with 1–10 and 11–20 as the first age intervals.[9] The vector of explanatory variables, X_{ij} includes child and parental cohort dummies, birth order dummies, father's and mother's education and age at childbirth, and family income. By controlling for cohorts, we avoid time trends in disability rates leading to spurious results. We estimate (2) by OLS and with siblings fixed effect. It is worth pointing out that with the siblings fixed effect, one is utilizing variation in the data within each family. All the variation between families is "swept out" by the fixed effects. Thus it is likely that the statistical significance decreases. Finally, earlier research on birth order effects, see for instance Black *et al.* (2005), and Lindahl (2008) lead us to expect that younger siblings have a disadvantage. Thus, controlling for birth order may also be crucial.

[9] Alternatively to equation (2), exposure could have been modeled linearly as $\beta 1(dp_j^{p}=1)*a_{ij}^{c}$. The dummy formulation was chosen because a_{ij}^{c} is censored from below, at age 3 for the 1963 cohort, up to age 15 for the 1951 cohort.

5. Results

Table 3 presents the effect of fathers' DP on their children's probability of becoming disability receivers. In the upper block, sons and daughters are pooled, whereafter they are estimated separately (mid and lower blocks).

[Table 3 about here]

Column (1) presents the OLS results, with no controls. As expected, there exists a strong and highly significant effect. From the descriptive statistics in Table 1 we see that 4% of the children in our sample become disability receivers before they turn 40 (3% of the sons and 5% of the daughters, respectively). This probability increases to 4.2% for sons (3% + 1.2%) and to 6.3% for daughters (5% + 1.3%). In relative terms these increases are quite substantial (29% and 21% for sons and daughters, respectively).

However, the revealed correlation does not answer the questions we ask in this paper. First, we want test whether the correlation is related to the time the children are exposed to their parents being disability receivers. For this we use the constructed indicators for different periods of the children's lives: ages 0–15, 16–20, 21–30, and 31–40, respectively. The first indicator covers the period up until high school,[10] the second, the high school period, while the final two represents periods of early and mid-adult life. In other words, we allow social interaction to play a role between and within families also after the children (typically) have left home. Column (2) sheds light on this hypothesis. We find the strongest effect for those children that were exposed early

[10] Too small samples prevent us performing more detailed stratifications.

in their lives and, hence, experienced disabled parents for the longest time. For example, children being exposed at the age of 0–15 have a 6.5% higher probability of becoming DP receivers themselves compared with children of the same age with nonreceiving parents. The effect decreases monotonically the higher the age group and the shorter the exposure.

In columns (3) and (4) we control for possible confounding factors, still within an OLS framework. Recent research indicates that younger siblings are at a disadvantage with respect to outcome in adult life, notably education (Black *et al*. 2005). Hence, we control for birth order's possible association to the receipt of DP (column (3)). As discussed in the introductory section, we do not want to confound fathers' disability with low earnings and low education (controlled for in column (4)). We see that inclusion of controls have only minor effects on our estimates. The coefficient decreases somewhat, particularly for the youngest group.[11] Turning to the differences between sons and daughter in blocks two and three, we see the same pattern as before we divided the effect of fathers' disability into periods of exposure, namely a stronger effect on daughters compared with that on sons.

It is reassuring that our estimates of the effect of different degrees of exposure survive the extension of the analysis to include several individual and parental characteristics. However, the fundamental question of unobserved heterogeneity must be addressed before we can claim any causal interpretation of our findings. It might very well be unobserved characteristics of a certain family, present before as well as after the father receive DP, that is driving the intergenerational correlation; an obvious candidate being bad health inherited from parent to child. Therefore, we include family

[11] It even becomes negatively significant for the oldest group - a result that vanishes in our preferred model.

fixed effects in columns (5) and (6). Hence, we forsake the variation between families in our sample, and isolate any effect that stems from the variation within families. To the degree that genes matter in deciding the correlation, e.g. through inherited weak health, it is integrated out by playing an identical role for all siblings in the respective family.[12] In the pooled sample (upper block) we see that the effect from long exposure diminishes compared with the OLS results, but that is what one might expect when all the between family effects are omitted. The significantly positive effect remains, though, before as well as after controlling for observable confounders.[13] The pattern is more or less the same for sons and daughters, but significant only at the 5% level for sons in the 16–20 age group. The reduced significance is likely to stem from the small sample sizes of age groups relative to the pooled sample.

It should be noted that when we apply the siblings fixed effects model, only siblings who are found in different age intervals contribute to the identification of the exposure variable. There is a trade-off between having more detailed age intervals, and the number of variables to be identified. In Table A1 we report how many of the observations actually contribute to the identification. We see that the variation is smallest in the 0–15 interval, thus there is little scope for finer intervals for those who were exposed at the youngest age.

[Table 4 about here]

[12] As always in fixed-effect models, this rests on the assumption of common trends within each group.

[13] The fixed-effect formulation means that only birth order remains relevant, while parental earnings and education become part of the fixed effect.

In Table 4, we report a similar intergenerational effect between mothers and children. The OLS results resemble the ones for fathers. However, when we turn to the family fixed-effect formulation, the coefficients tend to be smaller in magnitude, and the effect of having a mother on DP is significant only for the 0–15 age group and at the 10% level. When we split the sample into sons and daughters, none of the coefficients are significant at conventional levels. Hence, unobserved family effects appear to play a more important role for the intergenerational correlation between mothers and children than for fathers and children.

So far the reported regressions were based on a sample of siblings. To check whether this introduces any selection problems, we run the various OLS regressions for the full sample including families with only children. The results, reported in Tables A2 and A3, are quite similar to the OLS results in Tables 3 and 4, implying that biases due to the selection of families with more than one child is not an issue.

As noted in the data section, the uneven exposure time intervals used in Tables 3 and 4 reflect data limitations with regard to the first disability spells. If we exclude the 1951–1956 cohorts, we are able to split the exposure dummy at the age of 10 for the remaining cohorts, 1957–1963. These additional analyses are reported in Tables A4 and A5. The point estimates are quite close to the main results. However, the effect of fathers' DP on sons in the first age interval, which was significant in the FE regressions in Table 3, now turns out to be insignificant, possibly because of the reduced sample size.

Family dissolutions may affect the outcomes of the children—it is reasonable that the influence of a parent who moves out could be reduced. Therefore, we have also estimated the sibling model on a subsample of "stable" families to ensure that the parents are living together. The data do not include continuous observations on

parents' marital status, but we have census data from 1970 and 1980, and yearly records from 1987. The 1970 data also include marital duration. Thus we can back-track marital status from 1980–1987 to childbirth, and define the family as stable if the parents are married at all observation points.[14] In the main analysis, 87.5% of the sample belongs to stable families according to this definition. Tables A6 and A7 show the results. In sum, they are quite similar to the main results, but the effect of fathers' DP on sons does not quite survive in the fixed-effect regressions. Mothers' DP still has no effect when controlling for family fixed effects. Taking into account the reduced sample size and the strict stability definition, we conclude that our main conclusions are unchanged by this exercise.

6. Concluding remarks

In this paper we address the growing concern caused by rapidly rising disability rolls in several OECD countries. A particular reason for these concerns is that disability retirement for all practical purposes is an absorbing state. Thus, the increasing number of disability pensioners adds to the burden of unfavorable dependence ratios brought about by demographic changes. We focus on a particular aspect: a possible spillover effect between generations. Such an alleged spillover represents additional costs that are likely to be ignored in an analysis based on individuals instead of families. Our paper attempts to measure empirically to what degree children inherit their parents' propensity of becoming DP receivers. In doing so, we link the literature on disability

[14] The definition of stable depends on the cohort and is stricter for the older cohorts. For the 1951 cohort, our definition implies no parental break-up before offspring are aged 29, age 28 for the 1952 cohort, and so on to age 20 for the 1960 cohort. For the youngest cohorts the age limits are 26, 25, and 24.

insurance to the literature on intergenerational transmission of welfare benefits. DP is social insurance against severe health loss, but the probability of becoming a pension receiver also appears to be influenced by social norms, partly established in the homes and distributed from parents to children.

Norway is typical in the sense that the disability rates have increased dramatically since 1980 while on the other hand, public health has improved; trends similar to those described by Autor (2011) for the US. Fortunately, Norwegian data are also exceptionally rich by being based on full population registers, allowing parent–child links by personal identifiers, and covering a period long enough to address intergenerational correlations. In addition to having a number of control variables, the data also allow us to identify siblings. Thus we can consider the importance of exposure, and apply a family fixed effects model to separate the effect of parental disability receipts from unobserved family characteristics.

Our OLS results indicate a positive correlation in the probability of receiving disability benefits between children and parents. The effect is strongest for the children that experienced disabled parents for the longest time. Turning to the differences between fathers and mothers and sons and daughters, we observe a slightly stronger effect on daughters compared with that on sons, no matter which parent receives the benefit.

In our preferred model we restrict any effect stemming from the variation within and not between families. This integrates out (time invariant) unobserved family characteristics and takes us closer to a causal interpretation of our covariations. The significantly positive effect from father to children remains, before as well as after controlling for observable confounders, as long as we pool sons and daughters. When separating sons and daughters and thereby further reducing the sample size, the pattern

is more or less the same, but barely significant at the 5% level. The mother–children effect does not survive the fixed effect model, however.

The existence of a spillover effect between generations implies that DP is not only a concern for the receiving generation, but also for their children. The apparent pattern revealed in our paper calls for increased effort in preventing disability retirement, particularly in families with children that are facing long exposure time. On the other hand, the same spillover effect represents an extra potential for policies that manage to lower the DP uptake: the positive effect can be carried on to the next generation.

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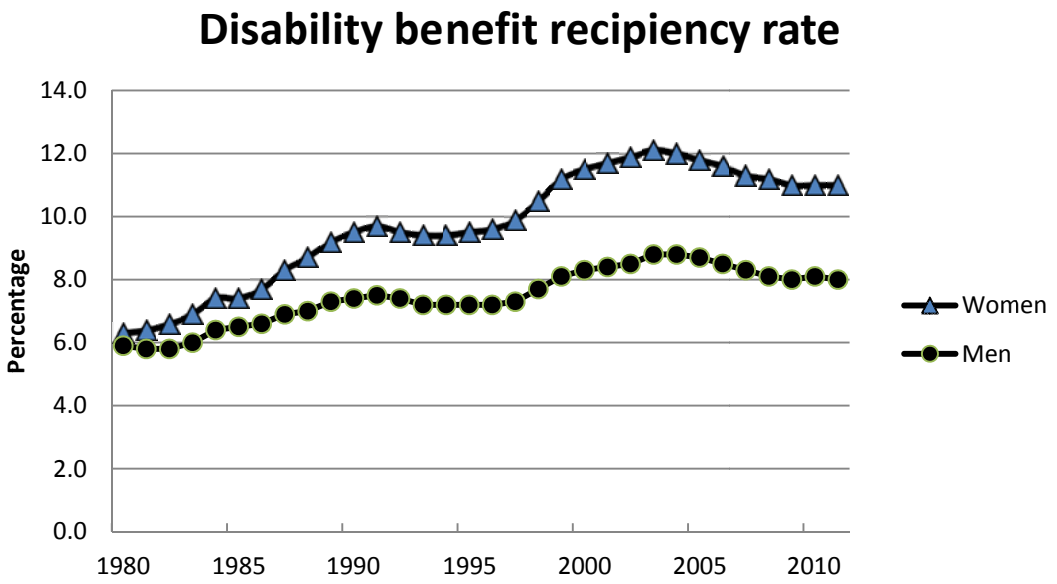
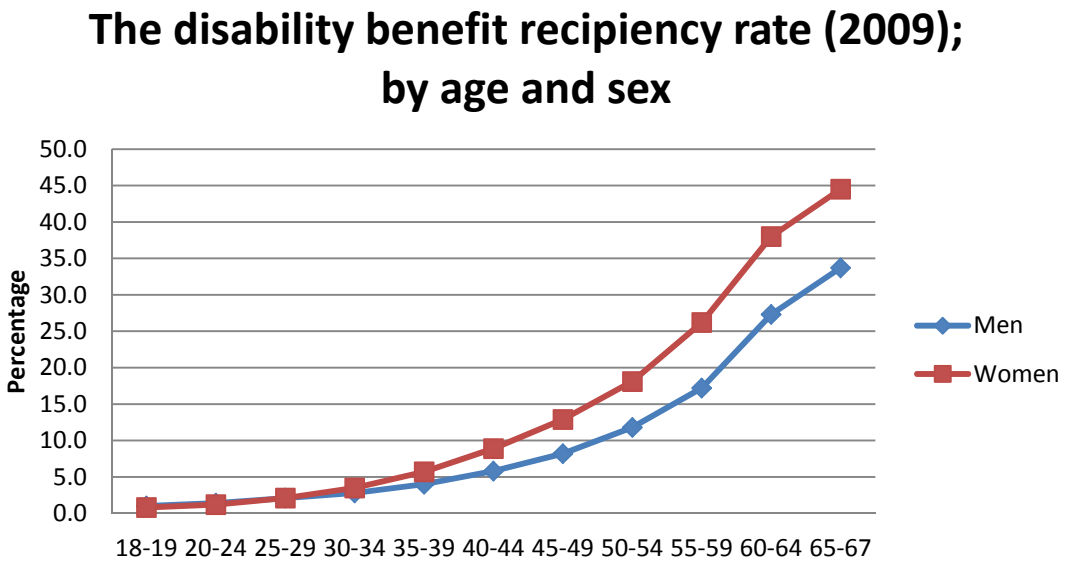
Figure 1:



Notes: The disability benefit recipiency rates are calculated as the number of disability pension recipients as a share of population aged 18–67. For 2011, the number is calculated based on numbers for September.
Source: NAV.

Figure 2:



Notes: The disability benefit recipiency rates are calculated as the number of disability pension recipients as a share of population for each age group.
Source: NAV.

Table 1 Descriptive statistics

	All			Sons			Daughters		
	Obs	Mean	Std. Dev.	Obs	Mean	Std. Dev.	Obs	Mean	Std. Dev.
Female	257,705	0.49	0.50	131,082		0	126,623		0
DP before 40	257,705	0.04	0.20	131,082	0.03	0.17	126,623	0.05	0.22
Father DP	257,705	0.44	0.50	131,082	0.44	0.50	126,623	0.44	0.50
F DP age ≤ 15	257,705	0.02	0.14	131,082	0.02	0.13	126,623	0.02	0.14
F DP age 16–20	257,705	0.03	0.16	131,082	0.03	0.16	126,623	0.02	0.16
F DP age 21–30	257,705	0.18	0.38	131,082	0.18	0.38	126,623	0.18	0.39
F DP age 31–40	257,705	0.21	0.41	131,082	0.22	0.41	126,623	0.21	0.41
Mother DP	257,705	0.46	0.50	131,082	0.46	0.50	126,623	0.46	0.50
M DP age ≤ 15	257,705	0.02	0.15	131,082	0.02	0.15	126,623	0.02	0.15
M DP age 16–20	257,705	0.03	0.17	131,082	0.03	0.17	126,623	0.03	0.17
M DP age 21–30	257,705	0.17	0.38	131,082	0.17	0.38	126,623	0.17	0.38
M DP age 31–40	257,705	0.21	0.41	131,082	0.21	0.41	126,623	0.21	0.41
Birth order 1	257,705	0.41	0.49	131,082	0.42	0.49	126,623	0.41	0.49
Birth order 2	257,705	0.41	0.49	131,082	0.41	0.49	126,623	0.41	0.49
Birth order 3	257,705	0.14	0.34	131,082	0.14	0.34	126,623	0.14	0.34
Birth order 4+	257,705	0.04	0.20	131,082	0.04	0.20	126,623	0.04	0.20
1951 cohort	257,705	0.02	0.14	131,082	0.02	0.14	126,623	0.02	0.13
1952 cohort	257,705	0.03	0.17	131,082	0.03	0.17	126,623	0.03	0.16
1953 cohort	257,705	0.04	0.20	131,082	0.04	0.20	126,623	0.04	0.20
1954 cohort	257,705	0.05	0.23	131,082	0.05	0.23	126,623	0.05	0.23
1955 cohort	257,705	0.07	0.25	131,082	0.07	0.25	126,623	0.07	0.25
1956 cohort	257,705	0.08	0.27	131,082	0.08	0.27	126,623	0.08	0.27
1957 cohort	257,705	0.09	0.29	131,082	0.09	0.28	126,623	0.09	0.29

1958 cohort	257,705	0.10	0.30	131,082	0.10	0.30	126,623	0.10	0.30
1959 cohort	257,705	0.11	0.31	131,082	0.11	0.31	126,623	0.11	0.31
1960 cohort	257,705	0.11	0.31	131,082	0.11	0.31	126,623	0.11	0.31
1961 cohort	257,705	0.11	0.31	131,082	0.11	0.31	126,623	0.11	0.31
1962 cohort	257,705	0.10	0.30	131,082	0.10	0.30	126,623	0.10	0.30
1963 cohort	257,705	0.10	0.30	131,082	0.10	0.30	126,623	0.10	0.30
Father's educ.	257,705	11.94	2.68	131,082	11.95	2.73	126,623	11.93	2.62
Mother's educ.	247,923	8.98	2.10	126,090	8.98	2.10	121,833	8.98	2.09
Father's age	257,705	27.87	4.00	131,082	27.86	4.00	126,623	27.87	4.00
Mother's age	249,814	25.19	4.07	127,032	25.18	4.07	122,782	25.20	4.06
Family income	254,929	10.87	4.57	129,696	10.90	4.59	125,233	10.85	4.55
Father's cohort	257,705	1930.50	3.92	131,082	1930.48	3.92	126,623	1930.53	3.92
Mother's cohort	249,814	1933.20	4.21	127,032	1933.17	4.22	122,782	1933.22	4.20

Notes: Family income in 10,000 1989 NOK, average for child aged 16–19. F/M DP age x–y: child aged x–y when father/mother became disabled.

Table 2 Family characteristics by fathers' disability status

	DP = 0			*DP = 1*		
	Obs	Mean	Std. Dev.	Obs	Mean	Std. Dev.
	All					
Female	145,050	0.49	0.50	112,655	0.49	0.50
Father's educ.	145,050	12.24	2.76	112,655	11.56	2.51
Mother's educ.	139,727	9.26	2.23	108,196	8.62	1.85
Father's age	145,050	27.90	4.03	112,655	27.82	3.96
Mother's age	140,835	25.33	4.07	108,979	25.01	4.06
Family income	144,325	11.78	4.68	110,604	9.69	4.14
Father's cohort	145,050	1930.51	3.90	112,655	1930.49	3.94
Mother's cohort	140,835	1933.10	4.18	108,979	1933.32	4.24
Birth order 1	145,050	0.43	0.49	112,655	0.40	0.49
Birth order 2	145,050	0.41	0.49	112,655	0.41	0.49
Birth order 3	145,050	0.13	0.33	112,655	0.15	0.35
Birth order 4+	145,050	0.04	0.19	112,655	0.05	0.22
	Sons			,		
Father's educ.	73,825	12.23	2.84	57,257	11.58	2.52
Mother's educ.	71,124	9.26	2.23	54,966	8.62	1.86
Father's age	73,825	27.91	4.02	57,257	27.80	3.97
Mother's age	71,665	25.33	4.07	55,367	24.99	4.07
Family income	73,436	11.81	4.72	56,260	9.71	4.14
Father's cohort	73,825	1930.48	3.90	57,257	1930.47	3.94
Mother's cohort	71,665	1933.08	4.19	55,367	1933.30	4.25
Birth order 1	73,825	0.43	0.49	57,257	0.40	0.49
Birth order 2	73,825	0.41	0.49	57,257	0.40	0.49
Birth order 3	73,825	0.13	0.33	57,257	0.15	0.35
Birth order 4+	73,825	0.04	0.19	57,257	0.05	0.22
	Daughters					
Father's educ.	71,225	12.24	2.68	55,398	11.54	2.50
Mother's educ.	68,603	9.26	2.23	53,230	8.61	1.84
Father's age	71,225	27.89	4.03	55,398	27.84	3.96
Mother's age	69,170	25.33	4.07	53,612	25.03	4.04
Family income	70,889	11.74	4.65	54,344	9.67	4.14
Father's cohort	71,225	1930.55	3.90	55,398	1930.50	3.93
Mother's cohort	69,170	1933.13	4.18	53,612	1933.33	4.23
Birth order 1	71,225	0.42	0.49	55,398	0.39	0.49
Birth order 2	71,225	0.41	0.49	55,398	0.41	0.49
Birth order 3	71,225	0.13	0.33	55,398	0.15	0.36
Birth order 4+	71,225	0.04	0.19	55,398	0.05	0.22

Table 3 Effect of fathers' DP on children's DP probability. OLS and family fixed effects (FE)

	OLS				FE	
	(1)	(2)	(3)	(4)	(5)	(6)
		All	***N = 257,705***			
Father DP	0.013					
	14.86					
Female	0.020	0.020	0.020	0.020	0.020	0.020
	25.49	25.47	25.41	25.35	20.23	20.21
F DP age ≤ 15		0.065	0.064	0.043	0.035	0.035
		13.9	13.67	7.86	2.80	2.82
F DP age 16–20		0.057	0.056	0.043	0.036	0.036
		15.46	15.18	11.68	4.22	4.19
F DP age 21–30		0.025	0.025	0.018	0.022	0.021
		20.46	19.84	14.45	3.7.0	3.53
F DP age 31–40		0.008	0.008	0.004	0.014	0.013
		8.32	8.00	4.17	2.70	2.43
		Sons	***N = 131,082***			
Father DP	0.012					
	11.57					
F DP age ≤ 15		0.057	0.056	0.035	0.033	0.033
		9.55	9.36	5.03	1.21	1.22
F DP age 16–20		0.052	0.051	0.038	0.036	0.036
		11.11	10.89	8.23	2.04	2.03
F DP age 21–30		0.023	0.022	0.017	0.024	0.023
		15.09	14.6	10.84	1.91	1.84
F DP age 31–40		0.006	0.006	0.003	0.017	0.016
		4.92	4.73	2.18	1.54	1.43
		Daughters	***N = 126,623***			
Father DP	0.013					
	10.26					
F DP age ≤ 15		0.074	0.072	0.051	0.038	0.039
		10.81	10.64	6.32	1.37	1.39
F DP age 16–20		0.063	0.061	0.049	0.036	0.036
		11.17	10.97	8.55	1.90	1.88
F DP age 21–30		0.028	0.027	0.02	0.021	0.02
		14.67	14.23	10.1	1.60	1.52
F DP age 31–40		0.011	0.01	0.006	0.012	0.011
		6.82	6.56	3.56	1.05	0.92
Controls:						
Birth order			X	X		X
Parents' education, earnings, age				X		
Family fixed effects					X	X

Robust *t*-values clustered by family. Controlled for fathers' and children's cohorts.
F DP age x–y: child aged x–y when father became disabled. Siblings sample, cohorts 1951–1963.

Table 4 Effect of mothers' DP on children's DP probability. OLS and family fixed effects (FE).

	OLS				FE	
	(1)	(2)	(3)	(4)	(5)	(6)
		All	***N = 289,032***			
Mother DP	0.011					
	13.24					
Female	0.020	0.020	0.020	0.020	0.020	0.020
	26.18	26.16	26.13	25.34	20.84	20.82
M DP age ≤ 15		0.057	0.056	0.043	0.018	0.018
		15.81	15.59	11.29	1.90	1.86
M DP age 16–20		0.051	0.051	0.041	0.007	0.007
		17.6	17.31	12.95	1.11	0.99
M DP age 21–30		0.031	0.031	0.025	0.003	0.001
		26.11	25.5	19.63	0.57	0.29
M DP age 31–40		0.011	0.011	0.009	0.001	0.000
		11.86	11.54	8.82	0.31	–0.09
		Sons	***N = 146,976***			
Mother DP	0.008					
	8.44					
M DP age ≤ 15		0.049	0.048	0.032	0.014	0.014
		10.86	10.70	7.10	0.67	0.67
M DP age 16–20		0.041	0.04	0.028	0.006	0.006
		11.22	10.99	7.48	0.44	0.39
M DP age 21–30		0.023	0.023	0.019	0.004	0.003
		16.10	15.67	12.03	0.42	0.28
M DP age 31–40		0.009	0.009	0.007	0.002	0.001
		7.80	7.62	6.03	0.33	0.12
		Daughters	***N = 142,056***			
Mother DP	0.013					
	10.62					
M DP age ≤ 15		0.065	0.064	0.054	0.022	0.022
		12.24	12.07	9.16	1.03	1.00
M DP age 16–20		0.063	0.062	0.054	0.008	0.007
		13.93	13.73	10.80	0.54	0.48
M DP age 21–30		0.039	0.039	0.032	0.001	0.00
		21.21	20.74	15.86	0.08	–0.04
M DP age 31–40		0.014	0.013	0.01	–0.001	–0.002
		9.11	8.86	6.56	–0.07	–0.24
Controls:						
Birth order			X	X		X
Parents' education, earnings, age				X		
Family fixed effects					X	X

Robust *t*-values clustered by family. Controlled for fathers' and children's cohorts.
F DP age x–y: child aged x–y when father became disabled. Siblings sample, cohorts 1951–1963.

APPENDIX

Table A1 Within family variation in exposure to father's or mother's DP.

	Father DP			Mother DP			
Child age when parent becomes DP	Same	Other	Total	Same	Other	Total	Total
Never	142,108	2,408	144,516	154,315	6,024	160,339	160,339
1–15	3,050	1,829	4,879	4,504	2,602	7,106	7,106
16–20	1,652	4,793	6,445	2,664	7,040	9,704	9,704
21–30	23,750	22,722	46,472	28,051	23,647	51,698	51,698
31–40	32,688	22,705	55,393	35,347	24,838	60,185	60,185
Total	203,248	54,457	257,705	224,881	64,151	289,032	289,032

Same: exposed at same age as siblings. Other: exposed at different age from siblings (at least one sibling).

Table A2 Effect of fathers' DP on children's DP probability. Full sample including single children. OLS.

	All			Sons			Daughters		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Father DP	0.013			0.012			0.014		
	17.03			13.13			11.73		
Female	0.021	0.02	0.021						
	29.3	29.25	29.54						
F DP age ≤ 15		0.07	0.05		0.063	0.043		0.077	0.057
		16.67	9.94		11.60	6.65		12.65	7.66
F DP age 16–20		0.057	0.043		0.051	0.036		0.063	0.049
		17.45	13.09		12.29	8.95		12.75	9.76
F DP age 21–30		0.027	0.02		0.024	0.018		0.03	0.021
		24.50	17.40		17.73	12.79		17.68	12.29
F DP age 31–40		0.009	0.005		0.007	0.003		0.011	0.007
		9.94	5.42		6.14	3.03		7.92	4.52
N	334,995	334,995	318,499	170,340	170,340	161,939	164,655	164,655	156,560

Robust *t*-values clustered by family. Controlled for fathers' and children's cohorts. Columns (3) controlled for parents' education and age, and family income.
F DP age x–y: child aged x–y when father became disabled.

Table A3 Effect of mothers' DP on children's DP probability. Full sample including single children. OLS.

	All			Sons			Daughters		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Mother DP	0.01			0.007			0.012		
	13.47			8.09			11.1		
Female	0.02	0.02	0.021						
	30.45	30.44	29.55						
M DP age ≤ 15		0.059	0.047		0.05	0.035		0.068	0.058
		19.03	13.95		12.7	8.71		14.84	11.2
M DP age 16–20		0.053	0.042		0.039	0.027		0.067	0.058
		20.63	15.24		12.48	8.24		16.76	13.06
M DP age 21–30		0.032	0.026		0.024	0.019		0.04	0.033
		30.2	23.09		18.34	14.18		24.6	18.51
M DP age 31–40		0.012	0.009		0.008	0.007		0.015	0.012
		13.89	10.43		8.04	6.19		11.53	8.48
N	374,307	374,307	318,486	190,301	190,301	161,933	184,006	184,006	156,553

Robust t-values clustered by family. Controlled for mothers' and children's cohorts. Columns (3) controlled for parents' education and age, and family income.
M DP age x–y: child aged x–y when mother became disabled.

Table A4 Effect of fathers' DP on children's DP probability. OLS and family fixed effects (FE). Cohorts 1957–1963.

	OLS				FE	
	(1)	(2)	(3)	(4)	(5)	(6)
		All	***N = 183,345***			
Father DP	0.012					
	12.09					
Female	0.02	0.02	0.02	0.02	0.02	0.02
	21.44	21.48	21.43	22.31	14.91	14.9
F DP age ≤ 10		0.062	0.061	0.039	0.022	0.022
		8.16	8.01	4.05	0.94	0.96
F DP age 11–20		0.058	0.057	0.042	0.034	0.034
		16.78	16.48	11.59	2.53	2.49
F DP age 21–30		0.024	0.023	0.017	0.025	0.023
		17.53	16.96	12.13	2.34	2.22
F DP age 31–40		0.007	0.007	0.003	0.02	0.018
		5.70	5.39	2.35	2.05	1.88
		Sons	***N = 92,829***			
Father DP	0.012					
	9.38					
F DP age ≤ 10		0.054	0.053	0.033	0.021	0.021
		5.61	5.48	2.72	0.38	0.39
F DP age 11–20		0.051	0.05	0.035	0.037	0.037
		11.63	11.38	7.82	1.09	1.08
F DP age 21–30		0.022	0.021	0.016	0.026	0.025
		12.94	12.48	9.17	0.99	0.94
F DP age 31–40		0.005	0.004	0.001	0.023	0.021
		2.93	2.72	0.77	0.92	0.86
		Daughters	***N = 90,516***			
Father DP	0.013					
	8.24					
F DP age ≤ 10		0.07	0.069	0.045	0.022	0.023
		6.14	6.05	3.08	0.36	0.37
F DP age 11–20		0.066	0.065	0.049	0.032	0.031
		12.5	12.3	8.77	0.91	0.89
F DP age 21–30		0.026	0.025	0.018	0.024	0.022
		12.39	12.03	8.34	0.89	0.83
F DP age 31–40		0.01	0.01	0.005	0.018	0.016
		5.01	4.78	2.38	0.73	0.65
Controls:						
Birth order			X	X		X
Parents' education, earnings, age				X		
Family fixed effects					X	X

Robust *t*-values clustered by family. Controlled for fathers' and children's cohorts.
F DP age x–y: child aged x–y when father became disabled. Siblings sample, cohorts 1957–1963.

Table A5 Effect of mothers' DP on children's DP probability. OLS and family fixed effects (FE). Cohorts 1957–1963.

	OLS				FE	
	(1)	(2)	(3)	(4)	(5)	(6)
		All	***N = 200,963***			
Mother DP	0.011					
	11.61					
Female	0.02	0.02	0.02	0.021	0.02	0.02
	21.63	21.62	21.59	22.26	15.03	15.01
M DP age ≤ 10		0.057	0.056	0.043	0.021	0.021
		9.85	9.71	6.97	1.00	1.00
M DP age 11–20		0.054	0.053	0.042	0.003	0.002
		19.1	18.75	13.92	0.3	0.23
M DP age 21–30		0.031	0.03	0.025	0.005	0.004
		22.63	22.00	17.10	0.71	0.55
M DP age 31–40		0.011	0.011	0.009	0.004	0.002
		9.3	9.01	6.88	0.66	0.42
		Sons	***N = 101,669***			
Mother DP	0.009					
	7.87					
M DP age ≤ 10		0.057	0.056	0.041	0.014	0.014
		7.38	7.28	5.07	0.24	0.24
M DP age 11–20		0.043	0.042	0.029	0.000	0.000
		12.3	12.04	8.06	0.01	-0.01
M DP age 21–30		0.024	0.023	0.019	0.001	0.000
		14.29	13.84	10.94	0.05	-0.02
M DP age 31–40		0.01	0.009	0.007	0.006	0.004
		6.33	6.15	4.70	0.40	0.30
		Daughters	***N = 99,294***			
Mother DP	0.013					
	8.81					
M DP age ≤ 10		0.057	0.056	0.045	0.029	0.029
		6.81	6.7	4.85	0.56	0.56
M DP age 11–20		0.066	0.065	0.056	0.005	0.004
		15.02	14.78	11.58	0.19	0.16
M DP age 21–30		0.038	0.037	0.030	0.008	0.007
		17.97	17.51	13.38	0.43	0.37
M DP age 31–40		0.013	0.013	0.010	0.001	0.000
		6.95	6.73	5.12	0.09	0.00
Controls:						
Birth order			X	X		X
Parents' education, earnings, age				X		
Family fixed effects					X	X

Robust *t*-values clustered by family. Controlled for mothers' and children's cohorts.
M DP age x–y: child aged x–y when mother became disabled. Siblings sample, cohorts 1957–1963.

Table A6 Effect of fathers' DP on children's DP probability. Stable families. OLS and family fixed effects (FE).

	OLS				FE	
	(1)	(2)	(3)	(4)	(5)	(6)
		All	***N =***			
Father DP	0.011					
	13.2					
Female	0.020	0.020	0.020	0.020	0.020	0.020
	24.58	24.57	24.52	24.38	19.44	19.42
F DP age ≤ 15		0.058	0.057	0.041	0.028	0.028
		11.28	11.07	6.98	2.05	2.08
F DP age 16–20		0.05	0.049	0.041	0.028	0.028
		12.46	12.17	10.16	3.09	3.08
F DP age 21–30		0.024	0.023	0.018	0.02	0.019
		18.6	18.02	13.92	3.23	3.08
F DP age 31–40		0.008	0.008	0.005	0.013	0.012
		8.20	7.89	4.40	2.43	2.18
		Sons	***N = 114,604***			
Father DP	0.01					
	9.65					
F DP age ≤ 15		0.052	0.05	0.033	0.026	0.027
		7.88	7.68	4.47	0.90	0.92
F DP age 16–20		0.045	0.044	0.035	0.032	0.032
		9.03	8.78	7.19	1.65	1.65
F DP age 21–30		0.021	0.02	0.016	0.023	0.022
		13.06	12.6	9.82	1.71	1.63
F DP age 31–40		0.006	0.006	0.003	0.016	0.015
		4.84	4.66	2.07	1.37	1.25
		Daughters	***N = 110,678***			
Father DP	0.013					
	9.56					
F DP age ≤ 15		0.065	0.064	0.049	0.031	0.031
		8.7	8.57	5.66	0.97	0.98
F DP age 16–20		0.055	0.054	0.046	0.024	0.023
		9.09	8.9	7.51	1.18	1.17
F DP age 21–30		0.028	0.027	0.021	0.018	0.017
		13.86	13.46	10.22	1.30	1.23
F DP age 31–40		0.011	0.011	0.007	0.011	0.01
		6.7	6.44	3.94	0.90	0.79
Controls:						
Birth order			X	X		X
Parents' education, earnings, age				X		
Family fixed effects					X	X

Robust *t*-values clustered by family. Controlled for fathers' and children's cohorts.
F DP age x–y: child aged x–y when father became disabled. Parents married throughout child's adolescence.

Table A7 Effect of mothers' DP on children's DP probability. Stable families. OLS and family fixed effects (FE).

	OLS				FE	
	(1)	(2)	(3)	(4)	(5)	(6)
		All	***N = 234,475***			
Mother DP	0.009					
	10.24					
Female	0.02	0.02	0.02	0.02	0.02	0.02
	24.81	24.78	24.73	24.39	19.83	19.82
M DP age ≤ 15		0.047	0.046	0.038	–0.001	–0.001
		11.67	11.44	9.29	–0.05	–0.04
M DP age 16–20		0.045	0.044	0.037	–0.003	–0.003
		13.1	12.82	10.65	-0.38	–0.44
M DP age 21–30		0.029	0.028	0.023	–0.004	–0.005
		21.93	21.33	17.33	–0.83	–1.02
M DP age 31–40		0.011	0.011	0.008	–0.003	–0.004
		10.73	10.39	8.17	–0.85	–1.16
		Sons	***N = 119,263***			
Mother DP	0.006					
	5.66					
M DP age ≤ 15		0.036	0.035	0.028	0.002	0.002
		7.29	7.12	5.75	0.06	0.06
M DP age 16–20		0.028	0.027	0.021	0.002	0.001
		7.22	6.98	5.29	0.09	0.06
M DP age 21–30		0.02	0.019	0.016	0	–0.001
		12.99	12.58	10.18	–0.01	–0.09
M DP age 31–40		0.009	0.008	0.007	–0.002	–0.003
		6.94	6.75	5.28	–0.43	–0.65
		Daughters	***N = 115,212***			
Mother DP	0.012					
	8.82					
M DP age ≤ 15		0.059	0.058	0.048	–0.003	–0.003
		9.47	9.31	7.57	–0.12	–0.12
M DP age 16–20		0.061	0.06	0.053	–0.008	–0.008
		11.3	11.12	9.54	–0.48	–0.50
M DP age 21–30		0.037	0.037	0.03	–0.009	–0.009
		18.17	17.72	14.35	–0.79	–0.88
M DP age 31–40		0.013	0.013	0.01	–0.005	–0.006
		8.33	8.04	6.31	–0.58	–0.74
Controls:						
Birth order			X	X		X
Parents' education, earnings, age				X		
Family fixed effects					X	X

Robust *t*-values clustered by family. Controlled for mothers' and children's cohorts.
M DP age x–y: child aged x–y when mother became disabled. Parents married throughout child's adolescence.

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