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MYOPIC MISERY: MATERNAL DEPRESSION, CHILD INVESTMENTS, AND THE NEUROBIOLOGICAL POVERTY TRAP

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In this paper, I explore in an overlapping generations framework, a mechanism motivating a neurobiological poverty trap. Poverty causes stress and depression in individuals susceptible to depression. Poor and depressed individuals discount the future at a higher rate and invest less in the human capital of their children than mentally healthy or rich individuals. This gene–environment interaction generates a vicious cycle in which poor individuals inherit not only susceptibility to depression, but also stress and poverty. I show that a successful *one-time* intervention has the power to *permanently* eliminate the neurobiological poverty trap.

Keywords: Child Investment, Depression, Discounting, Gene–Environment Interaction

1. INTRODUCTION

This paper proposes a simple theory on the joint intergenerational transmission of mental health, education, and income inequality. The model is based on gene–environment interaction and complements the literature on poverty traps and human capital transmission based on external constraints, such as imperfect credit markets [e.g., Galor and Zeira (1993)] and the child quality–quantity trade-off [e.g., Moav (2005)]. The main components of the theory are the observations that (i) depression is partly caused by low socioeconomic status, (ii) some genotypes are more susceptible to stress than others, (iii) depression increases present bias of parents (mothers), and (iv) the latter causes inferior investments in children through which low economic status and poor mental health are transmitted to the next generation.

Worldwide, around 350 million people suffer from depression [WHO (2015)], but not everyone is equally likely to be afflicted by the disease. Being poor and uneducated significantly increases the risk of becoming and being depressed, with causation running from low socioeconomic status to depression [Lorant et al.

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(2003), Patel and Kleinman (2003), Sareen (2011)]. Since there is also a strong causal association between chronic stress and depression [Hammen (2005)], the obvious pathway runs from poverty to stress, and then to risk of depression. Roy and Campbell (2013) provide an introduction to the biology of the poverty–stress–depression feedback mechanism.

Women are particularly susceptible to depression, and especially after giving birth. Postnatal depression is common in developed countries with prevalence rates of around 7%–13% and occurs even more frequently in developing countries with prevalence rates of up to 30% and more [Fisher et al. (2012), Parsons et al. (2012)]. These high prevalence rates are presumably the result of exposure to poverty and it is argued that low socioeconomic status is a key moderator of the effects of postnatal depression on parenting difficulties and subsequent child development [Fisher et al. (2012), Parsons et al. (2012)].

Susceptibility to depression is largely genetically transmitted with heritability estimates of between 40% and 70% [Lesch (2004)]. Genetic transmission, however, is complex and scholars increasingly believe that the disease is triggered by a gene–environment interaction, which explains why it does not equally afflict all genetically predisposed individuals (or all individuals suffering severe poverty and stress). Evidence for such a gene-by-environment interaction is provided by Caspi et al. (2003) for depression, in general, and by Mitchell et al. (2011) for the case of postnatal depression.

Depression affects economic behavior. It is typically characterized by lethargy, sleep disturbance, deficiency in concentration, and low mood. In particular, sad and depressed individuals discount the future more heavily than others [Ifcher and Zarghamee (2011), Lerner et al. (2013), Pulcu et al. (2014)]. Stress and (maternal) depression are associated with elevated cortisol levels [Hammen (2005)] and subjects administered cortisol have been found to discount the future more heavily [Cornelisse et al. (2013)]. The present bias may reduce the depressed mothers' investment in their children leading them to engage less in child play, reading to children, helping with homework, and general day-to-day interaction. As a result of inferior investments, children of depressed mothers develop fewer cognitive and noncognitive skills [e.g., Patel et al. (2001), Sohr-Preston and Scaramella (2006), Mensah and Kiernan (2010)].

In this paper, I focus on the impact of gene–environment interaction on present bias through depression. In short, the theory works as follows. In an overlapping generation (OLG) setup, parents (mothers) experience utility from consumption (immediate gratification) and from their children's future human capital, which can be increased by investing in child development in the present period. Society is stratified genetically such that only some individuals are prone to depression. If an individual prone to depression is below the poverty line, the gene–environment interaction kicks in, and she becomes depressed due to the stress associated with poverty. Depression increases present bias and lowers the discount factor for the child's human capital. Consequently, depressed mothers invest less in their children. Their children thus inherit not only genetic susceptibility to depression,

but also low human capital and income, paving the way for a subsequent gene–environment interaction of bad health and low socioeconomic status of the next generation. As a result, the dynasty converges toward a locally stable steady state of low income below the poverty line. For individuals who are genetically not predisposed to depression or sufficiently rich, the gene–environment interaction does not come into force. They do not suffer from present bias and invest more in their children such that their dynasties converge toward a high income level above the poverty line. A successful therapy (that eliminates the elevated present bias) or a sufficiently large income transfer (that moves the family out of poverty) is one-time policies that can permanently release a dynasty from poverty.[1]

In the intergenerational transmission process, education plays a double role. It is not only a protective factor for maternal mental health, but it also limits the mental health problems potentially transmitted to the next generation. For example, in the United States, Augustine and Crosnoe (2010) observe a negative association between maternal mental illness and child cognitive development only for less educated women. Similar results are obtained by Di Cesare et al. (2013) with Peruvian data. As a result, "children of more educated mothers will have a double academic advantage: their mothers will be less likely to exhibit depression and they themselves will be less affected by whatever symptoms their mothers do exhibit" [Augustine and Crosnoe (2010, p. 275)]. Vice versa, as shown in this paper, dynasties of low education levels and high genetic susceptibility to depression could be trapped in a vicious cycle of poverty.

In this paper, I discuss two alternative interventions that can break the cycle of poverty, mental health interventions, and income transfers. Mental health interventions address the disabilities associated with depression and try to restore future orientation and to reduce present bias. Financial interventions address poverty as the cause of stress and depression. A meta-analysis of intervention studies concludes a strong impact of health innovations on economic outcomes and a somewhat less conclusive impact of poverty alleviation on mental health outcomes [Lund et al. (2011)]. A recent study by Eyal and Burns (2016) provide a more optimistic outlook on the poverty alleviation channel. This longitudinal study is particularly relevant for the proposed theory, because it addresses the intergenerational transmission of depression. The study focusses on cash grants in South Africa and a sample where the intergenerational transmission of depression from parent to child is on average 33% (and 44% from mother to daughter). Cash grants, which were received unconditionally on the state of mental health, were found to lower the transmission rate by between 12 and 24 percentage points.

This paper is closely related to recent research on behavioral poverty traps [Bernheim et al. (2015), Dalton et al. (2016)] and to the literature on the interaction between wealth and time discounting, and their impact on economic development [e.g., Schumacher (2009), Strulik (2012)]. Haushofer (2011) provides a survey on the neurological foundations. It is also more broadly related to the literature on the role of genes and other physiological characteristics for investment and economic growth [Galor and Moav (2002), Ashraf and Galor (2013), Cronqvist

and Siegel (2015), Dalgaard and Strulik (2015, 2016)] and to a larger literature that discusses how the interaction of adult health and education impacts on economic development. This literature usually focusses on the mortality aspect of health and the Ben-Porath (1967) mechanism, according to which higher adult life expectancy induces investments in adult human capital [see e.g., Cervellati and Sunde (2005), Hazan (2009), Cervellati and Sunde (2013), Hansen and Strulik (2017), Strulik and Werner (2016)] or child human capital [e.g., Zhang et al. (2001), Soares (2005)]. In contrast to this literature, I focus on morbidity and gene–environment interaction. Maternal depression, as opposed to communicable diseases (like HIV) or aging-related diseases that afflict predominantly old people with grown-up children (like cancer), is triggered by poverty of genetically susceptible mothers with young children. The feedback loop that generates the neurobiological poverty trap through the double inheritance of genetic disposition and poverty does not apply to communicable diseases or old-age diseases because either the genetic or the behavioral part is missing.

The remainder of the paper is organized as follows. In Section 2, I set up the basic model and derive the main results. Motivated by the empirical background literature, the basic model assumes that the intervention that moves families out of the neurobiological poverty trap is purely exogenous. The interventions may be provided by governments or NGOs and motivated by limited knowledge or access to depression therapy in developing countries. In Section 3, I discuss an extension where the depressed have unlimited knowledge and access to therapy and where they consider treatment at their own expense. I then derive a poverty threshold below which individuals fail to take up the therapy. Although this decision is optimal from the individual's viewpoint, it is welfare reducing from the society's viewpoint since individuals do not take into account the impact of their behavior on the gene–environment interaction experienced by future generations of their dynasty. Section 4 concludes.[2]

2. THE MODEL

Consider an OLG model consisting of parent–child families. Since reproduction is asexual, as common in the related literature, we could best imagine a society of mother–daughter families. The parent (mother) cares about current consumption c_t and child quality h_{t+1}. Child quality is determined by child investment, i.e., it is observed only after some time (here, next period), whereas consumption provides immediate gratification. For simplicity, utility is logarithmic:

$$u_t = \log c_t + \beta_t \log h_{t+1}. \tag{1}$$

The only difference to the standard OLG model of child investment is that the discount factor β_t, $0 < \beta_t < 1$, is type-specific and potentially time variant.[3]

In order to capture the gene–environment interaction for depression, we assume that there are two types of dynasties. In d^H families, susceptibility to depression

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is high, and in d^L families, it is low. Depression is triggered by socioeconomic stress. Specifically, depression occurs only if a mother is susceptible to depression and her income is below the poverty line $\bar{h}$. Formally, this implies for the discount factor:

$$\beta_t = \beta(d^i, h_t) = \begin{cases} \beta^L & \text{iff } d^H \text{ and } h_t < \bar{h} \\ \beta^H & \text{otherwise.} \end{cases} \tag{2}$$

We measure child quality by human capital, which coincides with income when the production function is linear. Human capital is produced with decreasing returns to child investments, comprising not only education, but also other (early) investments in cognitive and noncognitive child development. Child investment consists of a part explicitly stemming from the parent e_t and an exogenous part σ, $\sigma \geq 0$, comprising, for example, compulsory education, learning subsistence agricultural techniques from observing and helping peers, etc. As such, the human capital production function is given by

$$h_{t+1} = (\sigma + e_t)^\gamma, \quad 0 < \gamma < 1. \tag{3}$$

Through expenditure on consumption and child investment, the parent maximizes (1) subject to (3) and her budget constraint,

$$h_t = c_t + e_t. \tag{4}$$

The solution is

$$e_t = \begin{cases} \dfrac{\gamma\beta_t h_t - \sigma}{1 + \gamma\beta_t} & \text{for } \beta_t > \sigma/(\gamma h_t) \\ 0 & \text{otherwise.} \end{cases} \tag{5}$$

There is, as usual, intergenerational transmission of human capital: Child investment is a positive function of parental human capital. The important feature here is that child investment is also an increasing function of the discount factor β_t and that there exists a discount factor sufficiently low such that the parent fails to invest in her child. Notice, furthermore, that the threshold for the critical discount factor is declining in human capital of the parent.

Dynamics depend on whether the solution (5) is interior or at the corner. If the solution is interior, human capital according to (3) evolves as

$$h^i_{t+1} = \left[\frac{\gamma\beta(d^i, h_t)}{1 + \gamma\beta(d^i, h_t)}\right]^\gamma (h_t + \sigma)^\gamma, \qquad i = L, H. \tag{6}$$

The main results are summarized in the following propositions.

PROPOSITION 1 (Neurobiological Poverty Trap). *Dynasties that are genetically susceptible to depression (d^H) and poor ($h < \bar{h}$) converge to a locally stable steady state of low income $h^*(\beta^L)$. Dynasties that are not genetically susceptible*

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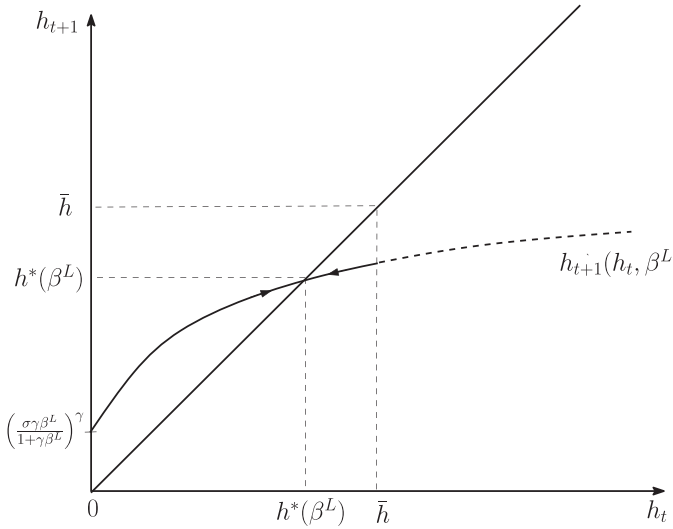

FIGURE 1. Neurobiological poverty trap.

to depression or not poor converge to a locally stable steady state of high income $h^*(\beta^H) > h^*(\beta^L)$.

PROPOSITION 2 (Big Push). *A sufficiently large one-time intervention, which could be either a therapy (restoring* β^H*) or a cash transfer (such that* $h_t > \bar{h}$*) permanently eliminates the neurobiological poverty trap.*

For the proof of Proposition 1, consider a diagrammatic exposition of the law of motion (6). The right-hand side (RHS) of (6) is a strictly concave function of h_t originating from $\gamma\beta^i/(1+\gamma\beta^i)^\gamma\sigma^\gamma \geq 0$, $i = L, H$. This means that there exists for any β^i a unique positive steady state. In Figure 1, the steady state is identified where the h_{t+1}-curve intersects the identity line (along which $h_{t+1} = h_t$). Since the h_{t+1}-curve lies below the identity line for large h_t and above the identity line for small h_t, the steady state is locally stable; dynasties with initial human capital above the steady state (but below the poverty line) converge from above, whereas dynasties with initial endowment below steady-state level converge from below (as indicated by arrows in the diagram).

The position of the steady state is situation specific. Figure 1 shows the dynamics for a dynasty susceptible to depression. Human capital is sufficiently low (below $\bar{h}$) such that socioeconomic stress triggers depression. Child investment of the depressed mother is low to the extent that her grown-up offspring remains below the poverty threshold such that the offspring herself also invest too little in her child. This means that, through a gene–environment interaction, susceptible children do not only inherit depression, but also low socioeconomic status from their mother. The gene–environment interaction, together with

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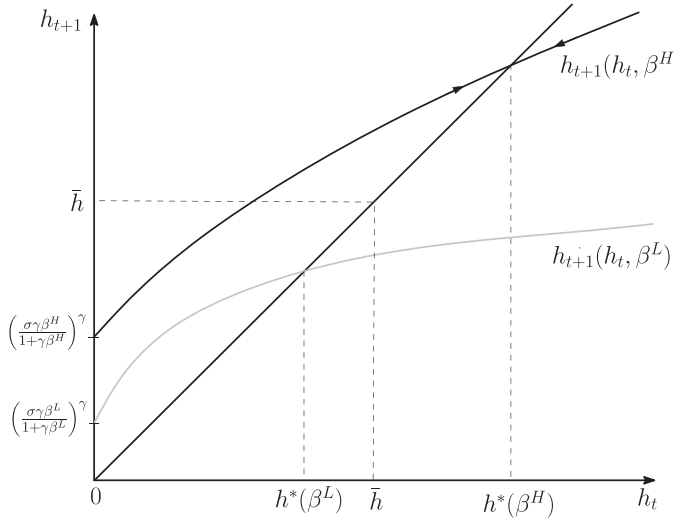

FIGURE 2. Escape from the neurobiological poverty trap.

decreasing returns of child investments, generates convergence toward the poverty trap $h^*(\beta^L)$.

The steady state $h^*(\beta^L)$ is a poverty trap because sufficiently rich individuals, with income above $\bar{h}$, do not develop stress from poverty and depression. Instead of following the dashed branch of the $h_{t+1}(h_t, \beta^L)$-curve, they follow the $h_{t+1}(h_t, \beta^H)$-curve, as shown in Figure 2. Since $\beta^H > \beta^L$, the $h_{t+1}(h_t, \beta^H)$-curve originates from a higher value and is steeper, implying that it intersects the identity line at a higher steady state $h^*(\beta^H)$, which is found to be locally stable by applying the same arguments as above. Intuitively, rich and genetically nonsusceptible individuals do not suffer from depression and present bias and thus invest more in their children, promoting convergence to the higher steady state of income and education.

From these observations, the proof of Proposition 2 follows immediately. A successful depression therapy—in the sense that it restores β^H—eliminates the present bias such that poor individuals invest a sufficient amount into their children to promote convergence toward $h^*(\beta^H)$. In this case, the $h_{t+1}(h_t, \beta^H)$-curve of Figure 2 applies at all income levels. The theory also provides a mechanism explaining why cash grants were found to reduce maternal depression and increase child investments. A sufficiently large income transfer moves family income above $\bar{h}$ and eliminates poverty as the trigger of stress and depression. In this case, the $h_{t+1}(h_t, \beta^H)$-curve of Figure 2 applies at all income levels above $\bar{h}$.[4]

Although the dynasty is still susceptible to depression, the trigger of low socioeconomic status has been removed, and the dynasty gradually converges to $h^*(\beta^H)$ where it shares the steady state with the initially rich and nondepressed dynasties. At the steady state, small negative shocks do not retrigger depression.

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Only drastic shocks (natural disasters, disease outbreak) that push income below the poverty threshold $\bar{h}$ would cause a relapse into the neurobiological poverty trap.

A successful outcome is reached independently from whether the therapy actually cures depression or whether it has an impact on child investment and is obtained only through encouraging thinking about the child's future and bonding with the child (captured in reduced form by the restored β^H). Alternatively, the intervention could address the child. In the model, this would be captured by an increase of exogenous child investment σ. Diagrammatically, an increase of σ shifts the $h_{t+1}(h_t, \beta^L)$-curve upward and if the shift is large enough, subsequent generations converge toward a steady state above the poverty line.[5]

A particularly strong case of the poverty trap occurs if $\gamma\beta^L\bar{h} < \sigma$ and $h_t < \bar{h}$. In this case, a poor dynasty susceptible to depression stagnates in an equilibrium without any private child investment efforts (the corner solution).

Greater realism (but not more insight into mechanisms) could be added by replacing the simple deterministic model with one where the income threshold at which stress and depression is triggered is individual specific and stochastic. Given a distribution of thresholds according to which income-related stress is more likely to be below the poverty line, the model would generate the result that depression also afflicts some rich people, but it would be more prevalent among the poor. The point is that only the poor would transmit their genetic disposition *and* poverty to the next generation. The depressed rich would invest somewhat less in their children but (most of them) not sufficiently little to move their children into poverty. To see this formally, notice that h_{t+1} in the law of motion (6) depends directly on the parent's income and education (h_t). This means that the gene–environment interaction that perpetuates poverty across generations would remain intact (aside from some stochastic perturbation).

3. EXTENSION: OUT OF POCKET THERAPY COSTS

Although interventions to cure or mitigate maternal depression in developing countries may be motivated by the patients (alleged) lack of knowledge or lack of access to therapy, it is also illuminating to consider the case of unlimited knowledge and access and to investigate to what extent fully rational individuals would then not take up a therapy because of poverty. For that purpose, assume that there exists a therapy that cures depression at cost τ, borne by the patient. If an individual takes up the therapy, the budget constraint modifies to $h_t = c_t + e_t + \tau$ and the discount factor becomes β^H, implying utility $U^T = \log(h_t - e_t - \tau) + \gamma\beta^H \log(e_t + \sigma)$. From the first-order condition, we obtain the solution

$$e_t = \frac{\gamma\beta^H(h_t - \tau) - \sigma}{1 + \gamma\beta^H}, \qquad c_t = \frac{h_t + \sigma}{1 + \gamma\beta^H}, \tag{7}$$

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such that indirect utility is obtained as

$$U^T = \log\left(\frac{h_t+\sigma}{1+\gamma\beta^H}\right) + \gamma\beta^H \log\left(\frac{\gamma\beta^H}{1+\gamma\beta^H}\right) + \gamma\beta^H \log(h_t+\sigma-\tau). \quad (8)$$

If a poor and depressed individual does not take up the therapy, the discount factor is β^L and the solution coincides with the one from Section 2. Notice that consumption is unambiguously larger without therapy, since $\beta^L < \beta^H$, whereas the effect on child investment is ambiguous. The therapy increases child investment by reducing the present bias of the individual. It reduces child investment because of the cost of therapy, which reduces the resources available for child investment.

To add more realism, suppose there exists an additional disutility from depression of $D \geq 0$ such that a greater discount factor is not the only motivation to take up therapy. As a result, indirect utility without the therapy is obtained as

$$U^W = \log\left(\frac{h_t+\sigma}{1+\gamma\beta^L}\right) + \gamma\beta^L \log\left(\frac{\gamma\beta^L}{1+\gamma\beta^L}\right) + \gamma\beta^L \log(h_t+\sigma) - D. \quad (9)$$

Comparing utilities, the individual takes up therapy if

$$\text{LHS} \equiv \gamma\beta^H \log(h_t+\sigma-\tau) + \tilde{D} > \gamma\beta^L \log(h_t+\sigma) \equiv \text{RHS}, \quad (10)$$

$$\tilde{D} \equiv D - \log\left(\frac{1+\gamma\beta^H}{1+\gamma\beta^L}\right) + \gamma\beta^H \log\left(\frac{\gamma\beta^H}{1+\gamma\beta^H}\right) - \gamma\beta^L \log\left(\frac{\gamma\beta^L}{1+\gamma\beta^L}\right), \quad (11)$$

where $\tilde{D}$ is a constant that may or may not be larger than D.

PROPOSITION 3. *For positive costs of depression therapy, there exists always a poverty threshold h^C such that individuals with income below h^C refrain from taking up the therapy.*

For the proof, consider a diagrammatic exposition of condition (11). The left-hand side (LHS) and the RHS of the condition are both concave functions of income h_t. LHS is steeper since $\beta^H > \beta^L$ and is thus situated above RHS for sufficiently large h_t. Moreover, LHS has a pole where $h_t = \tau - \sigma$, as shown in Figure 3. At the pole, LHS equals infinity, whereas RHS assumes a finite value. Thus, LHS is situated below RHS in the neighborhood of the pole [LHS is negative when $\tilde{D} < -\gamma\beta^H \log(h_t+\sigma-\tau)$]. Together, this means that there exists a unique intersection h^c, at which LHS=RHS. For income below the thus defined therapy threshold h^c, individuals refrain from taking up therapy because the benefits—from the perspective of the individual—fall short of the costs.

Intuitively, when income and thus consumption is low, the marginal utility from consumption is high. Since the therapy entails lower consumption, individuals with sufficiently low income (and education) do not take up the therapy. Notice

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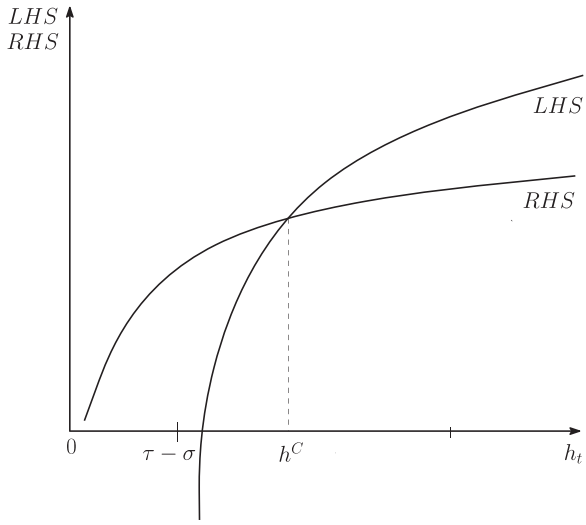

FIGURE 3. Therapy threshold.

that the individual is concerned about the human capital of her child but neglects the utility of more distant future generations. This imperfection originates from the OLG setup and would not occur if the individual was planning for the entire dynasty up to the infinite future [as in Becker and Barro (1988)]. It creates a further motive for intervention. Even in the case where fully rational individuals with perfect knowledge and access to therapy control the take up of therapy, an intervention (costless therapy or income transfer) may be desirable because poor individuals do not take into account that a one-time therapy could be sufficient to escape from depression of the whole dynasty.

4. CONCLUSION

This paper proposed a neurobiological poverty trap that explains how depression can generate a feedback loop according to which bad health and low socioeconomic status are inherited from one generation to the next. Sufficiently strong health interventions could break the gene–environment interaction and enable a permanent escape from poverty of the afflicted dynasty. For simplicity, the analysis focussed on a deterministic model. A longer text would consider the stochastic transmission of mental health, taking into account that genetic disposition and low socioeconomic status do not inevitably trigger depression but "only" increase the risk. Likewise, the kinked child investment function should be conceptualized as a first approximation of a smooth nonlinear function. Assuming, as suggested by Becker et al. (1990), that the rate of return on child investment is low when there is little human capital and then growing as human capital increases would produce qualitatively similar results.

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NOTES

1. Of course, this bold conclusion depends on the assumption that depression is caused by poverty and not associated with or triggered by other health problems of the parent. Moreover, the escape from the neurobiological poverty trap does not preclude entrapment into another poverty trap, for example, due to credit constraints for education. Multiple poverty traps and their interactions are not considered in the present paper.

2. In this paper, I focus on the impact of depression on discounting. A longer version of the paper, available on request, considers a complementing channel by showing that low aspirations by depressed individuals can generate a similar intergenerational feedback loop between depression and low socioeconomic status.

3. This means that all variables are type specific as well. In order to avoid notational clutter, a type index for variables is generally omitted and only introduced when needed for clarification.

4. If the dynasty rests at the steady state, the size of the transfer needed for escape from the poverty trap is given by the distance $\bar{h} - h^*(\beta^L)$ in the diagram. For the special case where $\sigma = 0$, it can be computed analytically as $\bar{h} - [\gamma\beta^L/(1+\gamma\beta^L)]^{\gamma/(1-\gamma)}$.

5. In the case of education, an increase in public investment could be motivated by the self-interest of other strata of society who are not afflicted by poverty and (to a lesser degree) by depression but who would benefit from mass education as, for example, the capitalists in Galor and Moav (2006). This implies that the take-off of public education would be associated with an "unintended" decline in depression. Although there exist no data on the prevalence of depression during industrialization, the earliest available quantitative study for the United States [Jarvis (1855)] suggests that mental illness was more prevalent at the dawn of mass education than today.

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