Understanding Mechanisms of Persistence in Prosocial Behavior: Evidence from a Large-Scale Field Experiment

Understanding Mechanisms of Persistence in Prosocial Behavior: Evidence from a Large-Scale Field Experiment*

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Abstract

We test whether asking individuals to donate blood leads to a persistent change in behavior, and examine the underlying mechanism. In a field experiment, we randomize a phone call, asking potential blood donors to turn out, and follow them over up to 18 months. We observe significant behavioral persistence for at least one year. Using naturally occurring adverse weather conditions as a second instrument for donor turnout allows us to distinguish between action-based persistence (or habit formation in Stigler and Becker, 1977) and motivation-based persistence. Our results strongly favor action-based persistence as the underlying mechanism.

Keywords: Prosocial behavior, Behavioral persistence, Habit formation, Field

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1 Introduction

Can temporary interventions have persistent effects on behavior? The standard model in economics predicts that behavior depends on incentives individuals are presently facing. However, in landmark contributions, Stigler and Becker (1977), and Becker and Murphy (1988) show how complementarities between current and past consumption can produce more complex behaviors, in which past and future incentives affect current behavior. Becker, Grossman and Murphy (1991), and Gruber and Kőszegi (2001) show that this mechanism is quantitatively important for addictive behaviors, such as cigarette consumption. More recently, studies demonstrate that policy interventions, such as price incentives, can induce behavioral change beyond the period during which the intervention is in place in a variety of domains.[1]

Yet, while the proposed mechanism of complementarities is well founded for some applications, e.g., addictive consumptions (Becker and Murphy 1988), the previous literature has made little attempt to distinguish it from other potential mechanisms. On the one hand, persistence may be *action-based*, or habit-forming in the terminology of Stigler and Becker (1977): engaging in an activity in the past increases the marginal utility of engaging in the same activity today. On the other hand, persistence can be *motivation-based*, if an intervention changes an individual's motivation for the activity over several periods, by, e.g., making its benefits more salient, irrespective of whether the individual initially engaged in the activity (Anderson, Laurent and Yantis 2011; Anderson 2016). Distinguishing between these two mechanisms matters for optimally targeting policies both in terms of the timing and type of intervention. If the persistence is driven by past actions, it is optimal to front-load interventions in order to generate a large initial impact. By contrast, if the persistence is driven by lasting changes in motivation, then spreading the intervention out over time is optimal (see, e.g., Byrne et al. 2019).

In this paper, we study whether asking individuals to donate blood leads to a persistent change in turnout, and whether this persistence is action- or motivation-based.[2] We present a theoretical framework that formalizes how the action of making a donation and changes in the donors' motivation can both lead to persistence in prosocial behavior. Our framework offers

[1]For instance, Charness and Gneezy (2009) and Acland and Levy (2015) pay individuals to exercise in the gym; Loewenstein, Price and Volpp (2016) provide children small financial incentives to eat fruits or vegetables; Yang and Lim (2017) rebates subway tickets to shift commuters to off-peak times; Hussam et al. (2022) find persistent changes in hand washing following a temporary incentive to do so.

[2]More recently, behavioral interventions have become popular as policy tools (Obama 2015; Benartzi et al. 2017). A particularly successful example is making a direct ask to elicit a desired behavior (Freeman 1997; Andreoni and Rao 2011; Andreoni, Rao and Trachtman 2017; Milkman et al. 2011, 2012; Adena and Huck 2020).[3]

two possible mechanisms that can lead to persistent effects of a one-time policy intervention. The first mechanism represents action-based persistence. It features a parameter, γ, to quantify the extent to which past donations increase the marginal utility of present donations as in Stigler and Becker (1977). The other possible mechanism is motivation-based. It is possible that a policy intervention impacts motivation over an extended period of time, even if it didn't cause an immediate donation. The framework defines our empirical strategy to discriminate between the two mechanisms. We treat action-based persistence as the null hypothesis and motivation-based persistence as the alternative. It also provides us with the structure to identify the key parameter of the model of action-based persistence, and shows how γ is key to welfare analysis of prosocial behavior with persistence.

Voluntary blood donations are a textbook example of an important prosocial behavior in the real world. Donating blood entails substantial personal costs in terms of time and discomfort, but benefits a large number of anonymous recipients. Moreover, most developed countries rely exclusively on voluntary blood donations (World Health Organization 2011), making effective interventions particularly relevant.[4]

We conduct a field experiment among voluntary blood donors at the Blood Transfusion Service of the Red Cross in Zurich, Switzerland (BTSRC) between 2015 and 2016. All individuals in our study have donated at least once and registered at the BTSRC before the onset of the study. We consider these individuals as potential donors in our study. Every six months, they receive a letter inviting them to an upcoming blood drive on a specific date. In addition, they also receive a text message on their mobile phone as a reminder one day before the blood drive they were invited to takes place.

The field experiment focuses on a subset of 1,400 donors who did not show up at any blood drive they were invited to for at least one year before the onset of the study.[5] We randomly assign each of these inactive donors to one of four experimental conditions. The intervention is to ask them via phone to commit to participating in the blood drive two days from the time being. Donors in conditions C1 and C2 receive only one phone call and are

[4]Blood donations in our setting are also well-suited to study prosocial behavior in general. First, the Red Cross is the only blood collection agency in the canton of Zurich. This makes it difficult to substitute away from any interventions, due to, e.g., ask avoidance (Damgaard and Gravert 2018; Adena and Huck 2020; Andreoni, Rao and Trachtman 2017; DellaVigna, List and Malmendier 2012). Second, a blood donation is in kind and does not contribute to the Red Cross' overhead, thus ruling out overhead aversion (Gneezy, Keenan and Gneezy 2014). Third, understanding persistence in prosocial behavior is important because it implies that temporary interventions will have benefits in the future. Further, continued engagement in prosocial behavior is essential for the formation of social capital (Putnam 1995).

[5]Inactive donors are of particular interest to the BTSRC for two reasons. First, inactive donors make up the majority of the pool, accounting for 60% of all registered donors at any given time. Meanwhile, more than 80% of the registrated donors are inactive at least for some period. Second, the maximum permitted frequency of donating blood at most every three months does not constrain inactive donors at the beginning of the study.

asked to donate in period 1 and 2, respectively. Donors in condition C12 receive two phone calls and are asked to donate in both periods 1 and 2. Donors in the control condition do not receive any phone call. The random variation in the intervention allows us to isolate its effects on behavioral persistence from other causes of serial correlation in donation rates, such as unobserved changes in the environment. We track the behavior of these potential donors over four invitation periods lasting six months each.

The reduced-form evidence indicates that the intervention has a persistent positive effect on donation rates for at least one year. Asking potential donors to make a donation at the upcoming blood drive increases their probability to donate by 18 to 26 percentage points, depending on the specification ($p < 0.01$ in all specifications). The impact of the intervention persists over time: six months later, the probability to donate is still 8 to 17 percentage points higher ($p < 0.01$); twelve months later, it is 5 to 13 percentage points higher ($p < 0.05$).

Next, we distinguish between action-based persistence and motivation-based persistence as the underlying mechanism. Receiving a phone call could lead to a donation in the present as well as directly affect the motivation to donate in the future, e.g., by making the social returns of donating more salient. More formally, if both mechanisms were at work, the intervention would not only affect a donor's current motivation but also their future motivation over several periods and, therefore, violate the exclusion restriction as an instrument to isolate action-based persistence.

We can test the exclusion restriction through an overidentifcation test that requires a second instrument for future donations. This second instrument must affect the donor's probability to donate in the present, but not their future motivation. We use day-to-day variation in weather as such an instrument.[6] We construct an indicator for adverse weather based on rainfall exceeding 10mm, cold days with temperature below 5 degrees Celsius, or heat days with average temperature above 25 degrees Celsius on the day of the blood drive. We use this indicator as the second instrument. The weather instrument has two key characteristics. First, it exogenously influences the costs (or the opportunity cost) of donating blood at the affected blood drive, e.g., by increasing the duration and discomfort of the commute, or by forgoing outdoor activities on a nice summerlike day. Second, adverse weather today plausibly leaves the motivation to donate blood six months later unaffected.

The overidentification test relies on the following intuition: if the intervention of asking donors to make a donation at the upcoming blood drive satisfies the exclusion restriction, then we should find similar estimates of persistence, regardless of which instrument we use.

[6]Naturally occurring weather conditions such as rainfall have been used as exogenous shocks in several contexts in the economics literature (e.g. Miguel, Satyanath and Sergenti 2004; Maccini and Yang 2009; Brückner and Ciccone 2011). In a setting similar to ours, Fujiwara, Meng and Vogl (2016) use daily rainfall on election days as an instrument for voting.

If, for example, the phone call had a positive direct effect on future motivation, then the estimated persistence in blood donations would be larger when we compare the estimate obtained via this instrument to the one obtained via the weather instrument.

The result of the overidentification test does not reject the null hypothesis that both indicators are valid instruments ($p = 0.94$). We obtain similar estimates regardless of whether we use the combined instrument of adverse weather conditions or each of its three components (heavy rainfall, coldness, heat) alone. While overidentification tests are known to have power issues in small samples (Bowsher 2002), simulation studies for our setup indicate that it is exceedingly unlikely to obtain test statistics as favorable to the null hypothesis as ours with even minor violations of the exclusion restriction. Therefore, we conclude that the persistent increase in the future turnout from asking donors is action-based, i.e., driven by the action of donating blood.

We also replicate the same analysis in a larger sample that implements a slightly different intervention that also asks potential donors to make a donation via a phone call. When we apply the same strategy to perform an overidentification test in this separate sample, we arrive at the same conclusion: there is no evidence of a violation of the exclusion restriction by the phone call.

Having confirmed action-based persistence as the underlying mechanism, we impose the structure of our theoretical framework to estimate the corresponding parameter γ. Our most conservative estimate of γ is 0.484 ($p < 0.01$). That is, asking increases the donation rate not only by at least 18 percentage points in the present period, but also in future periods: by $0.484 \times 0.18 = 9$ percentage points six months later, by $0.484^2 \times 0.18 = 4$ percentage points one year later, and so on.

Our study extends three strands of literature. First, it contributes to the literature that analyzes persistent effects of interventions on prosocial behavior. This literature has focused primarily on charitable donations. Meer (2013) shows that there is persistence in donations to universities, using their football team's past success as an instrument. Landry et al. (2006) provide lottery incentives to charity donors in a field experiment. They notice that the long-run effect of such an incentive on donations depends on whether the lottery signals good quality of the charity. In a related study, Landry et al. (2010) compare the effects of a door-to-door fund raiser, a small gift, and a large gift on charitable donations. The study finds that donors initially attracted by features that signal charitable quality are weakly more loyal in the future than donors attracted by a simple ask for money. Meier (2007) investigates the effects of matching charitable donations in a field experiment. He finds that donations increase while being matched but drop below the baseline after the matching ends – suggesting that such incentives can undermine the donors' intrinsic motivation. Adena

and Huck (2019) find that while anticipating a future fundraising letter reduces the current donation, future donations are higher if a donation was made in the current period, which suggests that habit formation may play an role.

Second, our study contributes to an emerging strand of literature that examines habit formation as one of the mechanisms behind persistence in various behaviors. Most of the evidence on habit formation comes from daily activities such as food consumption (Naik and Moore 1996; Fuhrer 2000; Carrasco, Labeaga and Lopez-Salido 2005), energy and water consumption (Allcott and Rogers 2014; Byrne et al. 2019), and handwashing (Hussam et al. 2022; Steiny Wellsjo 2021). More closely related to our setup are studies of persistence in voting. Gerber, Green and Shachar (2003) observe in a field experiment that urging registered voters to participate in the current election through direct mail or face-to-face canvassing increases turnout in the next election. Fujiwara, Meng and Vogl (2016), however, point out that this observed persistence in voting could be driven by a shift in voters' motivation rather than by habit formation. Similar to our study, they exploit random fluctuations in regional rainfall patterns as the instrument for voting, and still find strong evidence of persistence. Our study contributes to this literature by proposing an explicit test of whether an intervention affects future behavior through engaging in the action per se or via a persistent change in the underlying motivation.[7]

Finally, our study contributes to the strand of literature on voluntary blood donations. Recent papers have mainly focused on the role of incentives (Goette and Stutzer 2020; Goette et al. 2009; Lacetera, Macis and Slonim 2012a, 2013) and social interactions among blood donors (Bruhin et al. 2020). We show that a temporary intervention that pushes inactive donors to make a donation in the present also increases their future turnout, even after the intervention is terminated. Taking this into account, blood donation services could make their interventions to manage donor turnout more effective.

The paper is organized as follows. Section 2 illustrates the theoretical framework incorporating the two potential mechanisms behind the persistence in blood donations. Section 3 describes the experimental setup and data. Section 4 presents the reduced form evidence. Section 5 disentangles the two mechanisms and replicates our findings in a larger sample ex-

[7]The action-based persistence in our setup is also in line with consistency-based compliance discussed in the psychology literature (Freedman and Fraser 1966; Cialdini, Trost and Newsom 1995; Cialdini and Trost 1998; Cialdini and Goldstein 2004). This literature suggests that individuals have a need to maintain a positive self-concept and act consistently with their self-views and prior commitments in order to serve the ultimate motivation of maintaining or enhancing their self-esteem. This preference for consistency would make a future blood donation more desirable for individuals who donated already in the past. Similarly, in the planner-doer model of moral standards (Dal Bó and Terviö 2013), individuals build an introspective reputation for "moral capital" which reinforces intrinsic motivation, and therefore, a good action leads to sustained good behavior.

ploiting a quasi-experiment with a similar intervention. It also discusses the statistical power of the overidentification test. Section 6 structurally estimates the parameter that governs the extent of action-based persistence. Section 7 discusses the long-term treatment effects and links them to the underlying mechanisms in a broader literature. It also discusses the general concern of using weather conditions such as rainfall as instruments. Finally, Section 8 concludes the paper.

2 Mechanisms behind Behavioral Persistence

This section discusses the two potential mechanisms — action-based persistence and motivation-based persistence. Our theoretical framework features action-based persistence à la Stigler and Becker (1977) as our null hypothesis and motivation-based persistence as an alternative, which we discuss to highlight the identification problems. Subsequently, we focus on action-based persistence and discuss its dynamic treatment effects and welfare implications.

2.1 Theoretical Framework

In our theoretical framework, a potential donor's contemporaneous utility of donating blood in period t is a function of i) whether she donates in the current period, d_t; ii) two components, d_{t-1} and B; and iii) a random cost shock, $\tilde{c}_t$:

$$u_t = u(d_t, d_{t-1}, B_t, \tilde{c}_t). \tag{2.1}$$

The first component, d_{t-1}, represents the action of donating in the previous period.[8] The second component, B_t, represents the potential donor's baseline motivation to donate, which does not depend on previous donations but may, nevertheless, be influenced by policy interventions. The random cost shock, $\tilde{c}_t$, follows a distribution with cdf F_c. Time is discrete as in the model of habit formation by Stigler and Becker (1977), and we abstract from discounting.

2.1.1 Action-based Persistence

With action-based persistence, the intertemporal dependency is created through past action, whereas baseline motivation B stays constant. The potential donor's contemporaneous

[8]Notice that this does not imply that persistence only lasts one period as it can have indirect effects on future donations. With our specification , an initial behavioral impulse can lead to streaks of donations over several periods, as a donation in period t reinforces the motivation to donate in the next, and so on. Additionally, the component d_{t-1} could be generalized to $A_t = f(d_{t-1}, d_{t-2}, ..., d_{t-T})$, representing a general form of the habit formation where the habit stock does not fully depreciate after one period.

utility in period t can be expressed as

$$u(d_t, d_{t-1}, B, \tilde{c}_t) = \begin{cases} \gamma d_{t-1} + B - \tilde{c}_t & \text{if } d_t = 1 \\ 0 & \text{if } d_t = 0 \end{cases} . \tag{2.2}$$

The parameter γ governs action-based persistence, that is, the impact of a previous donation on the marginal utility of a present donation. The potential donor makes a donation in period t if

$$u(d_t = 1) - u(d_t = 0) = \gamma d_{t-1} + B - \tilde{c}_t \geq 0. \tag{2.3}$$

As $\tilde{c}_t$ is a random cost shock, the probability of donating in period t is given by

$$Pr(d_t = 1|d_{t-1}) = Pr(\tilde{c}_t \leq \gamma d_{t-1} + B) = F_c(\gamma d_{t-1} + B). \tag{2.4}$$

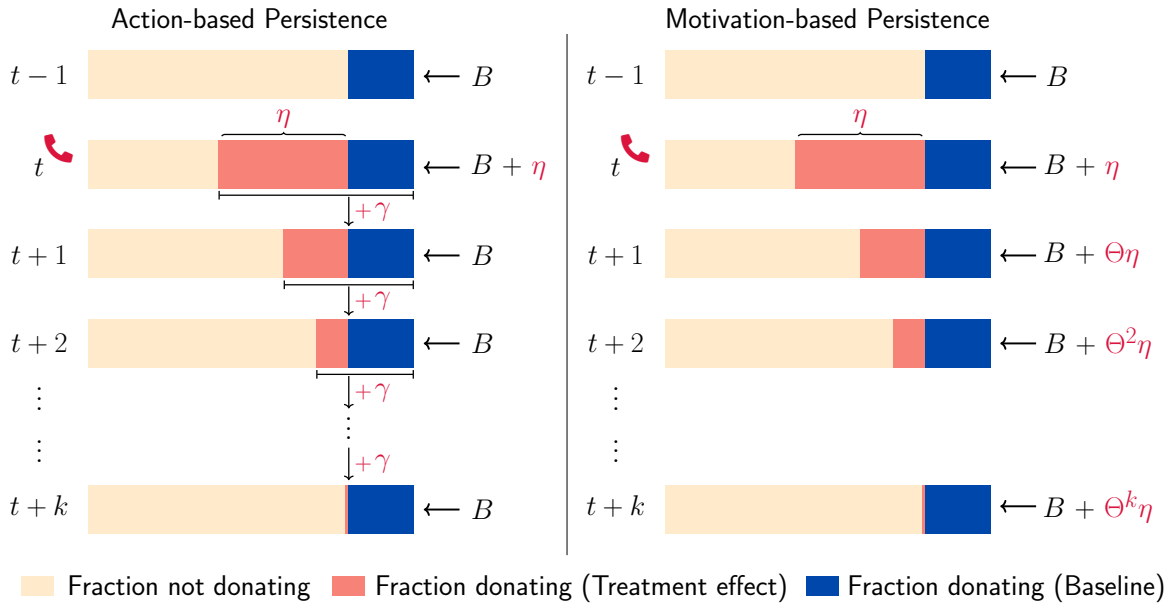
The left part of Figure 1 illustrates the pattern of persistence that the action-based mechanism induces. First, the intervention temporarily increases the donor's motivation from the baseline level B by η in period t, e.g., by making the benefits of donating more salient. This increases the probability to donate, in $t-1$. Subsequently, the donation d_t, or more precisely, the higher fraction of donations in the treatment group, increases the probability to donate in the following period $t+1$ by $F_c(B+\gamma) - F_c(B)$, even though the donor's motivation falls back to the baseline level, B. This continues, as, again, a fraction $F_c(B+\gamma) - F_c(B)$ of those repeat donors also donate in $t+2$, and so on. Thus, with action-based persistence, an intervention impacts the treatment group by jolting the motivation of a shrinking group (those who happen to have donated in each past period) by γ, but eventually trailing off. If $F_c()$ is uniform, then the reduced-form effects are η in the impact period, and $\gamma\eta$ in period $t+1$, $\gamma^2\eta$ in period $t+2$, $\gamma^3\eta$ in period $t+3$, and so on.

2.1.2 Motivation-based Persistence

A policy intervention may also lead to persistence that is not mediated by action. That is, a policy intervention may directly increase a potential donor's motivation over several periods. For instance, highlighting the benefits of the activity and increasing the salience of the social impact of donating may create a persistent shift in the attention to the highlighted benefits (Anderson 2016; Steiny Wellsjo 2021; Byrne et al. 2019).

We specify a parsimonious model of motivation-based persistence as follows: suppose

Figure 1: Illustrations of two types of persistences



Notes: The figure illustrates persistence under action-based and motivation-based model for the special case of uniform costs of donations c_i. In both models, the intervention causes an increase by η in the fraction of donors. In the action-based model, this increases the motivation of those who responded to the impulse in the previous period by γ in the next period. In the motivation-based model, all donors experience persistently higher motivation in subsequent periods, subject to a decay parameter θ.

there is an intervention in period $t-k, k \geq 0$. Utility is given by

$$u(d_t, P_{t-k}, B, \tilde{c}_t) = \begin{cases} \eta\theta^k P_{t-k} + B - \tilde{c}_t & \text{if } d_t = 1 \\ 0 & \text{if } d_t = 0 \end{cases}. \tag{2.5}$$

The parameter η again reflects the first-period impact of the intervention. Its impact decays exponentially over time, governed by the parameter θ as in Byrne et al. (2019).

The probability of donating is given by

$$Pr(d_t = 1|P_{t-k}) = Pr(\tilde{c}_t \leq \eta\theta^k P_{t-k} + B) = F_c(\eta\theta^k P_{t-k} + B). \tag{2.6}$$

The mechanics of motivation-based persistence are highlighted in the right part of Figure 1. In contrast to action-based persistence, the motivation of the entire treatment group is affected over several periods, but the push trails off.

Despite the differences in mechanisms, the two forms of persistence can have identical implications for the reduced form: for instance, if $\theta = \gamma$ and $F_c()$ is uniform, the persistence effect in the reduced form, i.e., comparing individuals who were treated with the intervention to those who were not, will produce the identical pattern of temporal effects $(\eta, \gamma\eta, \gamma^2\eta, \gamma^3\eta, ...)$ as derived above for action-based persistence.

2.2 Dynamic treatment effects under action-based persistence

In this subsection, we derive the general shape of the dynamic treatment effects under action-based persistence. Starting from steady state, donors in the treatment conditions will donate with probability $p_t^T = pF_c(B+\gamma+\eta)+(1-p)F_c(B+\eta)$, while those in the control condition will donate with probability $p_t^C = pF_c(B+\gamma)+(1-p)F_c(B)$.[9] The difference between the two is the treatment effect in the intervention period,

$$\Delta p_t = p(F_c(B+\gamma+\eta) - F_c(B+\gamma)) + (1-p)(F_c(B+\eta) - F_c(B)) \tag{2.8}$$

In period $t+1$, the impact of the intervention in period t continues to affect the behavior of donors in the treatment conditions through action-based persistence from period t. Their probability to donate is $p_{t+1}^T = p_t^T F_c(B+\gamma) + (1-p_t^T)F_c(B)$, whereas the probability to donate of their counterparts in the control condition is $p_{t+1}^C = p_t^C F_c(B+\gamma) + (1-p_t^C)F_c(B)$. The difference between the two corresponds to the treatment effect in period $t+1$,

$$\Delta p_{t+1} = \Delta p_t (F_c(B+\gamma) - F_c(B)) \,. \tag{2.9}$$

Iterating forward, we see a geometric sequence emerging: in period $t+2$, the impact of the intervention from period t continues to affect the behavior of donors in the treatment conditions through past actions. Their probability to donate is $p_{t+2}^T = p_{t+1}^T F_c(B+\gamma) + (1-p_{t+1}^T)F_c(B)$, while the probability to donate of the donors in the control condition is $p_{t+2}^C = p_{t+1}^C F_c(B+\gamma) + (1-p_{t+1}^C)F_c(B)$. Thus, the treatment effect is

$$\Delta p_{t+2} = \Delta p_{t+1}(F_c(B+\gamma) - F_c(B)) = \Delta p_t (F_c(B+\gamma) - F_c(B))^2 \,.$$

[9] Steady-state donation rate is given by $p(F+\gamma)+(1-p)F(B) = p$. This implies

$$p = \frac{F(B)}{1 - F(B+\gamma) + F(B)} \tag{2.7}$$

Generally, the treatment effect in period $t+k$ is

$$\Delta p_{t+k} = \Delta p_t (F_c(B+\gamma) - F_c(B))^k. \tag{2.10}$$

Thus, the intervention's lagged treatment effects in Equation (2.10) follow a geometric sequence proportional to the impact of the contemporaneous effect in Equation (2.8). The sum of (undiscounted) change in donation rates is therefore

$$\Delta P = \frac{1}{1 - F_c(B+\gamma) - F(B)} \Delta p_t \tag{2.11}$$

highlighting again that, if $\gamma > 0$, this may induce a substantial multiplier to the impact during the intervention period. If $F_c()$ is uniform, the expression simplifies to $\Delta P = \Delta p_t/(1-\gamma)$.

2.3 Welfare under action-based persistence

In this subsection, consider a one-time policy intervention that increases the motivation of donors by η. The costs of rolling out the intervention to a fraction q are denoted by $c(q)$. On top of generating utility for individuals according to Equation (2.2), donations also have a positive external effect of ϕ per donation, which the policy maker also takes into account. We follow Bernheim and Taubinsky (2019) and define welfare in the steady state with $q = 0$ is given by

$$W(0) = pF_c(B+\gamma)(E_{B+\gamma}(u) + \phi) + (1-p)F_c(B)(E_B(u) + \phi)$$

where $E_x(u) = E(u|c \leq x)$ denotes the expected utility of donation in Equation (2.2), conditional on $c \leq x$. We consider the change from baseline welfare as a function of q

$$\Delta W = W(q) - W(0) = q(W^T - W^C) - c(q)$$

where W^T and W^C are the welfare of the group treated with the intervention and the untreated group, respectively. As we show in appendix A, the undiscounted sum of the period welfare difference from the one-time intervention can be written as

$$\Delta W = q\big(\Delta W_0 + \frac{\Delta p_0}{1 - F_c(B+\gamma) - F(B)} \Delta w_1\big) - c(q) \tag{2.12}$$

where Δp_0 is the first-period impact on donation propensities from Equation (2.9). The expression ΔW_0 is the treatment effect on externalities and consumer surplus in the impact

period, stemming from higher donation probabilities and also an increase in consumer surplus due to the increase in the utility of donation of η (see Equation (A.7) in the appendix).

The expression Δw_1 is the externality ϕ plus difference in consumer surplus between donors who donated in the previous period and those who did not. Prior donation increases, both, the probability of donation and the consumer surplus for infra-marginal donors (see Equation (A.8) in the appendix). The policy maker takes the externality as well as the differences in consumer surplus into account. This differences scales with the persistence effect Δp_t of each period. Hence, summing up over periods, the same multiplier applies $\Delta p_0/(1 - F_c(B + \gamma) + F_c(B))$ applies to the term as in Equation (2.11).

If the policy maker were to disregard consumer surplus and only target the externality, then the gain in the policy maker's objective function becomes

$$\Delta V = q \cdot \phi \cdot \frac{\Delta p_0}{1 - F_c(B + \gamma) + F_c(B)} - c(q)$$

where, again, the same persistence multiplier appears (see Equation (A.1) in the appendix).Thus, policy makers may focus on the persistence multiplier in donor turnout when designing interventions.

3 Experimental Setup and Data

This section discusses the experimental setup and the data. We first outline the invitation procedure at the BTSRC, where our study took place. We then describe the field experiment which involves asking potential donors via a phone call to donate at the upcoming blood drive. We also perform randomization checks to ensure that the randomization led to balanced subgroups. Finally, we discuss the natural experiment, which relies on random fluctuations in daily weather conditions and provides our second instrument.

3.1 Invitation Procedure at the BTSRC

To ensure a stable supply of whole blood transfusions, the BTSRC follows a multi-stage invitation procedure for its blood drives. Blood drives are regular events where donations can be made. They take places twice per year and are sponsored by local organizations, such as church chapters or sports clubs, while the BTSRC invites potential donors and provides the required equipment and personnel. In total 96 drives are subject to the intervention in our study. They occur on 84 different work days during the sample period: 31% on Monday,

18% Tuesday, 40% on Wednesday, and 11% on Thursday.[10] For each blood drive, the BTSRC first sends an invitation letter to all potential donors, informing them about the event and highlighting the general benefits of blood donations for society. One day before the blood drive, it also sends a text message to all invited donors, reminding them about the time and the location of the event.

3.2 Targeted Subjects

Our study focuses on 1,400 registered donors who did not show up at any blood drive they were invited to within one year before the onset of the study, and whose blood types are O+, O-, or A-.[11] That is, they have been inactive in the donation record for at least 12 months.

Inactive donors are of particular interest to the BTSRC for two reasons. First, they make up the majority of the pool, accounting for 60% of all registered donors at any given time. Meanwhile, more than 80% of the registered donors are inactive at least for some period. Second, the maximum permitted frequency of donating blood at most every three months does not constrain inactive donors at the beginning of the study.

The majority of this sample would be donors with low baseline motivation who possibly have not donated in the past for several years. However, it is also likely that this sample contains some donors who have high baseline motivation but, for certain reasons, could not donate in the previous two consecutive blood drives.

Compared to a sample of all registered donors in another study by Bruhin et al. (2020), the inactive donors in our study are on average 2 years younger and there are 5 percentage points fewer males. The blood types in the current study are not comparable to the other study as the two interventions target different sets of donors with particular blood types.

3.3 Field experiment

We now turn to the field experiment that allows us to examine potential behavioral persistence in blood donations.

3.3.1 Intervention

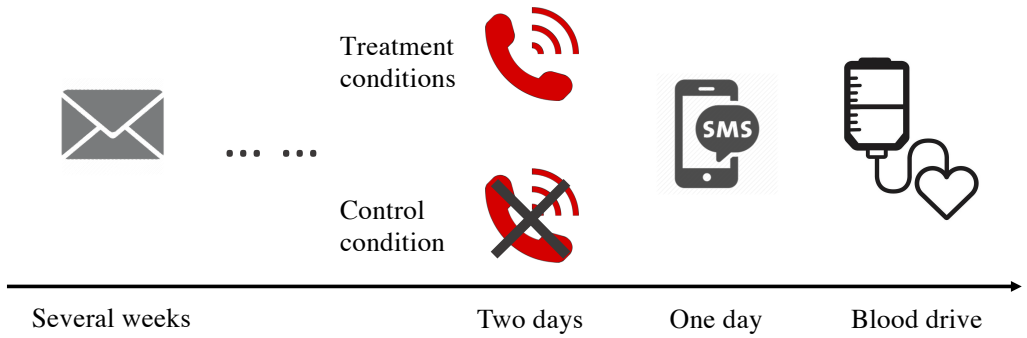
The randomized intervention is delivered by a phone call: the BTSRC calls invited potential donors two days before the upcoming blood drive they have been invited to and asks them

[10] One can find the map of the locations of the blood drives on `https://www.blutspendezurich.ch/spender/wo-kann-man-blut-spenden`.

[11] Other blood types are less useful to the BTSRC due to compatibility patterns. Thus, they were not targeted by the intervention.

to make a donation. The phone call makes a strong ask: *"Can I put you down as attending our blood drive in two days?"* Figure 2 illustrates the timing of the intervention along with the standard invitation procedure.

Figure 2: Timing of the intervention (time before blood drive)



Notes: The intervention asks invited donors via a phone call to make a donation. It takes place two days before the upcoming blood drive, between the invitation letter and the text message reminder.

3.3.2 Implementation

We implemented the field experiment between March 2015 and December 2016. During this period, each local sponsor organized four blood drives, dividing our experiment into four periods of six months each. We randomized the intervention within blood drive strata and defined four experimental conditions. Each condition comprises 350 of the 1,400 inactive registered donors.

Table 1 summarizes the schedule of the intervention across the four experimental conditions. In the first condition, C1, potential donors receive a phone call only in period 1. In the second condition, C2, potential donors receive a phone call only in period 2, i.e., six months after the calls in condition C1. In the third condition, C12, potential donors receive a phone call in both periods 1 and 2, which allows us to test whether there is any interaction effect between two phone calls. In the control condition, potential donors do not receive any phone calls. There are no further phone calls in periods 3 and 4, i.e., 12 and 18 months later. However, we keep observing donation outcomes in periods 3 and 4.

Table 1: Intervention across experimental conditions

	Period 1 (t_1)	Period 2 (+6 months)	Period 3 (+12 months)	Period 4 (+18 months)	Number of individuals
Condition C1	Call	No Call	No Call	No Call	350
Condition C2	No Call	Call	No Call	No Call	350
Condition C12	Call	Call	No Call	No Call	350
Control Condition	No Call	No Call	No Call	No Call	350

Note: All potential donors receive a postal invitation to the blood drive they are registered in each period. One day before the blood drive, all invited donors receive an additional text message reminder (Standards invitation procedure at BTSRC).

3.3.3 Randomization Checks

Table 2 presents the randomization checks of the intervention. Columns (1)-(4) show the means and standard deviations of the potential donors' age, gender, and blood types across the four experimental conditions. Column (5) reports the p-values of the joint F-tests for equality in means across the four conditions. As none of the F-tests indicates a significant difference in means, we conclude that the randomization of the intervention succeeded.

Table 2: Randomization checks

	(1) Condition C1	(2) Condition C2	(3) Condition C12	(4) Control	(5) F-test (p-val.)
Age	41.140	40.226	42.051	41.126	0.36
	(13.171)	(13.373)	(13.984)	(13.544)	
Male	0.506	0.483	0.511	0.546	0.42
	(0.501)	(0.500)	(0.501)	(0.499)	
O+ blood type	0.826	0.817	0.820	0.829	0.98
	(0.380)	(0.387)	(0.385)	(0.377)	
O- blood type	0.054	0.083	0.057	0.046	0.19
	(0.227)	(0.276)	(0.232)	(0.209)	
A- blood type	0.120	0.100	0.123	0.126	0.71
	(0.325)	(0.300)	(0.329)	(0.332)	
Observations	350	350	350	350	1,400

Notes: Means with standard deviations in parentheses.

3.4 Natural Experiment

Next, we turn to the natural experiment to discriminate between action-based persistence and motivation-based persistence in our setup. The natural experiment relies on random fluctuations in daily weather conditions in the greater Zurich region. It allows us to construct a second instrument that directly affects donations in the present but does not directly affect the motivation to donate in the future. The second instrument is needed for our identification strategy, which we explain in detail in Section 5.1.2.

Table 3: Adverse Weather Conditions and Donation Rate

Weather conditions	Rain		Cold		Heat		Any of the three	
Variable	No	Yes	No	Yes	No	Yes	No	Yes
Donation rate	0.182	0.141	0.181	0.148	0.184	0.131	0.190	0.138
	(0.386)	(0.348)	(0.385)	(0.356)	(0.388)	(0.385)	(0.392)	(0.345)
Observations	3,930	270	4,017	183	3,796	404	3,343	857
No. of blood drives	67	5	69	3	65	7	57	15
No. of days	59	4	60	3	58	5	51	12

Notes: Means with standard deviations in parentheses. Rain: daily rainfall $\geq$10mm; Cold: daily temperature high $<$ 5 degrees; Heat: daily temperature high $>$ 25 degrees. Notice this is the definition of a summer day in Switzerland.

The three weather conditions are heavy rainfall exceeding 10mm (Rain), temperature below 5 degrees (Cold), and temperature above 25 degrees (Heat). The rational behind the choice of conditions is that, if there is heavy rainfall or if it is particularly cold, the discomfort of the commute to the blood drive is higher; if it is hot, the opportunity cost of donating blood or the discomfort due to the heat-induced stress on the cardiovascular system are higher.[12]

Table 3 shows the donation rates under three adverse weather conditions on the day of the blood drives provided by the Swiss Federal Office of Meteorology and Climatology. Each of the adverse weather conditions has a negative effect on the donation rate. The donation rate drops by about 4 percentage points on day with heavy rainfall, 3 percentage points on a cold day, and 5 percentage points on a hot day. The same holds true when we compare days with and without any of the adverse weather conditions. We will use the indicator of any adverse weather condition to construct the second instrument.

[12]We thank an anonymous referee for proposing additional weather conditions as alternative instruments.

4 Behavioral Persistence

This section identifies behavioral persistence in blood donations using the randomized intervention of asking potential donors via a phone call to donate at the upcoming blood drive. We first show descriptive evidence. Subsequently, we introduce the econometric analysis and discuss the results.

4.1 Descriptive Evidence

Figure 3 exhibits descriptive evidence for behavioral persistence in blood donations.

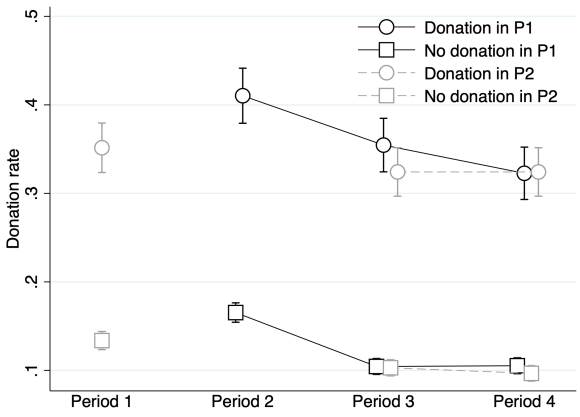
Panel (a) provides a first look at correlations between past and current donations. Lines with circles represent donation propensities conditional on having donated at the last invitation, while squares show donation frequences conditional on not having donated at the last invitation. For instance, the black lines show that there is a persistent differences between individuals who have donated in period 1 (the first round of invitations) and those who did not: there is a difference of roughly 25 percentage points in donations between the former and the latter group that persists over time. Similarly, the grey lines show a similar difference in periods 3 and 4 between donors who did and did not donate in period 2. This evidence is suggestive of action-based persistence, with a γ of about 0.25. However, the comparison selects on the dependent variable and can be biased due to individual-specific differences in donation propensities. Indeed, Panel (a) shows evidence of such bias: splitting the sample by whether an individual donated in period 2, we find nearly the same difference in donation rates in period 1, pre-dating the act of donation in period 2. This casts doubt on whether differences conditional on past behavior can be interpreted in a causal manner. As we further show in simulations in Figure F.4 in the appendix, differences in conditional donation rates can be severely biased even if there is strong action-based persistence.

Panel (b) shows how donation rates in the different experimental conditions evolved over the four periods of the field experiment. It allows us to get a first impression of the extent of persistence identified by the experimental variation.[13]

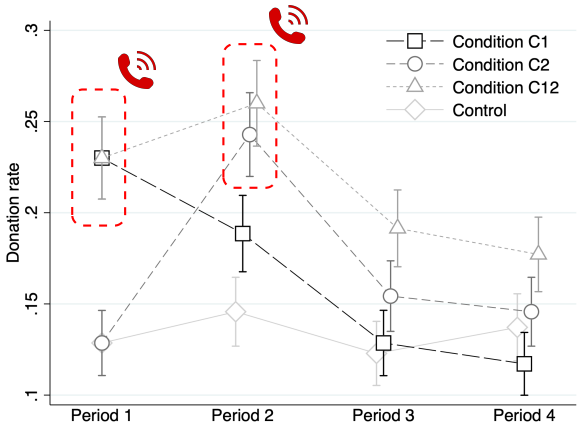
[13]The descriptive evidence takes an intention to treat (ITT) perspective, that is, whether the phone call is attempted by the BTSRC instead of whether it is actually answered by the potential donors.

Figure 3: Descriptive evidence for the persistent effects of the intervention

(a) Donation rates by past donation



(b) Donation rates by experimental conditions



Notes: Donation rate as a function of the intervention over time. Red dashed frames indicate that the members of the corresponding condition receive the intervention in that period. Scatters show the means per condition, along with standard error bars.

Based on three observations we conclude that there is behavioral persistence in blood donations. Consider first donations in group C1: in period 1, potential donors in condition C1 receive a phone call asking them to donate at the upcoming blood drive. Their donation rate is 23% in period 1, while their counterparts in the control condition and in condition C2 who are not called exhibit a donation rate of roughly 13%. Hence, asking for a donation directly increases the donation rate by 10 percentage points. In period 2, the donation rate in condition C1 declines to about 18% but remains well above the one in the control condition. In fact, about half of the initial effect is still visible six months later – pointing towards behavioral persistence in blood donations. Only in period 3, the donation rate in condition C1 falls back to the level in the control condition.

In condition C2, potential donors receive a phone call in period 2. Their donation rate immediately jumps by about 10 percentage points and, then, gradually declines over the following two periods.

In condition C12, individuals receive two phone calls, one in period 1 and another one in period 2. Again, we find evidence for behavioral persistence. Following two periods of phone calls, the group's donation rate remains substantially above the control group's donation rate in periods 3 and 4. Overall, the descriptive evidence in Panel (b) indicates that there is behavioral persistence in blood donations. If calibrated to an action-based model, the evidence suggests a persistence parameter of $\gamma \approx 0.5$, highlighting again the important differences in conclusions between Panels (a) and (b).

4.2 Econometric Analysis

We now turn to the econometric analysis which takes the panel structure of the data into account and features additional control variables. We first take an ITT perspective as in the descriptive analysis. Subsequently, we focus on potential donors who answered the phone call and estimate the local average treatment effect (LATE) of asking them to donate at the upcoming blood drive.

4.2.1 Setup

To obtain the ITT estimates, we estimate the following reduced-form specification:

$$\text{Donation}_{ib,t} = \beta_1 \text{Call}_{i,t} + \beta_2 \text{Call}_{i,t-1} + \beta_3 \text{Call}_{i,t-2} + \beta_4 \text{Call}_{i,t-3} + \phi' X_i + \delta_b + \epsilon_{ib,t}. \quad (4.1)$$

The binary outcome, $\text{Donation}_{ib,t}$, indicates whether potential donor i makes a donation at the upcoming blood drive b in the current period t. We include the indicator whether the donor receives a phone call, $\text{Call}_{i,t}$, in its contemporaneous form as well as with three lags. The vector X_i controls for individual characteristics gender, age, and blood type, while δ_b represents blood-drive-specific fixed effects. We control for individual characteristics to increase the precision of our estimates even though they are balanced as a result of randomization (Athey and Imbens 2017). In an alternative specification, we replace the individual characteristics with individual-specific fixed effects.

Furthermore, to explore the role of multiple repeated phone calls, we estimate a version of the specification that features interactions between different lags of the phone call:

$$\text{Donation}_{ib,t} = \sum_{k=0}^{3} \beta_{k+1} \text{Call}_{i,t-k} + \sum_{k=0}^{2} \alpha_{k+1} \text{Call}_{i,t-k} \times \text{Call}_{i,t-k-1} + \delta_b + \nu_{ib,t}. \quad (4.2)$$

Finally, we estimate the LATE of asking potential donors to donate at the upcoming blood drive. That is, we estimate the analogue of Equation (4.1) by two-stage-least-squares (2SLS), using attempted phone calls as the instrument for answered phone calls. The first-stage-equations have the following form:

$$\text{Ask}_{ib,t-l} = \sum_{k=0}^{3} \iota_{k+1} \text{Call}_{i,t-l-k} + \phi' X_i + \delta_b + \epsilon_{ib,t-l}\,, \quad (4.3)$$

where $\text{Ask}_{ib,t-l}$ indicates whether potential donor i answered the phone call asking her to donate at the upcoming blood drive b in period $t-l$. We need to estimate a separate first-stage-equation for the current period and each of the three lags, $l \in \{0, 1, 2, 3\}$. Based on

the first-stage-estimates, we can predict whether the potential donor answers the phone and estimate the following second-stage-equation:

$$\text{Donation}_{ib,t} = \sum_{k=0}^{3} \omega_{k+1} \widehat{\text{Ask}_{ib,t-k}} + \phi' X_i + \delta_b + \epsilon_{ib,t}, \tag{4.4}$$

where $\widehat{\text{Ask}_{ib,t-k}}$ denotes the predicted values. In an alternative specification, we replace the individual characteristics, X_i, with individual-specific fixed effects. Finally, we obtain the LATE estimates for Equation (4.2) with interactions in an analogous manner.

Notice that whether we estimate the ITT or the LATE only changes the interpretation of the effect of the intervention on the donation rate. It leaves the estimated extent of the behavioral persistence unchanged. This is because the estimate for the extent of behavioral persistence corresponds to the ratio of the lagged and the current intervention. Thus, defining the intervention as whether a phone call is attempted or whether a phone call is actually answered does not change this ratio.

4.2.2 Results

Table 4 reports the results. Columns (1)-(3) show the ITT estimates for the effects of the phone call on the donation rate, and Columns (4)-(6) contain the analogous LATE estimates of asking potential donors to donate.[14]

Column (1) shows the estimated coefficients of the specification in Equation (4.1) with individual characteristics and blood-drive-specific fixed effects. Column (2) shows the coefficients of the alternative specification that replaces the individual characteristics with individual-specific fixed effects. Column (3) displays the coefficients of the specification in Equation (4.2) with interactions between the different lags of the phone call. We find evidence for behavioral persistence in blood donations in all three specifications. The phone call not only directly increases the donation rate by 9 to 14 percentage points, but also has significant lagged effects: a phone call one period or six months ago increases the current donation rate by 4 to 9 percentage points, while a phone call two periods or twelve months ago still leads to an increase by 3 to 6 percentage points. Only after three periods, the lagged effects become insignificant.

There is no evidence for interactions between consecutive phone calls. The coefficients of the interactions in Column (3) are all insignificant both individually and jointly (p=0.35).

[14]Table B.1 in the Appendix reports the first stages for asking, showing that only roughly half of the phone calls (53% in Column (1)) are answered. Consequently, the LATE estimates are roughly twice the magnitude of those based on the ITT perspective.

Thus, the effect of a phone call does not depend on the phone call in the previous period.[15]

The LATE estimates in Columns (4)-(6) reveal that asking potential donors to make a donation at the upcoming blood drive directly increases the donation rate in the current period by 18 to 26 percentage points, depending on the specification. There are also substantial lagged effects: Asking potential donors one period or six months ago increases the current donation rate by 8 to 17 percentage points, while asking them two periods or twelve months ago leads to an increase by 5 to 13 percentage points. In line with the estimates based on the ITT perspective, the lagged effects after eighteen months and the interactions between the different lags are insignificant. Taken together, we find behavioral persistence in blood donations that prolongs the initial effect of an intervention on the donation rate for at least one year.

[15]We also looked at the potential interaction between the phone call and adverse weather conditions. However, we do not observe any significant interaction effects. Results are available upon request.

Table 4: Reduced-Form Results

Dependent variable		ITT		Dependent variable		LATE	
Donation_t	(1)	(2)	(3)	Donation_t	(4)	(5)	(6)
Call_t	0.0925***	0.125***	0.139***	Ask_t	0.175***	0.246***	0.263***
	(0.0159)	(0.0230)	(0.0248)		(0.0295)	(0.0451)	(0.0489)
Call_{t-1}	0.0385***	0.0710***	0.0896***	Ask_{t-1}	0.0767***	0.149***	0.171***
	(0.0139)	(0.0226)	(0.0277)		(0.0268)	(0.0447)	(0.0552)
Call_{t-2}	0.0275**	0.0600***	0.0634**	Ask_{t-2}	0.0535**	0.126***	0.125**
	(0.0129)	(0.0213)	(0.0283)		(0.0244)	(0.0426)	(0.0565)
Call_{t-3}	0.00373	0.0402	0.0376	Ask_{t-3}	0.00861	0.0942	0.0843
	(0.0205)	(0.0313)	(0.0429)		(0.0361)	(0.0595)	(0.0805)
$\text{Call}_t \times \text{Call}_{t-1}$			-0.0571	$\text{Ask}_t \times \text{Ask}_{t-1}$			-0.126
			(0.0368)				(0.122)
$\text{Call}_{t-1} \times \text{Call}_{t-2}$			-0.0184	$\text{Ask}_{t-1} \times \text{Ask}_{t-2}$			-0.0219
			(0.0374)				(0.122)
$\text{Call}_{t-2} \times \text{Call}_{t-3}$			0.00381	$\text{Ask}_{t-2} \times \text{Ask}_{t-3}$			0.0299
			(0.0420)				(0.134)
Control mean	0.13	0.13	0.13	Control mean	0.13	0.13	0.13
Joint F-tests:				Joint F-tests:			
All lagged Calls=0	0.0182	0.00890	0.00466	All lagged Asks=0	0.0165	0.00576	0.00807
All interactions=0			0.349	All interactions=0			0.603
				1^{st} stage instruments	155.7	123.8	28.13
Individual controls	Y			Individual controls	Y		
Blood drive FE	Y	Y	Y	Blood drive FE	Y	Y	Y
Individual FE		Y	Y	Individual FE		Y	Y
Observations	5,600	5,600	5,600	Observations	5,600	5,600	5,600

Notes: In Column (1) the coefficients on the individual characteristics gender, age, and blood type are not shown. Standard errors clustered at the individual and blood drive level in parentheses. ** $p < 0.05$, *** $p < 0.01$. The LATE coefficients are obtained from 2SLS regressions using attempted phone calls as instruments for asking for donations.

5 Mechanisms Behind Behavioral Persistence

In this section, we discriminate between action-based persistence and motivation-based persistence in blood donations. We first present the identification strategy and its implementation. Subsequently, we discuss the results. Finally, we replicate the results in a larger quasi-experimental sample with a similar intervention. Since the discussion in this section relates to the validity of our instruments, it takes the ITT perspective.

5.1 Identification Strategy

5.1.1 Identification Challenge

It is impossible to discriminate between the two mechanisms by relying exclusively on the intervention delivered by the phone call. On the one hand, the intervention may increase future donations through the action of making a donation in the present. However, on the other hand, it may also change the potential donors' future motivation over several periods. Even though the phone calls in our setup do not explicitly stress the social benefits of donating, we cannot rule out that receiving them may have a persistent impact on donor motivation. More formally, reconsider the probability to donate at the upcoming blood drive, as shown in Equation (2.4) of our theoretical framework:

$$Pr(d_t = 1) = Pr(\tilde{c}_t \leq \gamma d_{t-1} + B) = F_c(\gamma d_{t-1} + B)\,.$$

Suppose that we aim to identify the parameter γ that governs the extent of action-based persistence by using the phone call in period $t-1$ as an instrument for the donation in that period, d_{t-1}. Any persistent change the intervention may induce in the motivation B would be in the error term and, thus, would cause a violation of the exclusion restriction.

5.1.2 Second Instrument and Overidentification Test

To detect such a potential violation of the exclusion restriction, we require a second instrument for d_{t-1} which is both strong and valid. That is, the second instrument needs to affect the donors' probability to donate in the current period but leave their future motivation unchanged. Moreover, it should be at most only partially correlated with the phone call.

We use the indicator for adverse weather conditions as our second instrument, as it satisfies all aforementioned conditions. First, as we already showed in Section 3.4, adverse weather conditions have a negative effect on the donation rate, as they cause a temporary shock in the costs or opportunity costs of donating $\tilde{c}_t$. Second, the indicator for adverse weather

conditions is a valid instrument, as such a temporary shock in the costs of donating does not affect the donors' motivation six months later in the future. Finally, the indicator is orthogonal to the phone call, as the phone call is balanced within blood drives and, hence, daily weather conditions.[16]

We rely on this second instrument to conduct an overidentification test – that is, a *Sargan-Hansen J-test of Overidentifying Restrictions* (Sargan 1958; Hansen 1982) – and to verify whether the phone call satisfies the exclusion restriction. The test has the following intuition. If both instruments, the phone call and the indicator for adverse weather conditions, satisfy the exclusion restriction, namely, only affect present donations but not future motivation, the corresponding 2SLS-regression is valid and the residuals of the second stage are exogenous. However, if the phone call violates the exclusion restriction, e.g., if it also affects the donors' future motivation, then the 2SLS-regression would be invalid and the residuals of the second stage would be correlated with at least one of the two instruments.

The null hypothesis of the overidentification test is that all instruments are exogenous to these residuals (Stock and Watson 2015). It constructs the residuals using the coefficients estimated from the second stage and regresses them on both instruments to test whether the null of a joint zero effect can be rejected. If the null hypothesis is not rejected, we can conclude that the intervention of asking potential donors to make a donation at the upcoming blood drive has no direct effect on their future motivation and, therefore, the behavioral persistence is driven by engaging in the action of donating.

To implement the overidentification test, we first estimate the following instrumental variables regression with 2SLS. The first stage has the following form:

$$\text{Donation}_{is,t-1} = \gamma_1 \text{Call}_{is,t-1} + \gamma_2 \text{Adverse Weather}_{s,t-1} + \gamma_3 \text{Call}_{is,t} + \gamma_4 \text{Adverse Weather}_{s,t} + \gamma_5 X_i + \delta_w + \xi_{is,t-1}. \quad (5.1)$$

The dependent variable, $\text{Donation}_{is,t-1}$, is the indicator whether potential donor i donated at the blood drive of sponsor s in period $t-1$. The independent variables comprise the two instruments, i.e., the indicators for the phone call, $\text{Call}_{is,t-1}$, and for adverse weather conditions on the day of the blood drive, $\text{Adverse Weather}_{is,t-1}$. We also include the future values of the instruments, $\text{Call}_{is,t}$ and $\text{Adverse Weather}_{is,t}$, since they will appear in the second stage. Moreover, we also control for the individual characteristics, X_i, and fixed effects for the week of the year, δ_w.

[16] See balance checks in Appendix Table C.1.

The second stage has the following form:

$$\text{Donation}_{is,t} = \mu_1 \widehat{\text{Donation}_{is,t-1}} + \mu_2 \text{Call}_{is,t} + \mu_3 \text{Adverse Weather}_{s,t} + \mu_4 X_i + \mu_w + \zeta_{is,t} \tag{5.2}$$

It regresses donations in period t on the predicted values $\widehat{\text{Donation}_{is,t-1}}$ from the first stage; the future values of the instruments, $\text{Call}_{is,t}$ and $\text{Adverse Weather}_{is,t}$; the individual characteristics; and the fixed effects for the week of the year.

After estimating the second stage, we regress its residuals, $\hat{\zeta}^{2sls}_{is,t}$, on the two instruments and all other exogenous variables:[17]

$$\hat{\zeta}^{2sls}_{is,t} = \lambda_1 \text{Call}_{is,t-1} + \lambda_2 \text{Adverse Weather}_{s,t-1} + \lambda_3 \text{Call}_{is,t} + \lambda_4 \text{Adverse Weather}_{s,t} + \lambda_5 X_i + \lambda_w + \epsilon_{is,t}. \tag{5.3}$$

Under the null hypothesis that both instruments are exogenous the test statistic of the joint F-test $\lambda_1 = \lambda_2 = 0$ is χ^2_1 distributed.

5.2 Results

Table 5 displays the results. Columns (1) and (2) exhibit the first- and second-stage-estimates as well as the p-value of the overidentification test.[18] In the first stage, both excluded instruments are strong, and the joint Kleibergen/Paap F-statistic on both instruments is 35.16 – well above the conventional thresholds for strong instruments. In the second stage, the estimated coefficient on the past donation is 0.4. Importantly, the p-value of the overidentification test is 0.942. Thus, we do not reject the null hypothesis that both instruments are valid.

The conclusion from the overidentification test can also be illustrated by varying the instruments we use. In Columns (3) and (4), we show the second stage estimates when we include only one instrument at a time in the first stage – the indicator for the phone call in Column (3) and the indicator for adverse weather conditions in Column (4). If the phone call violated the exclusion restriction, the second stage coefficient on the effect of lagged donations in Column (3) would exhibit endogeneity bias and differ from the coefficient in Column (4) which is based on the indicator for adverse weather conditions. As we can

[17] Residuals are based on coefficient estimates from the second stage, but the true regressors rather than predicted values from the first stage (see Stock and Watson 2015).

[18] The p-value of the overidentification test reported in Table 5 relies on a cluster-robust version (see Hayashi 2000, pp. 227f).

see, the coefficients on the effect of lagged donations are remarkably similar in Columns (3) and (4). Thus, we find no evidence for endogeneity bias and confirm the result of the overidentification test that both instruments are valid.

Finally, we look at the test from yet another angle and perform the following additional check. If both instruments satisfy the exclusion restriction and only affect the future donation rate through the contemporaneous donation, the instruments should not correlate with future donations once we control for that channel. Columns (5)-(6) show the results of the second stage when we include only one instrument at a time in the first stage but *include the other instrument as a control variable* in the second stage. In line with the result of the overidentification test, neither the indicator for the lagged phone call nor the indicator for the lagged adverse weather conditions have a direct effect on current donations once we control for predicted past donations.

Taken together, these results indicate that the phone call has no direct effect on the donors' future motivation and that the behavioral persistence in blood donations is action-based.[19]

5.3 Replication Study

As a robustness check, we conduct a replication study in a larger quasi-experimental sample featuring a similar intervention. The quasi-experiment took place in the greater Zurich region between 2012 and 2014. The intervention is also delivered by a phone call. However, the phone call conveys a shortage message and is randomized conditional on blood types. That is, depending on the daily inventory in its blood stock, the BTSRC determines which blood types are in short supply and calls a random subset of invited donors with the required blood types. In the phone call, the BTSRC's staff tells potential donors that their blood type is in short supply and encourages them to donate at the upcoming blood drive. The message of the phone call differs from the one in field experiment in the sense that it points out the temporary shortage in the donors' blood types and does not ask them for a commitment.[20] Moreover, in this quasi-experiment, only 14% of the invited donors ever receive a phone call and most of them get called only once.[21]

Even though the quasi-experiment has less power to identify the action-based persistence, we find qualitatively identical results and, again, do not reject the null hypothesis that both instruments are valid (p=0.26). Appendix D presents the results in detail.

[19] We get qualitatively identical results if we use each of the three components of adverse weather conditions separately as the second instrument. Results are available on request.

[20] More details of the quasi-experiment setup can be found in Bruhin et al. (2020).

[21] We make the sample as comparable as possible to our field experiment by focusing on inactive donors who have not donated in the past year and have the same blood types O-, O+, and A-, as well as by limiting the data set to sponsors with blood drives scheduled regularly every 6 months.

Table 5: Action-based vs motivation-based persistence

	(1)	(2)	(3)	(4)	(5)	(6)
	1^{st} stage	2^{nd} stages				
Dependent variable:	Donation_{t-1}	Donation_t	Donation_t	Donation_t	Donation_t	Donation_t
$\widehat{\text{Donation}_{t-1}}$		0.395***	0.397***	0.380	0.398***	0.380
		(0.120)	(0.133)	(0.211)	(0.133)	(0.215)
Call_{t-1}	0.0949***					0.00166
	(0.0144)					(0.0230)
Adv. Weather_{t-1}	-0.0724***				0.00126	
	(0.0129)				(0.0178)	
Call_t	-0.0242	0.0935***	0.0935***	0.0935***	0.0935***	0.0931***
	(0.0162)	(0.0173)	(0.0174)	(0.0172)	(0.0173)	(0.0194)
Adv. Weather_t	-0.00702	-0.0128	-0.0127	-0.0132	-0.0129	-0.0130
	(0.0103)	(0.0143)	(0.0140)	(0.0170)	(0.0150)	(0.0158)
Instrument in the 1^{st} stage		Both	Call	Adv. Weather	Call	Adv. Weather
Kleibergen/Paap F-statistic		35.16	43.31	29.77	43.16	31.30
Sargan-Hansen J-test (p-val.)		0.942				
Observations	4,200	4,200	4,200	4,200	4,200	4,200

Notes: Regressions additionally include individual controls (gender, age, blood type) and week of the year fixed effects. Standard errors clustered at the individual and blood drive level in parentheses. ** $p < 0.05$, *** $p < 0.01$

5.4 Statistical power of the overidentification test

While the previous results indicate that we cannot reject the null of hypothesis of action-based persistence, it is unclear how sensitive the test statistic is to the presence of motivation-based persistence, which would lead to violations of the exclusion restriction. In general, it is also not clear that a stronger violation of the exclusion restriction always leads to higher statistical power.

In order to assess the power in our application, we assume the observational worst case scenario, in which action-based and motivation-based persistence lead to the exact same reduced-form persistence, i.e., $\gamma = \theta$ in terms of Equations (2.2) and (2.5), and choose the random cost to be uniformly distributed on $[0, 1]$. We then generate violations in the exclusion restriction by gradually shifting the impact of the phone call from action-based persistence to motivation-based persistence, with shares α and $1-\alpha$ respectively. This leaves the overall amount of reduced-form persistence approximately constant, irrespective of the

shares of α and $1-\alpha$.[22],[23]

We simulate a data set of the same dimensions as our experimental data, with the same treatment configurations and frequency of adverse weather. We use the parameter estimates of γ from Section 6 as our baseline for action-based persistence and rely on the first-stage coefficients from Column (1) in Table 5 to generate the immediate impacts of phone calls (η) and adverse weather on donations. These choices guarantee that we generate simulation results that mimic the statistical properties of our data as closely as possible.

For a given share of action-based persitence α, we simulate the above-described model and generate 10,000 replications. In each replication, we then perform the overidentification test. We use these simulated distributions of test statistics to obtain a simulation estimate of test statistic under the alternative hypothesis of a share of $1-\alpha$ of the persistence stemming from motivation-based persistence. Appendix F contains more details on the simulation procedure.

Hence, $\alpha = 1$ corresponds to the null hypothesis, and deviations from it correspond to various extents of violations of the exclusion restriction. Reducing α towards zero shifts an increasing proportion $1-\alpha$ of the reduced-form persistence from the action-based channel into the motivation-based channel, generating an increasingly strong violation of the exclusion restriction. The case $\alpha = 0$ reflects the case where persistence is entirely motivation-based. We also examine the statistical power of the test for values of α outside of $[0, 1]$. While this still leaves the overall amount of persistence roughly constant, negative values of α now create action-based substitution over time, counter-balanced by an increasingly strong motivation-based persistence. Conversely, for $1 < \alpha < 2$, we generate motivation-based substitution over time, while increasing the impact of action-based persistence to balance the reduced-form strength of the overall effect.

To assess the power of our test, we ask with what frequency we would find a test statistic less favorable to the null hypothesis than the one we found in our estimate of $p = 0.942$. Our simulations show that for any value of α, the probability of obtaining a test statistic

[22] This is an extremely conservative approach. If we started from a baseline level of action-based persistence and gradually added motivation-based persistence, this would increase the overall strength of persistence in the data, thus making the instruments stronger, and violations easier to detect.

[23] In the context of this evaluation, it is also useful to highlight what the effective sample size is of our study: under action-based persistence, the effective sample size is the additional individuals who were motivated to donate because of the intervention, i.e., about 10% of the treated group. This group then experiences a strong push γ to also donate in the next period. By contrast, under motivation-based persistence, the effective sample size is the entire group of treated donors, who may experience a - likely much smaller - push to donate in the subsequent period. Our test is based at the ITT level, and uses changes in the fraction to donate as a function of the treatment. Thus, intuitively, for a given reduced-form effect, it is irrelevant to the test whether this came about because nearly all of the individuals of a small subpopulation changed their mind, or because the intervention gives a small push that changes the behavior of a smaller fraction of individuals in a larger group. As Table 4 shows, the reduced-form is strong and precisely identified.

that is worse than the one we find is nearly 1 (see Panel (a) in Figure F.1 in Appendix F). Thus, obtaining the realization of the test statistic in Table 5 is nearly impossible in the presence of even a minimal share of motivation-based persistence, which vindicates our interpretation.[24] We obtain a similar result for the replication study detailed in Appendix D, where the p-value of the overidentification test was 0.26. The probability of obtaining a test statistic less favorable to the null than what we find is 90% if α is about 0.6 in that sample. Thus, the statistical power in the replication sample is slightly weaker than in the main study.[25]

Taken together, our results indicate that the probability of a type-2 error (accepting the null of action-based persistence even though motivation-based persistence is present) is small: it is nearly impossible to obtain a similar test statistic in the main experiment under any presence of motivation-based persistence. The replication study shows very similar coefficient estimates with regard to the persistence parameter and the overidentification test, and reasonable sensitivity of the test to the presence of motivation-based persistence.

6 Structural Estimation of the Parameter γ

After having confirmed that the mechanism behind the behavioral persistence in blood donations is action-based, we now structurally estimate the parameter γ that governs its extent. We impose the structure of the dynamic treatment effects from section 2.2 on our linear probability model to estimate the parameter γ and discuss the results. In keeping with the theoretical framework, we present this section in terms of the LATE.[26]

6.1 Estimation

To estimate the parameter γ, we impose the above structure on our linear probability model:

$$\text{Donation}_{ib,t} = \beta_1(\text{Ask}_{i,t} + \gamma_1\text{Ask}_{i,t-1} + \gamma_1^2\text{Ask}_{i,t-2} + \gamma_1^3\text{Ask}_{i,t-3}) + \beta_2' X_i + \delta_b + \epsilon_{ib,t}\,, \quad (6.1)$$

where $\beta_1 = p(F_c(B+\eta+\gamma) - F_c(B+\gamma)) + (1-p)(F_c(B+\eta) - F_c(B))$, and $\gamma_1 = F_c(B+\gamma) - F_c(B)$. If the costs, $\tilde{c}_t$, follow a uniform distribution, $\beta_1 = \eta$ and $\gamma_1 = \gamma$

[24] We also simulate the statistical power of the test statistic at the 5% level (see Panel (b) in Figure F.1 in Appendix F. The power to reject the hypothesis of no motivation-based persistence at the 5% significance level is, of course, significantly lower, reflecting some of the well-known power issues of overidentification tests (see, e.g., Bowsher 2002).

[25] This is likely due to the lower frequency of phone calls in that sample, which makes the instrument weaker and hence affects power of the overidentification test.

[26] In Appendix E we alternatively take the ITT perspective to estimate the parameter γ. The results are virtually identical.

are structural parameters of our framework, normalized by a constant. If the costs follow a general distribution, one could fully model the non-linearity from such distributions using the expressions involving $F_c()$ above. However, note that departures from uniformity would imply that the effect of the intervention in a given period depends on the intervention in the previous period. We found no such evidence in Table 4 (Column (3)). Thus, we focus on the uniform case and ignore these higher-order terms in our estimation below. Furthermore, we treat the control variables and fixed effects as outside the habit-forming structure. This is justified because the treatment is randomized and hence orthogonal to other variables.

Since γ_1 enters Equation (6.1) non-linearly, we search over a grid of γ in the range of [0.1,0.9] in steps of 0.01. Given γ_1, we then estimate the remaining parameters by OLS. The optimal γ^* in terms of minimizing the residual sum of squares feeds into the second step, where we estimate the same regression model for a grid of γ in the range of $[\gamma^*-0.01,\gamma^*+0.01]$ in steps of 0.001 to obtain a more precise estimate γ^{**}. Due to this procedure, we need to bootstrap the standard errors, while maintaining the clustering at the sponsor and individual level from the earlier specifications. Compared to estimating the parameter γ in the IV model, this approach is more efficient, because it exploits the implications of the structural model for higher lags of the intervention.

6.2 Results

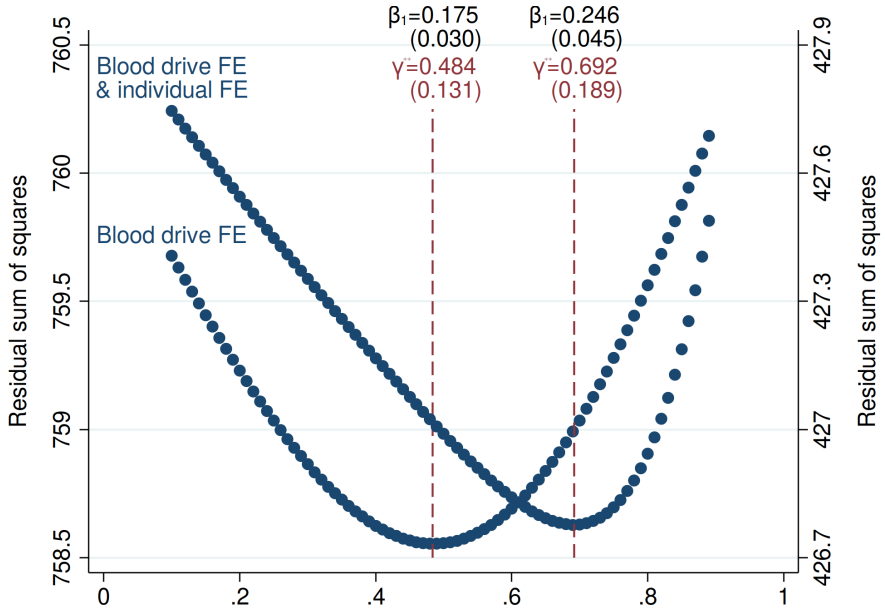
Figure 4 shows the residual sum of squares for different values of the parameter γ. One specification corresponds to Equation (6.1) that controls for individual characteristics and blood drive fixed effects. The other, alternative specification is more general and replaces the individual characteristics with individual fixed effects. For both specifications, the residual sum of squares exhibits a unique minimum. Thus, γ is well-identified in both specifications.

The estimates of the parameter, γ^{**}, are 0.484 (se=0.131) and 0.692 (se=0.189), respectively.[27] Hence, we reject the null hypothesis of no action-based persistence. Both estimates are also significantly smaller than 1 ($p < 0.01$ and $p = 0.05$, respectively), allowing us to also reject a model in which action-based persistence leads to a permanent change in behavior. Quantitatively, the estimates imply that donating at a blood drive today increases the probability of donating at the consecutive blood drive of the same sponsor six months later by 48 or 69 percentage points.

To put the effect of action-based persistence into perspective, we compare it to the direct effect of the intervention. The estimates indicate that the effect of donating today on the probability of donating at the consecutive blood drive is almost three times larger than the

[27]The estimated γ reflects action-based persistence net of time discounting, as we do not separately estimate the discount rate.

Figure 4: Grid search of the parameter γ



Notes: Relationship between γ and the residual sum of squares. Optimal γ^{**} is chosen to minimize the residual sum of squares. Bootstrapped standard errors in parentheses, corrected for two-way clustering on individuals and blood drives. The standard errors are generated using 1,000 bootstrap replications.

direct effect of asking potential donors by phone to make a donation.

Due to this amplification of the direct effect of a policy intervention, action-based persistence has a substantial economic impact. Consider our example of calling potential donors and asking them to donate at the upcoming blood drive. If we use the more conservative LATE estimates from Table 4 and γ, the direct effect of the successful phone calls on the donation rate is 18 percentage points. However, according to the geometric series in Equation (2.10), action-based persistence leads to a multiplier that amplifies the direct effect by $1/(1-\gamma)$. Thus, with our estimate of γ equal 0.484 action-based persistence amplifies the effect of successful phone call to a total of 34 percentage points – making the intervention almost twice as effective. In other words, to get one additional donation, the BTSRC would have to reach 1/0.18 or roughly six donors by phone without action-based persistence. However, with action-based persistence, it only needs to reach three donors.

The structural analysis could also be used to identify the welfare effects discussed in Section 2.3. Given the structural estimates for η and γ, we could also calculate the relevant expressions for Δw_0 and Δw_1.[28] This would also require identifying B, which could be calibrated as the mean donation frequency in the control group using Equation (A.2).

[28]See Appendix A for a derivation of the relevant expressions for uniform $F_c()$

7 Discussion

In this session we first discuss the long-term treatment effects and link them to the underlying mechanisms in a broader literature. Subsequently, we discuss the potential concern of using naturally occurring weather as instruments and argue why the choice of the instrument in our setup does not have such concerns.

7.1 Mechanisms leading to positive long-term treatment effects

The persistence induced by the phone call in our intervention takes on the same form as persistence induced by variation in weather, thus strongly corroborating our interpretation of action-based persistence in the sense of Stigler and Becker (1977). There are at least two possible interpretations of the mechanisms through which giving blood affects the subsequent motivation to donate: on the one hand, it could be self-signaling as in Dal Bó and Terviö (2013), by which donors with limited memory make inferences from their past behavior about their type. A donation leads them to update on their type, raising the probability to donate in the future, thus generating action-based persistence. This interpretation is also consistent with the effect trailing off after a year, as donors are Bayesian, and will return to the mean belief over time. On the other hand, it is also possible that giving blood may lead individuals to update the perceived costs and benefits of donating.[29] Since we are sampling from a population of donors who chose not to donate for at least a year, it is possible that we selected donors who received negative signals about the costs and benefits in the past. The additional push from the phone call may cause them to donate nevertheless, and the experience may lead them to update their perceptions, thus generating the persistence effect. However, our reduced-form evidence shows that persistence effects trail off and disappear completely after a year (Table 4). Thus for this mechanism to explain the evidence, it is also necessary that donors subsequently re-acquire their biased perceptions.

Several other studies also likekly fall into the category of action-based persistence: the persistence effects on voting (Fujiwara, Meng and Vogl 2016), social media use (Allcott, Gentzkow and Song 2022), and going to the gym (Charness and Gneezy 2009; Acland and Levy 2015; Royer, Stehr and Sydnor 2015) are all cases where action-based persistence offers a consistent and straightforward explanation of the evidence.

By contrast, other studies have found persistence that is consistent with a motivation-based mechanism. Byrne et al. (2019) vary the duration of feedback and persistence phases to model temporal variations in the treatment effects predicted by action-based vs. motivation-based models. Their evidence favors a mechanism in which habit formation occurs in the

[29] We are grateful to a referee for suggesting this interpretation.

attention weights on consumption, i.e., a motivation-based mechanism in our terminology. Similarly, Allcott and Rogers (2014) observe temporal variation in electirity use in the weeks around the time of arrival of home-energy reports. This "action and backsliding" is most plausibly also interpreted as motivation-based persistence, with the arrival of a home-energy report temporarily increasing attention to electrity use. While these tests rely on long panels of high-frequency data to distinguish between mechanisms, our approach uses two separate instruments for behavior.

It has also been observed that a large fraction of the home-energy reports' treatment effect persists even after they were discontinued (Allcott and Rogers 2014). However, as Brandon et al. (2017) show, this may in large part be due to households replacing inefficient appliances. They show that these treatment effects persist after the treated households moved out of the house and new untreated residents moved in. This suggests these treatment effects persist because of energy efficient appliances (e.g., LEDs) were left behind when the treated households moved out.

There are several studies that find persistence of treatment effects, where the underlying mechanism is not easily identifiable: Ferraro, Miranda and Price (2011) find that a strongly-framed normative message leads to reductions water consumption over at least two years, with the treatment effect gradually eroding. Both, motivation-based mechanisms (a persistent effect on perceived norms) or action-based mechsims are plausible in this case. Hussam et al. (2022) find persistent effects of interventions aimed at increasing the frequency of handwashing. While the authors also observe an anticipatory effect of the treatment that could be indicative of a forward-looking model of action-based persistence as in Becker and Murphy (1988), it is also possible that the announcement of the treatments had a direct persistent effect on motivation.[30]

Some studies fail to find persistence effects. In the context of blood donations, Goette and Stutzer (2020) find significant immediate effects of a lottery ticket on blood donation, but no persistence. In the context of of charitable donations, some treatments, e.g. an attractive solicitor (Landry et al. 2006), or the inclusion of a gift (Falk 2007), have an effect on contemporaneous donations, but there is no evidence of persistence.

7.2 Using Adverse Weather Conditions as an Instrument

Among all naturally occurring adverse weather conditions, rainfall has been used most frequently as a cause of exogenous variation in the economics literature (Fujiwara, Meng and Vogl 2016; Miguel, Satyanath and Sergenti 2004; Maccini and Yang 2009; Brückner and Ci-

[30] Allcott, Gentzkow and Song (2022) have a design that allows to test for a forward-looking component in habit formation, but their structural estimates indicate no significant evidence thereof.

ccone 2011). However, weather instruments are not without critics. Mellon (2023) assesses and documents potential exclusion-restriction violations from 159 studies in social science that use weather conditions as instruments. Most exclusion-restriction violations seem to happen when weather shocks are used to predict social and economic phenomena that occur much later or develop over a long time. For example, Sarsons (2015) criticises the use of rain shocks as an instrument to identify the effect of conflicts on economic growth. Using both monthly and yearly rainfall shocks matched to districts in India, she identifies exclusion-restriction violations in the rainfall instrument on income. In districts where dams protect against rain shocks, rainfall does not affect income. However, rainfall still correlates with conflict in all districts, regardless of the protection against rain shocks. That is, rain predicts conflict even when income is unaffected.

Unlike studies that use monthly or yearly weather shocks to instrument long-term outcomes, Fujiwara, Meng and Vogl (2016) uses daily rainfall as an instrument on voter turnout on the same day. The effect of rainfall on the costs of voting is much more immediate. Similarly, in our study, we use daily adverse weather conditions to predict donor tournout on the same day.

Overall, the effects of adverse weather conditions on behavior are immediate in Fujiwara, Meng and Vogl (2016) and in our study. Therefore, these studies should be unaffected by the prominent concerns regarding the use of weather instruments to identify the mechanisms behind complex, long-term social phenomena, such as conflicts or social and economic development. Moreover, our study relies on three different adverse weather conditions that may affect donations through distinct (opportunity) cost channels. Yet, they all lead to qualitatively identical results.

8 Conclusion

In this paper, we quantify the behavioral persistence in voluntary blood donations from a simple intervention and discriminate between action-based and motivation-based persistence. We combine a field experiment, asking a random subset of inactive registered blood donors to donate, with a natural experiment, exploiting random fluctuations in rainfall on the days of blood drives. This combination of two experiments allows us to demonstrate that the behavioral persistence in voluntary blood donations is action-based.

Identifying action-based persistence as the underlying mechanism is relevant for the design of effective policy interventions in the future. Since behavioral persistence is action-based, any policy intervention triggering an initial engagement in the prosocial activity benefits from a multiplier, as individuals who initially act and continue to engage in the prosocial

activity even after the intervention ended. If policy makers aim to maximize the long-run impact of an intervention, our results suggest that they should choose the intervention with the strongest immediate impact. In contrast, if behavioral persistence were motivation-based, i.e., driven by changes in motivation, the long-term effect of a policy intervention would depend on its ability to persistently change the individuals' future motivation. In that case, policy makers would require detailed understanding of how different interventions affect future motivation.

Our results also raise questions for future research. We show that action-based persistence in blood donations is consistent with the habit-forming mechanism in Stigler and Becker (1977). However, it is not clear which aspect of the behavior is habit forming: is it the narrowly-defined act of donating blood, or is it a potentially broader warm glow from prosocial behavior that exhibits a complementarity between current and future donation? While narrowly-defined habit formation with regard to one behavior already has important policy implications for the specific prosocial behavior under study, discovery of a broader "moral habit capital" could have more sweeping implications: one charity's work would impact the future willingness of its donors to engage in a wide array of prosocial behaviors. Future research should thus study whether and how interventions on one type of prosocial behavior spill over to other behaviors in subsequent periods.

9 Data Availability

Code replicating the tables, figures, and simulations in this article can be found in Bruhin et al. (2024) in the Harvard Dataverse, `https://doi.org/10.7910/DVN/OMB74R`.

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Appendix A Welfare Ananlysis under action-based persistence

In this section, we detail the derivation of the welfare effects of our intervention.
We begin by defining $E_x(u)$ to be the expected utility attained from donating blood. More specifically, $E_x(u) \equiv E(U \mid c < x)$. The right-hand side is interpreted as the expected utility conditional on donation occurring, which is when the utility of donation, x, is greater than the corresponding cost, c. The other parameters are defined as in the main text.
In formulating the welfare effects, we first consider the welfare of donors in the absence of the intervention:

$$W = p_{t-1} \cdot F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi) + (1 - p_{t-1}) \cdot F_c(B) \cdot (E_B(u) + \phi) \tag{A.1}$$

Here, p_{t-1} is the fraction of the population that have donated in period $t-1$, and $F_c(\cdot)$ is the probability of donating in period t. More explicitly, under the assumption of action-based persistence, $F_c(B+\gamma) = \mathbb{P}(c_t \leq \gamma d_{t-1} + B)$ captures the probability of donors donating in period t, conditional on them having donated in period $t-1$. As for the donors who have not donated in period $t-1$, action-based persistence is irrelevant. Thus, their probability of donation in period t is $F_c(B) = \mathbb{P}(c_t \leq B)$. On top of $E(u)$, each donation generates a positive external effect of ϕ per donation.
Further, we make the assumption that the donation has attained steady-state[31] , i.e., the fraction of population donating across time have stabilised:

$$p = p_t = p_{t-1} \tag{A.3}$$

Substituting equation A.3 into equation A.1,

$$W = p \cdot F_c(B+\gamma) \cdot E_{B+\gamma}(u) + (1-p) \cdot F_c(B) \cdot E_B(u) \tag{A.4}$$

Next, we examine what happens when the intervention takes place. There are two groups of donors to consider: donors covered by the intervention and donors not covered by the intervention. Hereinafter, we refer to these groups of donors as the *treated group, T,* and the

[31] Steady-state donation rate is given by $p(F+\gamma) + (1-p)F(B) = p$. This implies

$$p = \frac{F(B)}{1 - F(B+\gamma) + F(B)} \tag{A.2}$$

control group, C, respectively.

The intervention alters the welfare equation by increasing the utility of donation for the *treated group* by η, entering both $E(u)$ and $F_c(\cdot)$. Starting with the initial period, $t = 0$, where the intervention was implemented, the welfare of the *treated group*, W_0^T, and that of the *control group*, W_0^C, are:

$$W_0^T = p \cdot F_c(B+\gamma+\eta) \cdot (E_{B+\gamma+\eta}(u)+\phi) + (1-p) \cdot F_c(B+\eta) \cdot (E_{B+\eta}(u)+\phi) \quad \text{(A.5)}$$

$$W_0^C = p \cdot F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi) + (1-p) \cdot F_c(B) \cdot (E_B(u)+\phi) \quad \text{(A.6)}$$

We can then compute the welfare effects of the intervention in period $t = 0$ by abstracting Eqn. A.6 from Eqn. A.5:

$$\begin{aligned}
\Delta W_0 &= W_0^T - W_0^C \\
&= p \cdot \{F_c(B+\gamma+\eta) \cdot [E_{B+\gamma+\eta}(u)+\phi] - F_c(B+\gamma) \cdot [E_B(u)+\phi]\} \\
&\quad + (1-p) \cdot [F_c(B+\eta) \cdot (E_{B+\eta}(u)+\phi) - F_c(B) \cdot (E_B(u)+\phi)] \qquad \text{(A.7)}
\end{aligned}$$

As the intervention is one-off, in the next period $t = 1$, η no longer enters the utility function while the positive external effect ϕ remains. By denoting the fraction of donors in period $t = 0$ in the *treated group* and *control group* to be p_0^T and p_0^C, the welfare effects in period $t = 1$ can be written as:

$$\begin{aligned}
W_1^T &= p_0^T \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u)+\phi] + (1-p_0^T) \cdot F_c(B) \cdot [E_B(u)+\phi] \\
W_1^C &= p_0^C \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u)+\phi] + (1-p_0^C) \cdot F_c(B) \cdot [E_B(u)+\phi]
\end{aligned}$$

$$\begin{aligned}
\Delta W_1 &= W_1^T - W_1^C \\
&= (p_0^T - p_0^C) \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi)] - (p_0^T - p_0^C) \cdot [F_c(B) \cdot (E_B(u)+\phi)] \\
&= \Delta p_0 \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi)] - \Delta p_0 \cdot [F_c(B) \cdot (E_B(u)+\phi)] \\
&= \Delta p_0 \cdot \underbrace{[F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi) - F_c(B) \cdot (E_B(u)+\phi)]}_{\equiv \Delta w_1} \qquad \text{(A.8)}
\end{aligned}$$

where the last expression in equation (A.8) is defined as Δw_1 in equation (2.12) in the main

text. Similarly for period $t = 2$,

$$W_2^T = p_1^T \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u) + \phi] + (1 - p_1^T) \cdot F_c(B) \cdot [E_B(u) + \phi]$$
$$W_2^C = p_1^C \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u) + \phi] + (1 - p_1^C) \cdot F_c(B) \cdot [E_B(u) + \phi]$$

$$\begin{aligned}
\Delta W_2 &= W_2^T - W_2^C \\
&= (p_1^T - p_1^C) \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi)] - (p_1^T - p_1^C) \cdot [F_c(B) \cdot (E_B(u) + \phi)] \\
&= \Delta p_1 \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi)] - \Delta p_1 \cdot [F_c(B) \cdot (E_B(u) + \phi)] \\
&= \Delta p_1 \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi) - F_c(B) \cdot (E_B(u) + \phi)] \\
&= \frac{\Delta p_1}{\Delta p_0} \cdot \Delta p_0 \Delta w_1 \\
&= [F_c(B+\gamma) - F_c(B)] \cdot \Delta p_0 \Delta w_1
\end{aligned} \tag{A.9}$$

since

$$\begin{aligned}
\Delta p_1 &= p_1^T - p_1^C \\
&= p_0 \cdot F_c(B+\gamma) - p_0 \cdot F_c(B) \\
&= p_0 \cdot [F_c(B+\gamma) - F_c(B)]
\end{aligned}$$

Continuing this logic, for period $t = n$, the welfare effects will be $\Delta W_n = \frac{\Delta p_{n-1}}{\Delta p_0} \cdot \Delta p_0 \Delta w_1$. Also, noting that $\Delta p_n = p_0 \cdot [F_c(B+\gamma) - F_c(B)]^n$, we can generalize ΔW_n as:

$$\begin{aligned}
\Delta W_n &= \frac{\Delta p_{n-1}}{\Delta p_0} \cdot \Delta p_0 \Delta w_1 \\
&= [F_c(B+\gamma) - F_c(B)]^{n-1} \cdot \Delta p_0 \Delta w_1
\end{aligned}$$

Thus, summing over periods we get

$$\Delta W = \sum_{t=0}^{n} \Delta W_t = \Delta W_0 + \sum_{t=1}^{n} \Delta p_0 \Delta w_1 (F_c(B+\gamma) - F_c(B))^{t-1}$$

Taking the limit $t \to \infty$, we obtain

$$\Delta W = \Delta W_0 + \frac{\Delta p_0 \Delta w_1}{F_c(B+\gamma) - F_c(B)}$$

Appendix B First stages of LATE estimations

Table B.1: LATE First Stages

	Specification (1)				Specification (2)				Specification (3)						
Dependent variable	(1) Ask_t	(2) Ask_{t-1}	(3) Ask_{t-2}	(4) Ask_{t-3}	(5) Ask_t	(6) Ask_{t-1}	(7) Ask_{t-2}	(8) Ask_{t-3}	(9) Ask_t	(10) Ask_{t-1}	(11) Ask_{t-2}	(12) Ask_{t-3}	(13) $\text{Ask}_t \times \text{Ask}_{t-1}$	(14) $\text{Ask}_{t-1} \times \text{Ask}_{t-2}$	(15) $\text{Ask}_{t-2} \times \text{Ask}_{t-3}$
Call_t	0.530***	-0.00137	0.000457	0.000162	0.524***	-0.0118	-0.0142	-0.000608	0.546***	-0.0186	-0.0128	1.91e-05	0.000123	7.78e-05	0.000117
	(0.0195)	(0.00760)	(0.000409)	(0.000225)	(0.0211)	(0.0129)	(0.00879)	(0.00141)	(0.0189)	(0.0161)	(0.0151)	(0.00605)	(0.000194)	(0.000161)	(0.000153)
Call_{t-1}	-0.0121	0.530***	-0.00136	0.000166	-0.0183	0.519***	-0.0160	-0.000608	0.00757	0.529***	-0.0220	3.28e-05	3.53e-05	0.000248	0.000201
	(0.00984)	(0.0195)	(0.00760)	(0.000227)	(0.0171)	(0.0173)	(0.0127)	(0.00164)	(0.0128)	(0.0221)	(0.0172)	(0.00435)	(0.000183)	(0.000282)	(0.000216)
Call_{t-2}	0.000453	-0.0121	0.530***	-0.00166	-0.00574	-0.0225**	0.515***	-0.00243	-0.00215	-0.0108	0.528***	6.83e-05	-6.00e-05	0.000190	0.000418
	(0.000417)	(0.00984)	(0.0195)	(0.00763)	(0.0169)	(0.0107)	(0.0173)	(0.0110)	(0.0121)	(0.0137)	(0.0236)	(0.00272)	(0.000198)	(0.000237)	(0.000353)
Call_{t-3}	0.000505	0.000506	-0.0246	0.570***	-0.0151	-0.0200	-0.0534**	0.569***	-0.0154	-0.0297	-0.0135	0.574***	-7.07e-05	0.000165	0.000543
	(0.000633)	(0.000654)	(0.0196)	(0.0284)	(0.0211)	(0.0156)	(0.0226)	(0.0253)	(0.0181)	(0.0218)	(0.0183)	(0.0280)	(0.000298)	(0.000292)	(0.000533)
$\text{Call}_t \times \text{Call}_{t-1}$									-0.0886**	0.0223	0.0147	-2.19e-05	0.312***	-0.000165	-0.000134
									(0.0348)	(0.0295)	(0.0229)	(0.0118)	(0.0339)	(0.0152)	(0.0152)
$\text{Call}_{t-1} \times \text{Call}_{t-2}$									-0.0149	-0.0664**	0.0293	-5.46e-05	9.85e-05	0.312***	-0.000335
									(0.0309)	(0.0337)	(0.0327)	(0.0116)	(0.0152)	(0.0339)	(0.0152)
$\text{Call}_{t-2} \times \text{Call}_{t-3}$									0.000408	0.0146	-0.0599	-0.00746	0.000169	-0.000185	0.312***
									(0.0314)	(0.0232)	(0.0366)	(0.0280)	(0.0152)	(0.0152)	(0.0339)
Joint F-tests:															
1^{st} stage instruments		155.7				123.8							28.13		
Individual controls		Y													
Blood drive FE		Y				Y							Y		
Individual FE						Y							Y		
Observations		5,600				5,600							5,600		

Notes: In Column (1-3) the coefficients on the individual characteristics gender, age, and blood type are not shown. Standard errors clustered at the individual and blood drive level in parentheses. ** $p < 0.05$, *** $p < 0.01$..

Appendix C Checks on weather instrument

Table C.1: Balance Checks of Covariates

	(1) No adverse weather	(2) Adverse weather	(3) Difference
Call	0.4985	0.5067	0.0083
	(0.5001)	(0.5004)	(0.0243)
Age	41.2630	41.7881	0.5250
	(13.4889)	(13.6467)	(0.6574)
Male	0.5147	0.4971	-0.0176
	(0.4999)	(0.5005)	(0.0243)
O+ blood type	0.8181	0.8439	0.0259
	(0.3859)	(0.3633)	(0.0186)
O- blood type	0.0618	0.0520	-0.0098
	(0.2409)	(0.2223)	(0.0116)
A- blood type	0.1201	0.1040	-0.0161
	(0.3252)	(0.3056)	(0.0156)
Observations	2,281	519	2,800

Notes: Means with standard deviations in parentheses. Balance checks focus on the first two periods of the experiment, as the phone call was administered during these periods (see Table 1). The variable Call indicates the frequency of the phone call.

Appendix D Replication Study

Table D.1 shows the results, analogously to Table 5. In the first stage in Column (1), both excluded instruments are strong with a joint F-statistic of 34. Compared to Table 5, the coefficients on the excluded instruments are within the confidence bounds. The estimated second stage coefficient on the past donation in Column (2) is 0.29. The p-value of the Sargan-Hansen J-test of overidentifying restrictions is 0.26, indicating that the null hypothesis that both instruments are exogenous is not rejected.

Columns (3)-(4) show the second stage estimates when excluding only one instrument at a time. Consistent with the insignificant overidentifying restrictions test the estimated effect of past donation is within the confidence bounds in both columns and both instruments are individually strong. The coefficient on past donation is even somewhat *smaller* when using the phone call compared to using adverse weather as instrument - the opposite as would be expected if the phone call had persistent effects on the motivation to donate.

Columns (5)-(6) exclude only one instrument at a time, while *including the additional instrument* as control variable. As expected, neither the lagged phone call, nor lagged adverse weather have a persistent direct effect on donations, once controlling for past donations.

Even tough the quasi-experimental data is not directly comparable to the field experiment and is considerably more noisy, qualitatively the results from this replication exercise are reassuringly consistent.

Table D.1: Action-based vs motivation-based persistence (replication study)

	(1)	(2)	(3)	(4)	(5)	(6)
Dependent variable:	Donation_{t-1}	Donation_t	Donation_t	Donation_t	Donation_t	Donation_t
$\widehat{\text{Donation}_{t-1}}$		0.288**	0.225	0.672	0.225	0.672
		(0.140)	(0.155)	(0.391)	(0.155)	(0.396)
Phone call_{t-1}	0.122***					-0.0543
	(0.0178)					(0.0532)
$\text{Adverse weather}_{t-1}$	-0.0906***				-0.0405	
	(0.0184)				(0.0328)	
Phone call_t	-0.0228	0.0626***	0.0599***	0.0785***	0.0610***	0.0712***
	(0.0198)	(0.0206)	(0.0206)	(0.0284)	(0.0205)	(0.0251)
Adverse weather_t	-0.0228	0.00201	0.000969	0.00833	-0.00416	0.00602
	(0.0252)	(0.0194)	(0.0192)	(0.0237)	(0.0195)	(0.0228)
Excluded instrument		Both	Phone call	Adverse weather	Phone call	Adverse weather
Kleibergen/Paap F-statistic		33.76	46.23	18.56	46.76	24.19
Sargan-Hansen J-test (p-val.)		0.263				
Observations	9,611	9,611	9,611	9,611	9,611	9,611

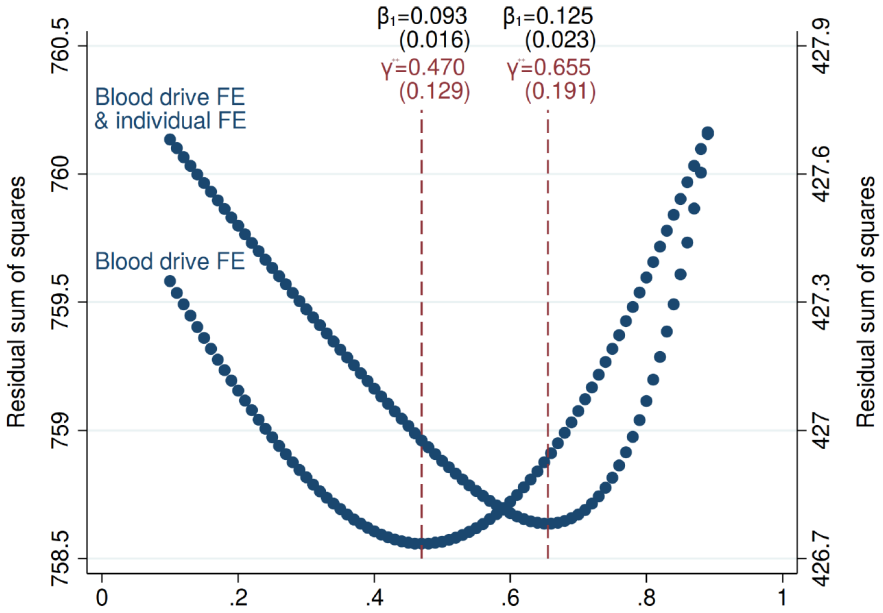
Notes: Regressions additionally include individual controls (gender, age, blood types) and week of the year and sponsor fixed effects. Standard errors clustered at the individual and blood drive level in parentheses. ** $p < 0.05$, *** $p < 0.01$

Appendix E ITT Parameter γ

Figure E.1 shows the ITT version of Figure 4 of the distribution of γ for the models with blood drive fixed effects and additionally with individual fixed effects, in line with Columns (4)-(5) in Table 4. The plot also includes the residual sum of squares to show that the parameters are well identified: in each of the specifications, there is a clear minimum.

The resulting γ parameters are 0.470 ($p < 0.01$) and 0.655 ($p < 0.01$), respectively. The estimates are very similar as in Figure 4 and clearly reject the benchmark of no action-based persistence.

Figure E.1: Grid search of the parameter γ



Notes: Relationship between γ and the residual sum of squares. Optimal γ^{**} is chosen to minimize the residual sum of squares. Two-way individual and blood drive cluster robust standard standard errors from 1,000 bootstrap replications in parentheses.

Appendix F Simulation Evidence

Figure F.1 Panel (a) shows the share of cases from 10,000 simulations with p-value of the Sargan-Hansen J-test of over-identifying restrictions being less than the observed value of 0.942 in Table 5 (experimental sample). Figure F.2 Panel (a) features an analogous plot for using the larger replication sample, with the p-value of 0.263 obtained from Table C.1. We also simulate the statistical power of the test statistic at the 5% level in the respective figures' Panel (b). The following steps outline the methodology employed in conducting these simulations.

F.1 Persistence Shifting

The persistence in the simulation were artificially distributed between action-based and motivation-based, while keeping the overall level of persistence constant. Table F.1 illustrates this main idea in Column (3), contrasting it to the case where persistence is purely action-based (Column (2)).

Table F.1: Reduce-form Persistence Effect

(1) Period	(2) Action-based persistence only	(3) α% Action + $(1-\alpha)$% Motivation-based persistence
t = 0	0	0
t = 1	$\beta_{call}\gamma$	$\alpha\beta_{call}\gamma + (1-\alpha)\beta_{call}\gamma$
t = 2	$\beta_{call}\gamma^2$	$\alpha\beta_{call}\gamma^2 + (1-\alpha)\beta_{call}\gamma^2$
t = 3	$\beta_{call}\gamma^3$	$\alpha\beta_{call}\gamma^3 + (1-\alpha)\beta_{call}\gamma^3$

In the context of Table F.1, the phone call intervention occurs in period 0. There is no persistence in this period under either scenarios, as no phone calls were made in the preceding period. In period 1, the overall level of persistence is $\beta_{call}\gamma$, where β_{call} is the effect of the phone call intervention on donation in the previous period, and γ is the persistence coefficient. $\beta_{call}\gamma$ is then divided between action-based and motivation-based persistence by ascribing α% persistence to action-based, and the remainder $(1-\alpha)$% to motivation-based. Where persistence is action-based only, $\alpha = 1$ yielding $\beta_{call}\gamma$.

Extrapolating the reasoning above into periods 2 and 3, the overall persistence levels are $(\beta_{call}\gamma)\cdot\gamma = \beta_{call}\gamma^2$ and $(\beta_{call}\gamma^2)\cdot\gamma = \beta_{call}\gamma^3$ respectively. The fraction of persistence that is action-based in period 2 is $\alpha\gamma\cdot\beta_{call}\gamma = \alpha\beta_{call}\gamma^2$ and $\alpha\gamma\cdot\beta_{call}\gamma^2 = \alpha\beta_{call}\gamma^3$ in period 3.

The balance are the motivation-based persistence in those periods i.e., $\beta_{call}\gamma^2 - \alpha\beta_{call}\gamma^2 = \beta_{call}(1-\alpha)\gamma^2$ and $\beta_{call}\gamma^3 - \alpha\beta_{call}\gamma^3 = \beta_{call}(1-\alpha)\gamma^3$. This idea of persistence-shifting is incorporated in the simulation model detailed below.

F.2 Simulation Model for Field Experiment

The first step in running the simulation is to construct a blood donation dataset that mimics that of the experimental dataset. To do so, we require a model that predicts whether an individual would make a donation, given conditions similar to that of the experiment. The model that we have formulated is:

$$u_{it} = \beta_0 + \beta_{call} \cdot P_{it} + \beta_{adv} \cdot R_{it} + \Delta B_{it} + \alpha \cdot \gamma \cdot d_{it-1} + \varepsilon_{it} \tag{F.1}$$

$$\varepsilon_{it} \sim U(0,1)$$

where:

α, β_{call}, and γ are as defined previously
β_{adv} is the adverse weather coefficient
d_{it-1} is the blood donation in the previous period
P_{it} is the indicator for phone call
R_{it} is the indicator for adverse weather
ΔB_{it} is the change in baseline motivation

The coefficients β_{call} (0.0949) and β_{adv} (–0.0724) are obtained from Table 5, while the two γ values (0.484 and 0.692) are from Section 6.3. α is chosen from 0.1 – 1.0 in increments of 0.1. R_{it} is a random binary indicator where $\mathbb{P}(R_{it} = 1) = 0.204$, mirroring the actual frequency of adverse weather.

Described in Table F.2 below, P_{it} replicates the indicator of phone call intervention from the experimental sample with 350 individuals under each condition.

Table F.2: Phone call intervention indicator, P_{it}

Group	Individuals	P_{i1}	P_{i2}	P_{i3}	P_{i4}
C1	$i \in \{1, 2 \ldots 350\}$	1	0	0	0
C2	$i \in \{351, 352 \ldots 700\}$	0	1	0	0
C12	$i \in \{701, 702 \ldots 1050\}$	1	1	0	0
Control	$i \in \{1051, 1052 \ldots 1,400\}$	0	0	0	0

Next, ΔB_{it} is defined in accordance with Table 1, while accounting for the period(s) of phone call intervention. The individual ΔB_{it} of each group over the 4 experimental periods are specified in Table F.3 below:

Table F.3: Change in baseline motivation, ΔB_{it}

Group	ΔB_{i1}	ΔB_{i2}	ΔB_{i3}	ΔB_{i4}
C1	0	$\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^2$	$\beta_{call}(1-\alpha)\gamma^3$
C2	0	0	$\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^2$
C12	0	$\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^2+$ $\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^3+$ $\beta_{call}(1-\alpha)\gamma^2$
Control	0	0	0	0

F.3 Running the Simulation

The simulation commences in period $t = 1$, where β_0 is selected such that the mean donation of the control group is 0.1275 as per Figure 6. Next, for each individual i, ε_{i1} is randomly drawn from a continuous uniform distribution $U(0, 1)$. If $u_{i1} \geq \varepsilon_{i1}$, $d_{i1} = 1$, else $d_{i1} = 0$. This process is then repeated for $t = 2$, $t = 3$ and $t = 4$ to generate d_{i2}, d_{i3} and d_{i4} respectively. Thereafter, the Sargan-Hansen J-test is conducted on the simulated dataset.

The entire process is repeated 10,000 times and the share of cases with Sargan p-values less than 0.942 (or 0.263, under the larger replication sample) is computed.

F.4 Simulation for Replication Sample Study

The steps in simulating the larger replication sample is largely similar to what is described above. The only difference are:

1. Table F.2 will have 801 individuals instead of 350 in each group.
2. $\mathbb{P}(R_{it} = 1) = 0.233$ to reflect the higher adverse weather frequency in the replication sample.
3. $\beta_{call} = 0.122$ and $\beta_{adv} = -0.0906$, which are obtained from Table D.1.

F.5 Description of Utility/Cost Distribution Plots

Noting the presence of non-monotonicities in Figure F.1, particularly at $\alpha < -0.2$ and $\alpha > 1.7$, this section attempts to explain those patterns using the utility and cost distributions found in Figure F.3. We begin with a description of Figure F.3 and how it should be

interpreted in relation to the non-monotonic powers found in Figure F.1.

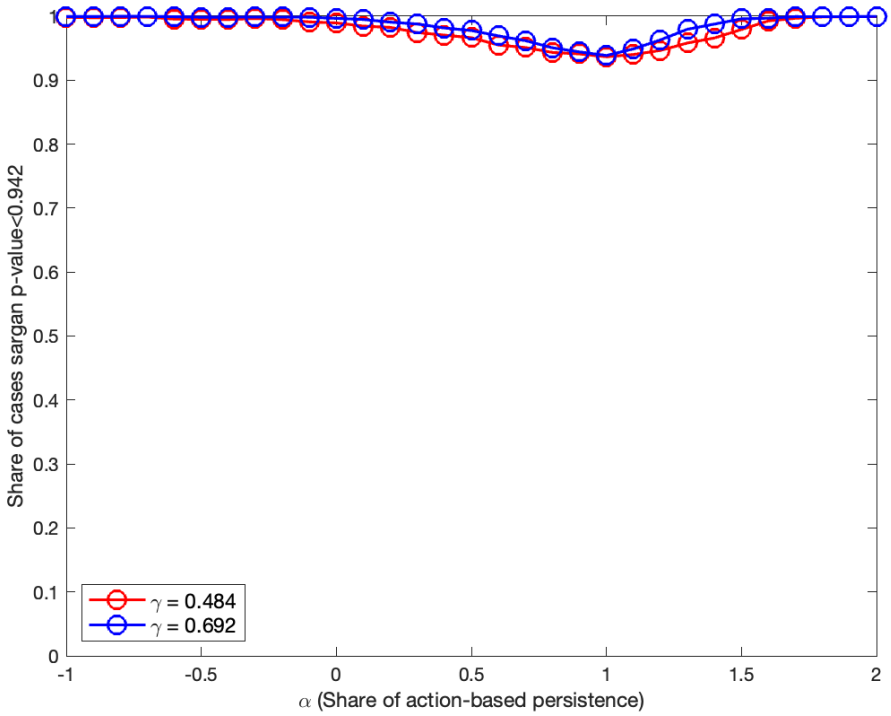
Figure F.3 comprises of 15 sub-figures, each of which takes on an alpha value between $[-1, 2)$ in increments of 0.2. Within each sub-plot, there are 4 periods: $t = 1$, $t = 2$, $t = 3$ and $t = 4$, corresponding to the 4 donation periods of the experiment. The same 3,204 individuals appear in all 4 periods. They are divided into the control group (Control) and treatment groups (C1, C2, C12) with 804 individuals respectively.

In any given period t, the light green markers simulate the individual cost, e_{it}, which is randomly drawn from the uniform distribution $U(0, 1)$. The dark green markers simulate the individual's utility, u_{it}, which is computed using Equation (F.1). Note that the number of unique dark green lines in each period corresponds to the number of unique utility values that can be obtained. This is in turn dependent on the number of possible combinations of R_{it} and P_{it}. Taking Figure 3(a) as an example, in period one $|R_{i,1}| = 2$ and $|P_{i,1}| = 2$, therefore there would be $2 \times 2 = 4$ dark green lines. For period 2, $|R_{i,1}| = 2$, $|P_{i,1}| = 2$, $|R_{i,2}| = 2$ and $|P_{i,2}| = 2$, giving $2 \times 2 \times 2 \times 2 = 16$ lines. Using a similar argument, period 3 and 4 will have 32 and 64 lines respectively.

Next, on how Figure F.3 explains the non-monotonicities in power: first, we discern that the dark green markers may be interpreted as the probability that an individual will make a donation. Next, by scrutinizing the shifting patterns of the green markers as α changes, we find that for the figures with $\alpha \leq -0.6$ and $\alpha \geq 1.2$, there are individuals that always donate or never donate regardless of the cost they incur. The implication of this is that the sample size is effectively reduced, which would in turn decreases the power of the Sargan test, resulting in the non-monotonic patterns observed in Figure F.1.

Figure F.1: Field Experiment Sample

(a) Share of cases with Sargan p-value<0.942



(b) Share of cases with Sargan p-value<0.05

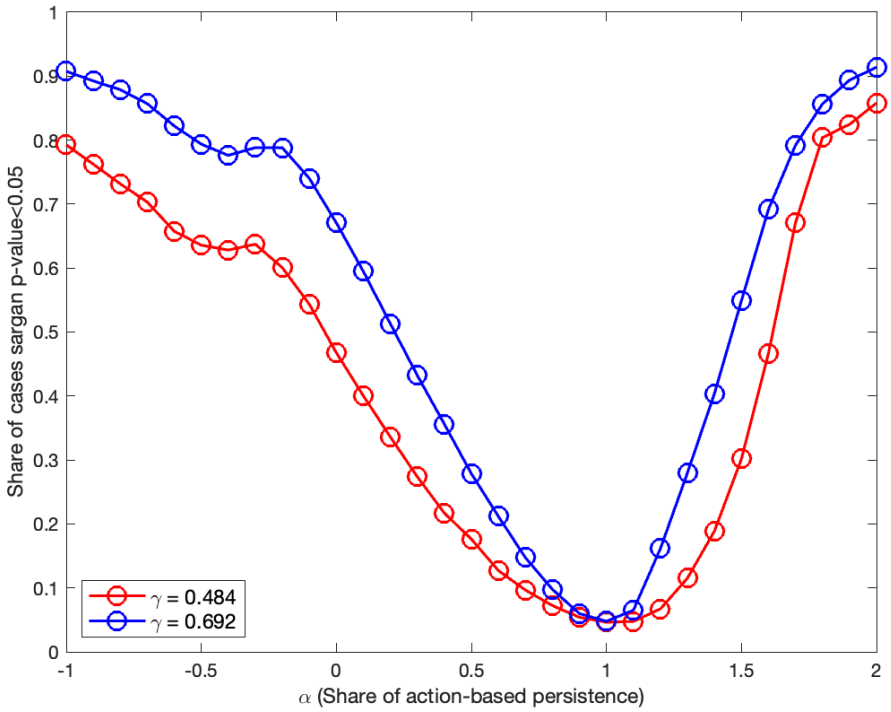
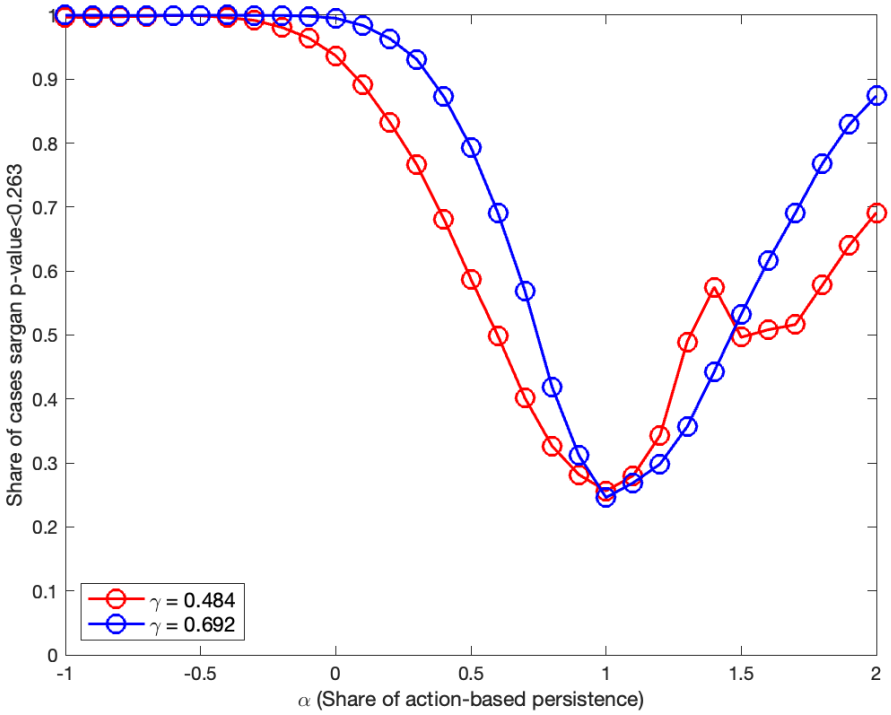


Figure F.2: Replication Sample

(a) Share of cases with Sargan p-value<0.263



(b) Share of cases with Sargan p-value<0.05

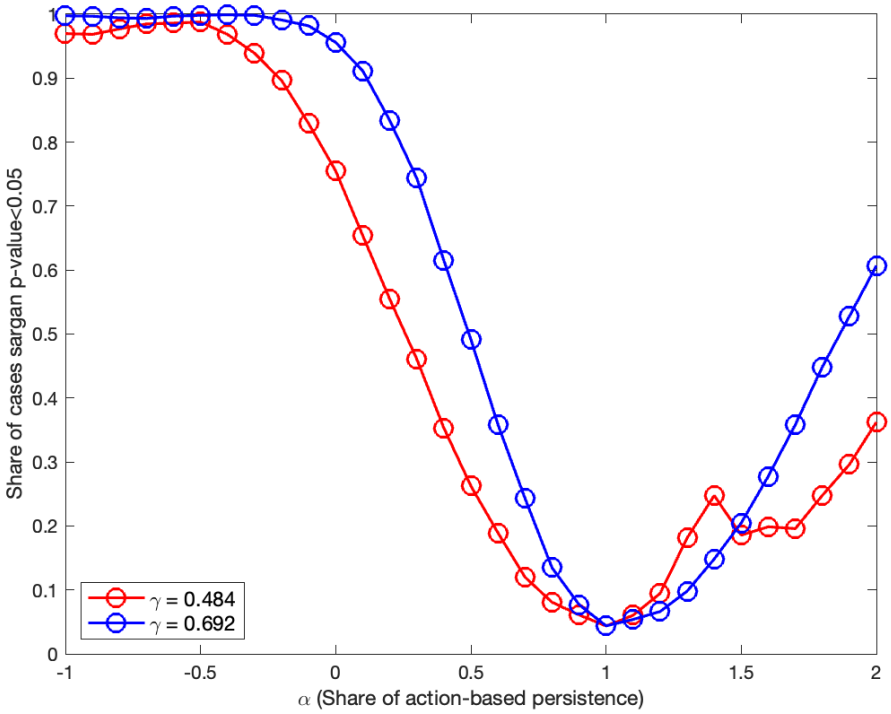
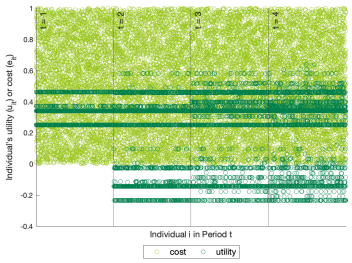
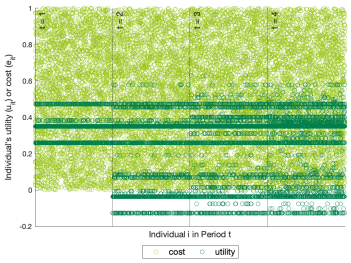


Figure F.3: Utility/Cost Distribution

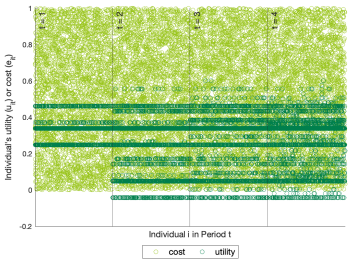
(a) $\alpha = -1.0$



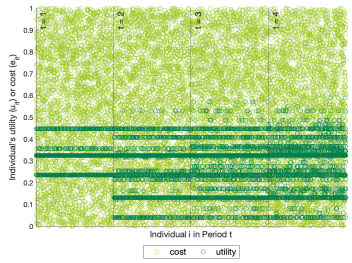
(b) $\alpha = -0.8$



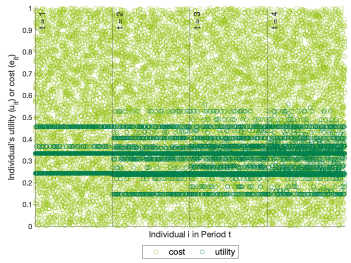
(c) $\alpha = -0.6$



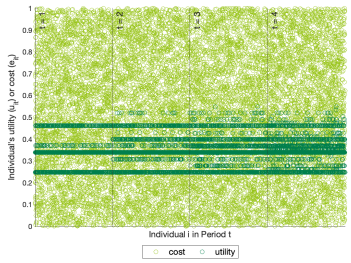
(d) $\alpha = -0.4$



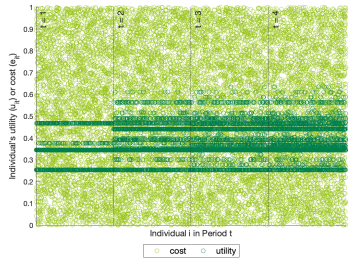
(e) $\alpha = -0.2$



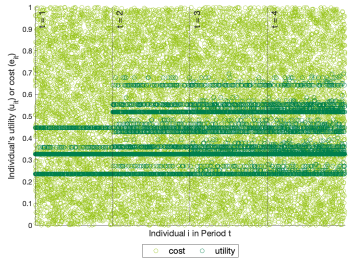
(f) $\alpha = 0.0$



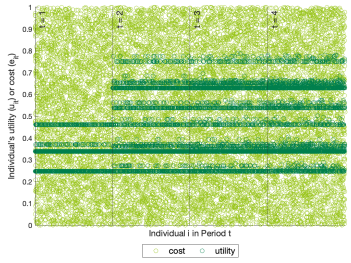
(g) $\alpha = 0.2$



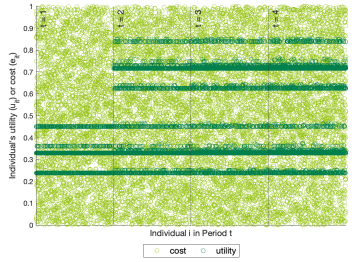
(h) $\alpha = 0.4$



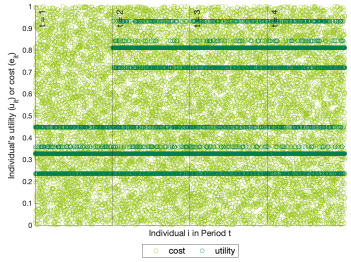
(i) $\alpha = 0.6$



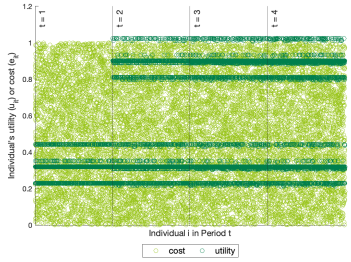
(j) $\alpha = 0.8$



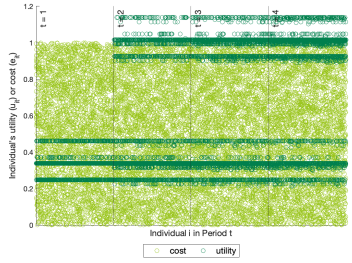
(k) $\alpha = 1.0$



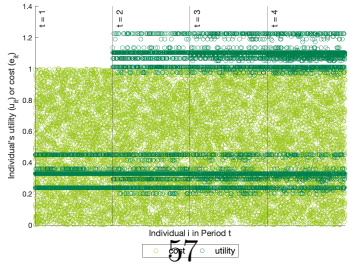
(l) $\alpha = 1.2$



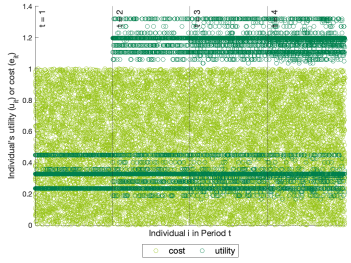
(m) $\alpha = 1.4$



(n) $\alpha = 1.6$



(o) $\alpha = 1.8$



F.6 Setup of Persistence Example

In this section, we simulate the persistency in blood donors with cyclical change in baseline motivation. Our simulation is based on the assumption that there are 2 types of donors, summertime donors and wintertime donors.

We simulate the persistence of donors across 4 periods, t_1, t_2, t_3 and t_4, over a span of 2 years. We define t_1 and t_3 to be summertime, whilst t_2 and t_4 to be wintertime. To model the seasonal preference in donating blood of the respective donor types, we incorporated asymmetric cyclicality in the donor's baseline motivation, as illustrated in Table F.4.

Table F.4: Cyclical Change in Baseline Motivation

	Baseline Motivation			
Donor Type	$t = 1$	$t = 2$	$t = 3$	$t = 4$
Summer	$B + \delta B$	$B - \delta B - \eta$	$B + \delta B$	$B - \delta B - \eta$
Winter	$B - \delta B - \eta$	$B + \delta B$	$B - \delta B - \eta$	$B + \delta B$

In the absence of seasonality, both types of donors possess a baseline motivation of B. During summer, i.e., $t = 1$ and $t = 3$, the increase in motivation for summer donors to donate is equal to δB, while their demotivated winter counterparts' motivation falls by a slightly larger magnitude of $\delta B + \eta$. This change in baseline motivation reverses during wintertime, i.e., $t = 2$ and $t = 4$.

With these baseline motivations along with Equation (F.1), we simulate the donor's persistence in the simplest case by setting $\delta = 1$ while also assuming no motivation-based persistence by setting $\alpha = 1$, which yields the following:

$$u_{it} = \beta_0 + \beta_{call} \cdot P_{it} + \beta_{adv} \cdot R_{it} + B + I_{on} \cdot B - I_{off} \cdot (B + \eta) + \gamma \cdot d_{it-1} + \varepsilon_{it} \tag{F.2}$$

$$\varepsilon_{it} \sim U(0, 1)$$

where:

β_{call}, β_{adv}, d_{it-1}, P_{it}, R_{it}, and γ are as defined previously

I_{on} is an indicator of *"on-cycle"* donation

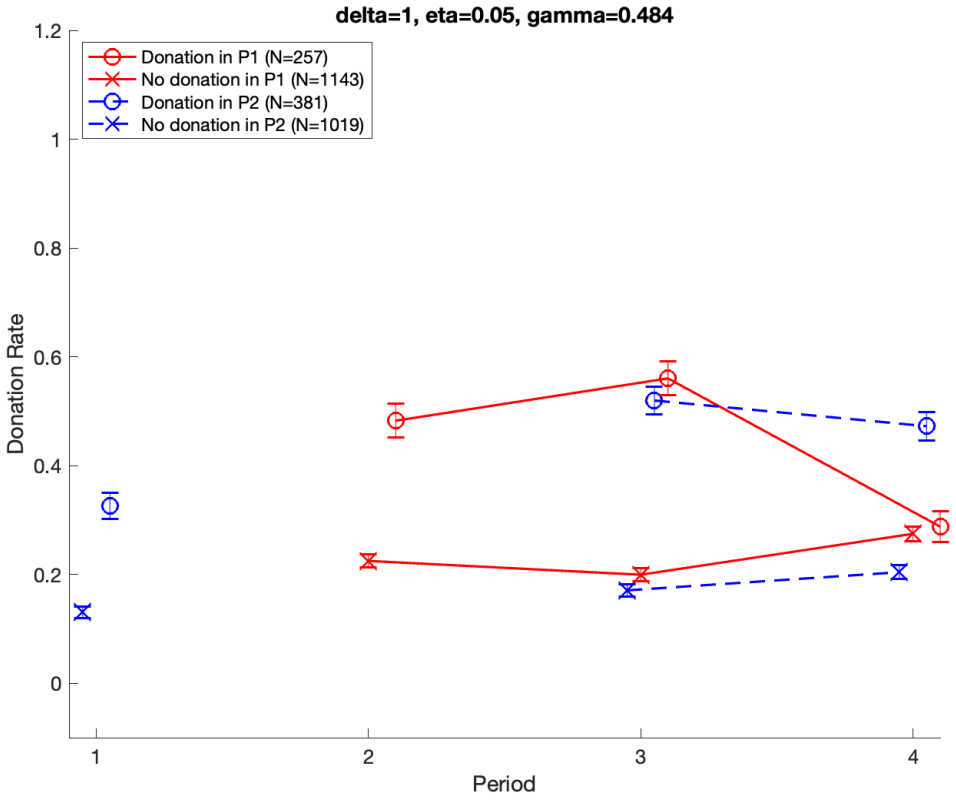
e.g. summer donors during summertime, vice versa

I_{off} is an indicator of *"off-cycle"* donation

e.g. summer donors during wintertime, vice versa

The results of the simulation are plotted in Figure F.4. The circle markers are the donation rates conditional on the donors having donated in period 1 (red) or period 2 (blue), while the crosses are conditional on the donors having not donated in period 1 (red) or period 2 (blue).

Figure F.4: Persistency in Donors with Cyclical Change in Baseline Motivation



Appendix A Welfare Ananlysis under action-based persistence

In this section, we detail the derivation of the welfare effects of our intervention.
We begin by defining $E_x(u)$ to be the expected utility attained from donating blood. More specifically, $E_x(u) \equiv E(U \mid c < x)$. The right-hand side is interpreted as the expected utility conditional on donation occurring, which is when the utility of donation, x, is greater than the corresponding cost, c. The other parameters are defined as in the main text.
In formulating the welfare effects, we first consider the welfare of donors in the absence of the intervention:

$$W = p_{t-1} \cdot F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi) + (1 - p_{t-1}) \cdot F_c(B) \cdot (E_B(u) + \phi) \tag{A.1}$$

Here, p_{t-1} is the fraction of the population that have donated in period $t-1$, and $F_c(\cdot)$ is the probability of donating in period t. More explicitly, under the assumption of action-based persistence, $F_c(B+\gamma) = \mathbb{P}(c_t \leq \gamma d_{t-1} + B)$ captures the probability of donors donating in period t, conditional on them having donated in period $t-1$. As for the donors who have not donated in period $t-1$, action-based persistence is irrelevant. Thus, their probability of donation in period t is $F_c(B) = \mathbb{P}(c_t \leq B)$. On top of $E(u)$, each donation generates a positive external effect of ϕ per donation.
Further, we make the assumption that the donation has attained steady-state[31] , i.e., the fraction of population donating across time have stabilised:

$$p = p_t = p_{t-1} \tag{A.3}$$

Substituting equation A.3 into equation A.1,

$$W = p \cdot F_c(B+\gamma) \cdot E_{B+\gamma}(u) + (1-p) \cdot F_c(B) \cdot E_B(u) \tag{A.4}$$

Next, we examine what happens when the intervention takes place. There are two groups of donors to consider: donors covered by the intervention and donors not covered by the intervention. Hereinafter, we refer to these groups of donors as the *treated group, T,* and the

[31] Steady-state donation rate is given by $p(F+\gamma) + (1-p)F(B) = p$. This implies

$$p = \frac{F(B)}{1 - F(B+\gamma) + F(B)} \tag{A.2}$$

control group, C, respectively.

The intervention alters the welfare equation by increasing the utility of donation for the *treated group* by η, entering both $E(u)$ and $F_c(\cdot)$. Starting with the initial period, $t = 0$, where the intervention was implemented, the welfare of the *treated group*, W_0^T, and that of the *control group*, W_0^C, are:

$$W_0^T = p \cdot F_c(B+\gamma+\eta) \cdot (E_{B+\gamma+\eta}(u)+\phi) + (1-p) \cdot F_c(B+\eta) \cdot (E_{B+\eta}(u)+\phi) \quad \text{(A.5)}$$

$$W_0^C = p \cdot F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi) + (1-p) \cdot F_c(B) \cdot (E_B(u)+\phi) \quad \text{(A.6)}$$

We can then compute the welfare effects of the intervention in period $t = 0$ by abstracting Eqn. A.6 from Eqn. A.5:

$$\begin{aligned}
\Delta W_0 &= W_0^T - W_0^C \\
&= p \cdot \{F_c(B+\gamma+\eta) \cdot [E_{B+\gamma+\eta}(u)+\phi] - F_c(B+\gamma) \cdot [E_B(u)+\phi]\} \\
&\quad + (1-p) \cdot [F_c(B+\eta) \cdot (E_{B+\eta}(u)+\phi) - F_c(B) \cdot (E_B(u)+\phi)] \qquad \text{(A.7)}
\end{aligned}$$

As the intervention is one-off, in the next period $t = 1$, η no longer enters the utility function while the positive external effect ϕ remains. By denoting the fraction of donors in period $t = 0$ in the *treated group* and *control group* to be p_0^T and p_0^C, the welfare effects in period $t = 1$ can be written as:

$$\begin{aligned}
W_1^T &= p_0^T \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u)+\phi] + (1-p_0^T) \cdot F_c(B) \cdot [E_B(u)+\phi] \\
W_1^C &= p_0^C \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u)+\phi] + (1-p_0^C) \cdot F_c(B) \cdot [E_B(u)+\phi]
\end{aligned}$$

$$\begin{aligned}
\Delta W_1 &= W_1^T - W_1^C \\
&= (p_0^T - p_0^C) \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi)] - (p_0^T - p_0^C) \cdot [F_c(B) \cdot (E_B(u)+\phi)] \\
&= \Delta p_0 \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi)] - \Delta p_0 \cdot [F_c(B) \cdot (E_B(u)+\phi)] \\
&= \Delta p_0 \cdot \underbrace{[F_c(B+\gamma) \cdot (E_{B+\gamma}(u)+\phi) - F_c(B) \cdot (E_B(u)+\phi)]}_{\equiv \Delta w_1} \qquad \text{(A.8)}
\end{aligned}$$

where the last expression in equation (A.8) is defined as Δw_1 in equation (2.12) in the main

text. Similarly for period $t = 2$,

$$W_2^T = p_1^T \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u) + \phi] + (1 - p_1^T) \cdot F_c(B) \cdot [E_B(u) + \phi]$$
$$W_2^C = p_1^C \cdot F_c(B+\gamma) \cdot [E_{B+\gamma}(u) + \phi] + (1 - p_1^C) \cdot F_c(B) \cdot [E_B(u) + \phi]$$

$$\begin{aligned}
\Delta W_2 &= W_2^T - W_2^C \\
&= (p_1^T - p_1^C) \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi)] - (p_1^T - p_1^C) \cdot [F_c(B) \cdot (E_B(u) + \phi)] \\
&= \Delta p_1 \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi)] - \Delta p_1 \cdot [F_c(B) \cdot (E_B(u) + \phi)] \\
&= \Delta p_1 \cdot [F_c(B+\gamma) \cdot (E_{B+\gamma}(u) + \phi) - F_c(B) \cdot (E_B(u) + \phi)] \\
&= \frac{\Delta p_1}{\Delta p_0} \cdot \Delta p_0 \Delta w_1 \\
&= [F_c(B+\gamma) - F_c(B)] \cdot \Delta p_0 \Delta w_1
\end{aligned} \tag{A.9}$$

since

$$\begin{aligned}
\Delta p_1 &= p_1^T - p_1^C \\
&= p_0 \cdot F_c(B+\gamma) - p_0 \cdot F_c(B) \\
&= p_0 \cdot [F_c(B+\gamma) - F_c(B)]
\end{aligned}$$

Continuing this logic, for period $t = n$, the welfare effects will be $\Delta W_n = \frac{\Delta p_{n-1}}{\Delta p_0} \cdot \Delta p_0 \Delta w_1$. Also, noting that $\Delta p_n = p_0 \cdot [F_c(B+\gamma) - F_c(B)]^n$, we can generalize ΔW_n as:

$$\begin{aligned}
\Delta W_n &= \frac{\Delta p_{n-1}}{\Delta p_0} \cdot \Delta p_0 \Delta w_1 \\
&= [F_c(B+\gamma) - F_c(B)]^{n-1} \cdot \Delta p_0 \Delta w_1
\end{aligned}$$

Thus, summing over periods we get

$$\Delta W = \sum_{t=0}^{n} \Delta W_t = \Delta W_0 + \sum_{t=1}^{n} \Delta p_0 \Delta w_1 (F_c(B+\gamma) - F_c(B))^{t-1}$$

Taking the limit $t \to \infty$, we obtain

$$\Delta W = \Delta W_0 + \frac{\Delta p_0 \Delta w_1}{F_c(B+\gamma) - F_c(B)}$$

Appendix B First stages of LATE estimations

Table B.1: LATE First Stages

	Specification (1)				Specification (2)				Specification (3)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)
Dependent variable	Ask_t	Ask_{t-1}	Ask_{t-2}	Ask_{t-3}	Ask_t	Ask_{t-1}	Ask_{t-2}	Ask_{t-3}	Ask_t	Ask_{t-1}	Ask_{t-2}	Ask_{t-3}	$Ask_t \times Ask_{t-1}$	$Ask_{t-1} \times Ask_{t-2}$	$Ask_{t-2} \times Ask_{t-3}$
$Call_t$	0.530***	-0.00137	0.000457	0.000162	0.524***	-0.0118	-0.0142	-0.000608	0.546***	-0.0186	-0.0128	1.91e-05	0.000123	7.78e-05	0.000117
	(0.0195)	(0.00760)	(0.000409)	(0.000225)	(0.0211)	(0.0129)	(0.00879)	(0.00141)	(0.0189)	(0.0161)	(0.0151)	(0.00605)	(0.000194)	(0.000161)	(0.000153)
$Call_{t-1}$	-0.0121	0.530***	-0.00136	0.000166	-0.0183	0.519***	-0.0160	-0.000608	0.00757	0.529***	-0.0220	3.28e-05	3.53e-05	0.000248	0.000201
	(0.00984)	(0.0195)	(0.00760)	(0.000227)	(0.0171)	(0.0173)	(0.0127)	(0.00164)	(0.0128)	(0.0221)	(0.0172)	(0.00435)	(0.000183)	(0.000282)	(0.000216)
$Call_{t-2}$	0.000453	-0.0121	0.530***	-0.00166	-0.00574	-0.0225**	0.515***	-0.00243	-0.00215	-0.0108	0.528***	6.83e-05	-6.00e-05	0.000190	0.000418
	(0.000417)	(0.00984)	(0.0195)	(0.00763)	(0.0169)	(0.0107)	(0.0173)	(0.0110)	(0.0121)	(0.0137)	(0.0236)	(0.00272)	(0.000198)	(0.000237)	(0.000353)
$Call_{t-3}$	0.000505	0.000506	-0.0246	0.570***	-0.0151	-0.0200	-0.0534**	0.569***	-0.0154	-0.0297	-0.0135	0.574***	-7.07e-05	0.000165	0.000543
	(0.000633)	(0.000654)	(0.0196)	(0.0284)	(0.0211)	(0.0156)	(0.0226)	(0.0253)	(0.0181)	(0.0218)	(0.0183)	(0.0280)	(0.000298)	(0.000292)	(0.000533)
$Call_t \times Call_{t-1}$									-0.0886**	0.0223	0.0147	-2.19e-05	0.312***	-0.000165	-0.000134
									(0.0348)	(0.0295)	(0.0229)	(0.0118)	(0.0339)	(0.0152)	(0.0152)
$Call_{t-1} \times Call_{t-2}$									-0.0149	-0.0664**	0.0293	-5.46e-05	9.85e-05	0.312***	-0.000335
									(0.0309)	(0.0337)	(0.0327)	(0.0116)	(0.0152)	(0.0339)	(0.0152)
$Call_{t-2} \times Call_{t-3}$									0.000408	0.0146	-0.0599	-0.00746	0.000169	-0.000185	0.312***
									(0.0314)	(0.0232)	(0.0366)	(0.0280)	(0.0152)	(0.0152)	(0.0339)
Joint F-tests:															
1^{st} stage instruments		155.7				123.8							28.13		
Individual controls		Y													
Blood drive FE		Y				Y							Y		
Individual FE						Y							Y		
Observations		5,600				5,600							5,600		

Notes: In Column (1-3) the coefficients on the individual characteristics gender, age, and blood type are not shown. Standard errors clustered at the individual and blood drive level in parentheses. ** $p < 0.05$, *** $p < 0.01$..

Appendix C Checks on weather instrument

Table C.1: Balance Checks of Covariates

	(1) No adverse weather	(2) Adverse weather	(3) Difference
Call	0.4985	0.5067	0.0083
	(0.5001)	(0.5004)	(0.0243)
Age	41.2630	41.7881	0.5250
	(13.4889)	(13.6467)	(0.6574)
Male	0.5147	0.4971	-0.0176
	(0.4999)	(0.5005)	(0.0243)
O+ blood type	0.8181	0.8439	0.0259
	(0.3859)	(0.3633)	(0.0186)
O- blood type	0.0618	0.0520	-0.0098
	(0.2409)	(0.2223)	(0.0116)
A- blood type	0.1201	0.1040	-0.0161
	(0.3252)	(0.3056)	(0.0156)
Observations	2,281	519	2,800

Notes: Means with standard deviations in parentheses. Balance checks focus on the first two periods of the experiment, as the phone call was administered during these periods (see Table 1). The variable Call indicates the frequency of the phone call.

Appendix D Replication Study

Table D.1 shows the results, analogously to Table 5. In the first stage in Column (1), both excluded instruments are strong with a joint F-statistic of 34. Compared to Table 5, the coefficients on the excluded instruments are within the confidence bounds. The estimated second stage coefficient on the past donation in Column (2) is 0.29. The p-value of the Sargan-Hansen J-test of overidentifying restrictions is 0.26, indicating that the null hypothesis that both instruments are exogenous is not rejected.

Columns (3)-(4) show the second stage estimates when excluding only one instrument at a time. Consistent with the insignificant overidentifying restrictions test the estimated effect of past donation is within the confidence bounds in both columns and both instruments are individually strong. The coefficient on past donation is even somewhat *smaller* when using the phone call compared to using adverse weather as instrument - the opposite as would be expected if the phone call had persistent effects on the motivation to donate.

Columns (5)-(6) exclude only one instrument at a time, while *including the additional instrument* as control variable. As expected, neither the lagged phone call, nor lagged adverse weather have a persistent direct effect on donations, once controlling for past donations.

Even tough the quasi-experimental data is not directly comparable to the field experiment and is considerably more noisy, qualitatively the results from this replication exercise are reassuringly consistent.

Table D.1: Action-based vs motivation-based persistence (replication study)

	(1)	(2)	(3)	(4)	(5)	(6)
Dependent variable:	Donation_{t-1}	Donation_t	Donation_t	Donation_t	Donation_t	Donation_t
$\widehat{\text{Donation}_{t-1}}$		0.288**	0.225	0.672	0.225	0.672
		(0.140)	(0.155)	(0.391)	(0.155)	(0.396)
Phone call_{t-1}	0.122***					-0.0543
	(0.0178)					(0.0532)
$\text{Adverse weather}_{t-1}$	-0.0906***				-0.0405	
	(0.0184)				(0.0328)	
Phone call_t	-0.0228	0.0626***	0.0599***	0.0785***	0.0610***	0.0712***
	(0.0198)	(0.0206)	(0.0206)	(0.0284)	(0.0205)	(0.0251)
Adverse weather_t	-0.0228	0.00201	0.000969	0.00833	-0.00416	0.00602
	(0.0252)	(0.0194)	(0.0192)	(0.0237)	(0.0195)	(0.0228)
Excluded instrument		Both	Phone call	Adverse weather	Phone call	Adverse weather
Kleibergen/Paap F-statistic		33.76	46.23	18.56	46.76	24.19
Sargan-Hansen J-test (p-val.)		0.263				
Observations	9,611	9,611	9,611	9,611	9,611	9,611

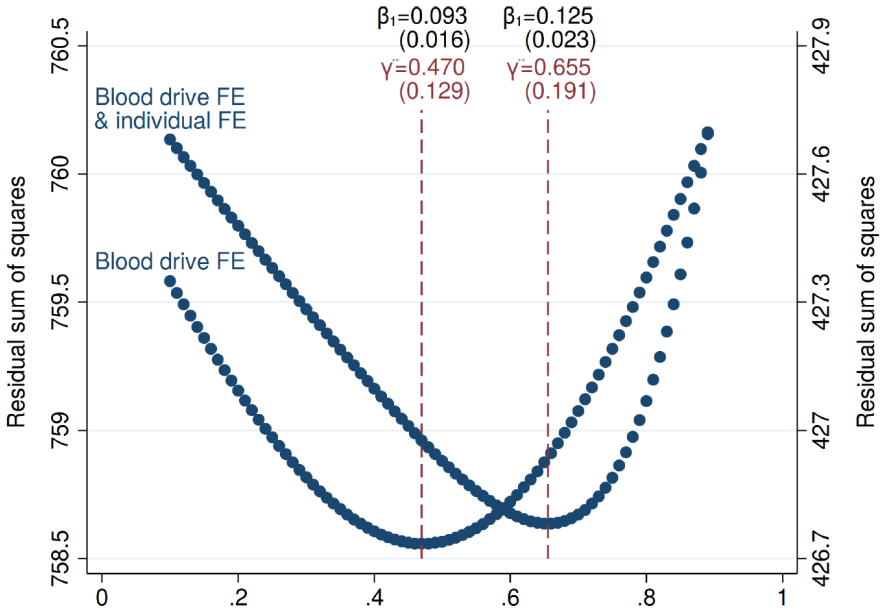
Notes: Regressions additionally include individual controls (gender, age, blood types) and week of the year and sponsor fixed effects. Standard errors clustered at the individual and blood drive level in parentheses. ** $p < 0.05$, *** $p < 0.01$

Appendix E ITT Parameter γ

Figure E.1 shows the ITT version of Figure 4 of the distribution of γ for the models with blood drive fixed effects and additionally with individual fixed effects, in line with Columns (4)-(5) in Table 4. The plot also includes the residual sum of squares to show that the parameters are well identified: in each of the specifications, there is a clear minimum.

The resulting γ parameters are 0.470 ($p < 0.01$) and 0.655 ($p < 0.01$), respectively. The estimates are very similar as in Figure 4 and clearly reject the benchmark of no action-based persistence.

Figure E.1: Grid search of the parameter γ



Notes: Relationship between γ and the residual sum of squares. Optimal γ^{**} is chosen to minimize the residual sum of squares. Two-way individual and blood drive cluster robust standard standard errors from 1,000 bootstrap replications in parentheses.

Appendix F Simulation Evidence

Figure F.1 Panel (a) shows the share of cases from 10,000 simulations with p-value of the Sargan-Hansen J-test of over-identifying restrictions being less than the observed value of 0.942 in Table 5 (experimental sample). Figure F.2 Panel (a) features an analogous plot for using the larger replication sample, with the p-value of 0.263 obtained from Table C.1. We also simulate the statistical power of the test statistic at the 5% level in the respective figures' Panel (b). The following steps outline the methodology employed in conducting these simulations.

F.1 Persistence Shifting

The persistence in the simulation were artificially distributed between action-based and motivation-based, while keeping the overall level of persistence constant. Table F.1 illustrates this main idea in Column (3), contrasting it to the case where persistence is purely action-based (Column (2)).

Table F.1: Reduce-form Persistence Effect

(1) Period	(2) Action-based persistence only	(3) α% Action + $(1-\alpha)$% Motivation-based persistence
t = 0	0	0
t = 1	$\beta_{call}\gamma$	$\alpha\beta_{call}\gamma + (1-\alpha)\beta_{call}\gamma$
t = 2	$\beta_{call}\gamma^2$	$\alpha\beta_{call}\gamma^2 + (1-\alpha)\beta_{call}\gamma^2$
t = 3	$\beta_{call}\gamma^3$	$\alpha\beta_{call}\gamma^3 + (1-\alpha)\beta_{call}\gamma^3$

In the context of Table F.1, the phone call intervention occurs in period 0. There is no persistence in this period under either scenarios, as no phone calls were made in the preceding period. In period 1, the overall level of persistence is $\beta_{call}\gamma$, where β_{call} is the effect of the phone call intervention on donation in the previous period, and γ is the persistence coefficient. $\beta_{call}\gamma$ is then divided between action-based and motivation-based persistence by ascribing α% persistence to action-based, and the remainder $(1-\alpha)$% to motivation-based. Where persistence is action-based only, $\alpha = 1$ yielding $\beta_{call}\gamma$.

Extrapolating the reasoning above into periods 2 and 3, the overall persistence levels are $(\beta_{call}\gamma)\cdot\gamma = \beta_{call}\gamma^2$ and $(\beta_{call}\gamma^2)\cdot\gamma = \beta_{call}\gamma^3$ respectively. The fraction of persistence that is action-based in period 2 is $\alpha\gamma\cdot\beta_{call}\gamma = \alpha\beta_{call}\gamma^2$ and $\alpha\gamma\cdot\beta_{call}\gamma^2 = \alpha\beta_{call}\gamma^3$ in period 3.

The balance are the motivation-based persistence in those periods i.e., $\beta_{call}\gamma^2 - \alpha\beta_{call}\gamma^2 = \beta_{call}(1-\alpha)\gamma^2$ and $\beta_{call}\gamma^3 - \alpha\beta_{call}\gamma^3 = \beta_{call}(1-\alpha)\gamma^3$. This idea of persistence-shifting is incorporated in the simulation model detailed below.

F.2 Simulation Model for Field Experiment

The first step in running the simulation is to construct a blood donation dataset that mimics that of the experimental dataset. To do so, we require a model that predicts whether an individual would make a donation, given conditions similar to that of the experiment. The model that we have formulated is:

$$u_{it} = \beta_0 + \beta_{call} \cdot P_{it} + \beta_{adv} \cdot R_{it} + \Delta B_{it} + \alpha \cdot \gamma \cdot d_{it-1} + \varepsilon_{it} \tag{F.1}$$

$$\varepsilon_{it} \sim U(0,1)$$

where:

α, β_{call}, and γ are as defined previously
β_{adv} is the adverse weather coefficient
d_{it-1} is the blood donation in the previous period
P_{it} is the indicator for phone call
R_{it} is the indicator for adverse weather
ΔB_{it} is the change in baseline motivation

The coefficients β_{call} (0.0949) and β_{adv} (–0.0724) are obtained from Table 5, while the two γ values (0.484 and 0.692) are from Section 6.3. α is chosen from 0.1 – 1.0 in increments of 0.1. R_{it} is a random binary indicator where $\mathbb{P}(R_{it} = 1) = 0.204$, mirroring the actual frequency of adverse weather.

Described in Table F.2 below, P_{it} replicates the indicator of phone call intervention from the experimental sample with 350 individuals under each condition.

Table F.2: Phone call intervention indicator, P_{it}

Group	Individuals	P_{i1}	P_{i2}	P_{i3}	P_{i4}
C1	$i \in \{1, 2 \ldots 350\}$	1	0	0	0
C2	$i \in \{351, 352 \ldots 700\}$	0	1	0	0
C12	$i \in \{701, 702 \ldots 1050\}$	1	1	0	0
Control	$i \in \{1051, 1052 \ldots 1,400\}$	0	0	0	0

Next, ΔB_{it} is defined in accordance with Table 1, while accounting for the period(s) of phone call intervention. The individual ΔB_{it} of each group over the 4 experimental periods are specified in Table F.3 below:

Table F.3: Change in baseline motivation, ΔB_{it}

Group	ΔB_{i1}	ΔB_{i2}	ΔB_{i3}	ΔB_{i4}
C1	0	$\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^2$	$\beta_{call}(1-\alpha)\gamma^3$
C2	0	0	$\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^2$
C12	0	$\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^2+$ $\beta_{call}(1-\alpha)\gamma$	$\beta_{call}(1-\alpha)\gamma^3+$ $\beta_{call}(1-\alpha)\gamma^2$
Control	0	0	0	0

F.3 Running the Simulation

The simulation commences in period $t = 1$, where β_0 is selected such that the mean donation of the control group is 0.1275 as per Figure 6. Next, for each individual i, ε_{i1} is randomly drawn from a continuous uniform distribution $U(0, 1)$. If $u_{i1} \geq \varepsilon_{i1}$, $d_{i1} = 1$, else $d_{i1} = 0$. This process is then repeated for $t = 2$, $t = 3$ and $t = 4$ to generate d_{i2}, d_{i3} and d_{i4} respectively. Thereafter, the Sargan-Hansen J-test is conducted on the simulated dataset.

The entire process is repeated 10,000 times and the share of cases with Sargan p-values less than 0.942 (or 0.263, under the larger replication sample) is computed.

F.4 Simulation for Replication Sample Study

The steps in simulating the larger replication sample is largely similar to what is described above. The only difference are:

1. Table F.2 will have 801 individuals instead of 350 in each group.
2. $\mathbb{P}(R_{it} = 1) = 0.233$ to reflect the higher adverse weather frequency in the replication sample.
3. $\beta_{call} = 0.122$ and $\beta_{adv} = -0.0906$, which are obtained from Table D.1.

F.5 Description of Utility/Cost Distribution Plots

Noting the presence of non-monotonicities in Figure F.1, particularly at $\alpha < -0.2$ and $\alpha > 1.7$, this section attempts to explain those patterns using the utility and cost distributions found in Figure F.3. We begin with a description of Figure F.3 and how it should be

interpreted in relation to the non-monotonic powers found in Figure F.1.

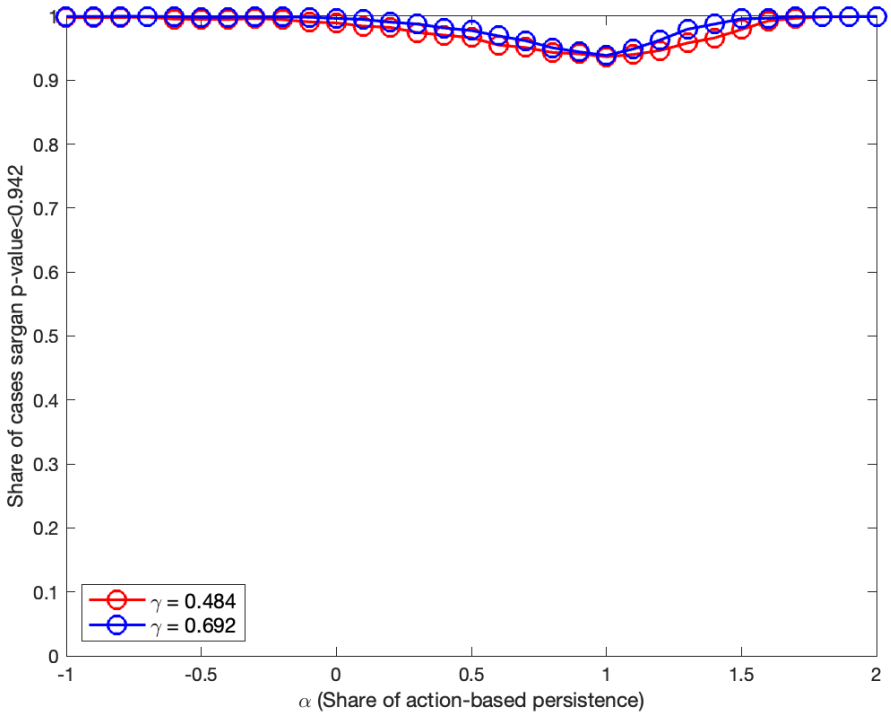
Figure F.3 comprises of 15 sub-figures, each of which takes on an alpha value between $[-1, 2)$ in increments of 0.2. Within each sub-plot, there are 4 periods: $t = 1$, $t = 2$, $t = 3$ and $t = 4$, corresponding to the 4 donation periods of the experiment. The same 3,204 individuals appear in all 4 periods. They are divided into the control group (Control) and treatment groups (C1, C2, C12) with 804 individuals respectively.

In any given period t, the light green markers simulate the individual cost, e_{it}, which is randomly drawn from the uniform distribution $U(0, 1)$. The dark green markers simulate the individual's utility, u_{it}, which is computed using Equation (F.1). Note that the number of unique dark green lines in each period corresponds to the number of unique utility values that can be obtained. This is in turn dependent on the number of possible combinations of R_{it} and P_{it}. Taking Figure 3(a) as an example, in period one $|R_{i,1}| = 2$ and $|P_{i,1}| = 2$, therefore there would be $2 \times 2 = 4$ dark green lines. For period 2, $|R_{i,1}| = 2$, $|P_{i,1}| = 2$, $|R_{i,2}| = 2$ and $|P_{i,2}| = 2$, giving $2 \times 2 \times 2 \times 2 = 16$ lines. Using a similar argument, period 3 and 4 will have 32 and 64 lines respectively.

Next, on how Figure F.3 explains the non-monotonicities in power: first, we discern that the dark green markers may be interpreted as the probability that an individual will make a donation. Next, by scrutinizing the shifting patterns of the green markers as α changes, we find that for the figures with $\alpha \leq -0.6$ and $\alpha \geq 1.2$, there are individuals that always donate or never donate regardless of the cost they incur. The implication of this is that the sample size is effectively reduced, which would in turn decreases the power of the Sargan test, resulting in the non-monotonic patterns observed in Figure F.1.

Figure F.1: Field Experiment Sample

(a) Share of cases with Sargan p-value<0.942



(b) Share of cases with Sargan p-value<0.05

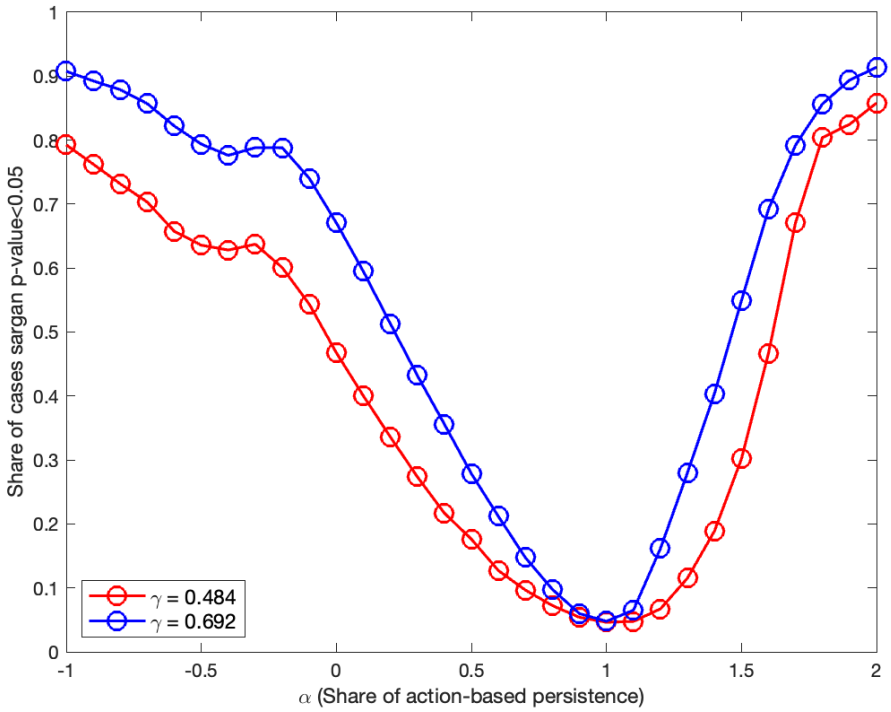
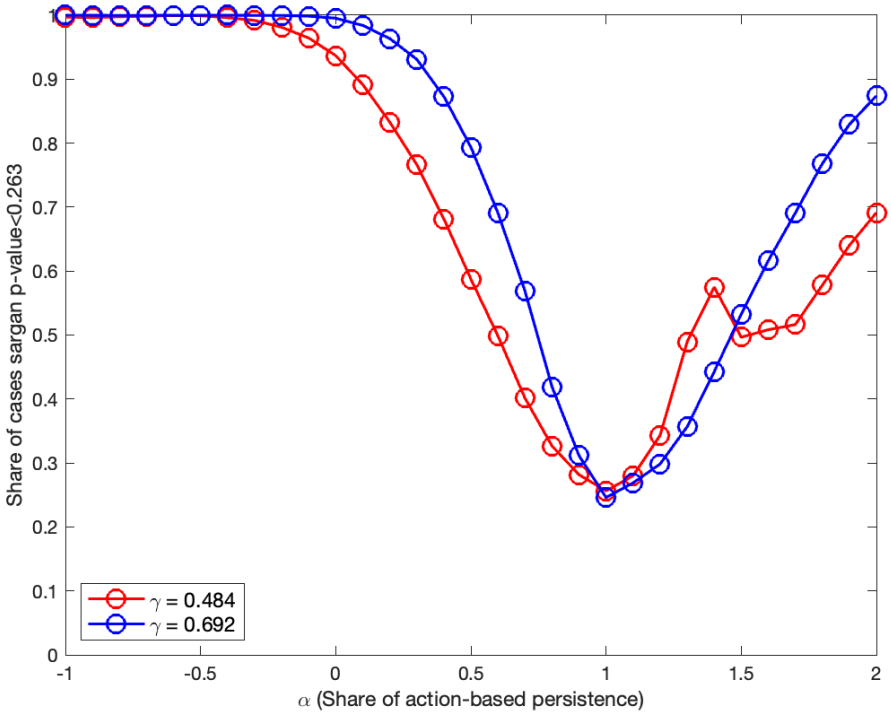


Figure F.2: Replication Sample

(a) Share of cases with Sargan p-value<0.263



(b) Share of cases with Sargan p-value<0.05

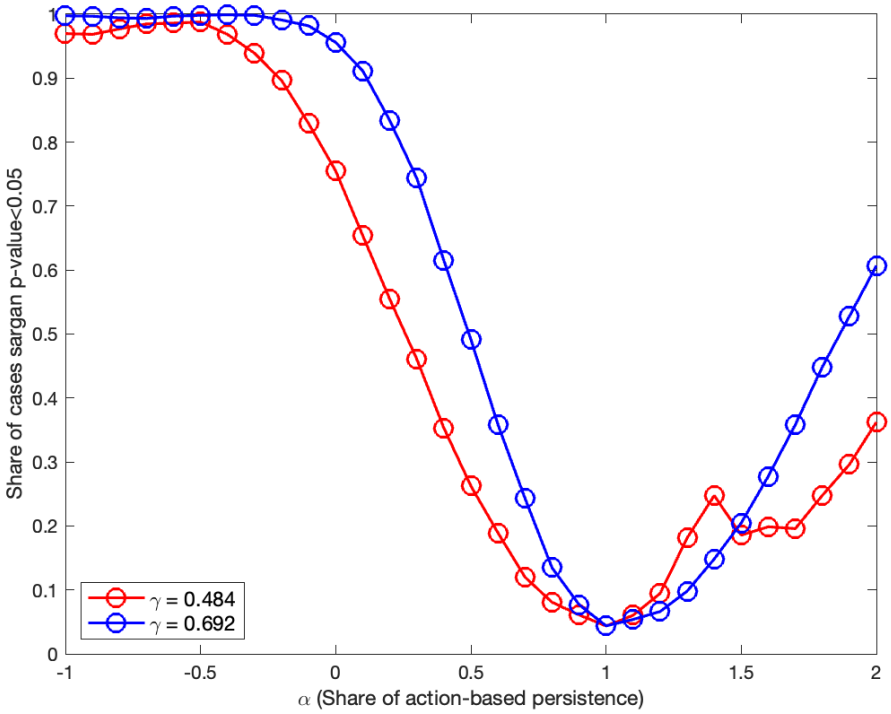
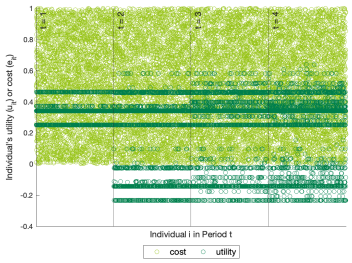
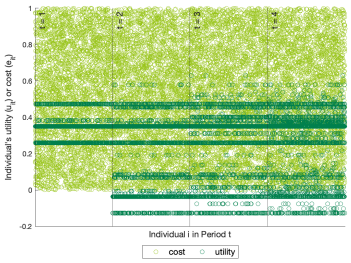


Figure F.3: Utility/Cost Distribution

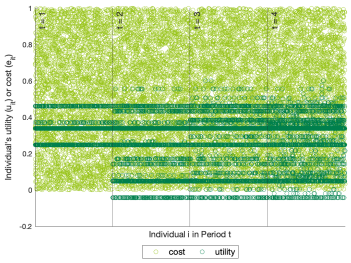
(a) $\alpha = -1.0$



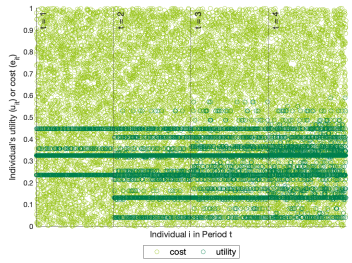
(b) $\alpha = -0.8$



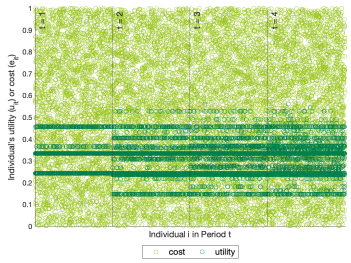
(c) $\alpha = -0.6$



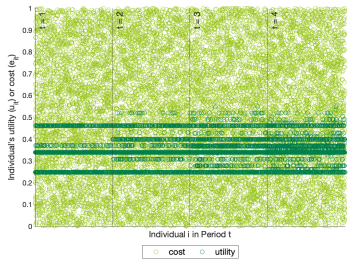
(d) $\alpha = -0.4$



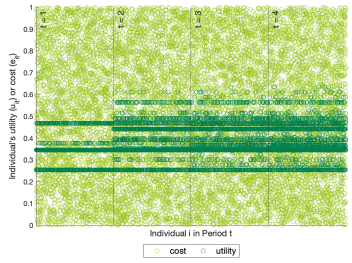
(e) $\alpha = -0.2$



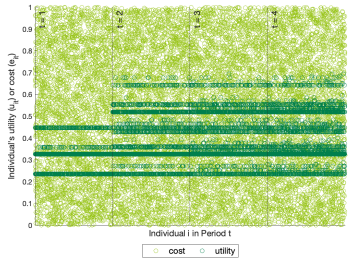
(f) $\alpha = 0.0$



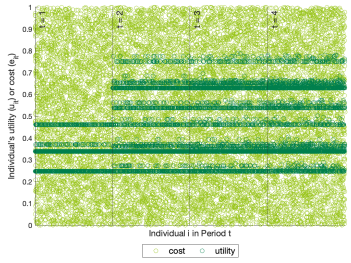
(g) $\alpha = 0.2$



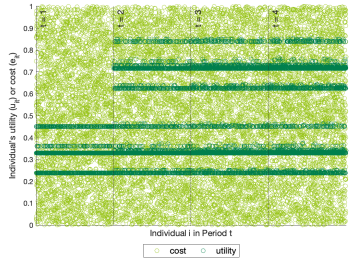
(h) $\alpha = 0.4$



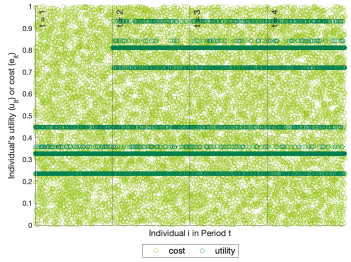
(i) $\alpha = 0.6$



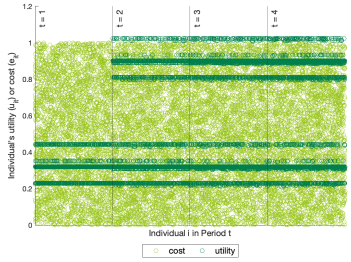
(j) $\alpha = 0.8$



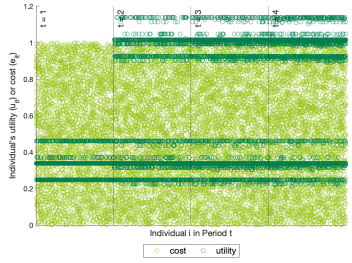
(k) $\alpha = 1.0$



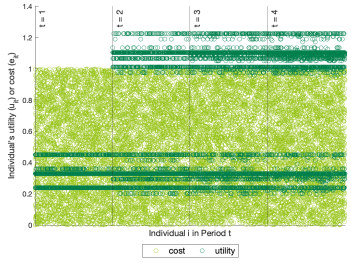
(l) $\alpha = 1.2$



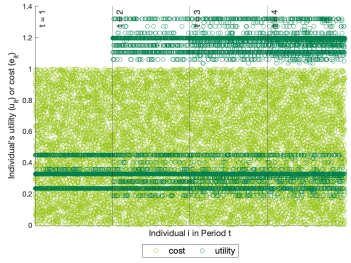
(m) $\alpha = 1.4$



(n) $\alpha = 1.6$



(o) $\alpha = 1.8$



F.6 Setup of Persistence Example

In this section, we simulate the persistency in blood donors with cyclical change in baseline motivation. Our simulation is based on the assumption that there are 2 types of donors, summertime donors and wintertime donors.

We simulate the persistence of donors across 4 periods, t_1, t_2, t_3 and t_4, over a span of 2 years. We define t_1 and t_3 to be summertime, whilst t_2 and t_4 to be wintertime. To model the seasonal preference in donating blood of the respective donor types, we incorporated asymmetric cyclicality in the donor's baseline motivation, as illustrated in Table F.4.

Table F.4: Cyclical Change in Baseline Motivation

	Baseline Motivation			
Donor Type	$t = 1$	$t = 2$	$t = 3$	$t = 4$
Summer	$B + \delta B$	$B - \delta B - \eta$	$B + \delta B$	$B - \delta B - \eta$
Winter	$B - \delta B - \eta$	$B + \delta B$	$B - \delta B - \eta$	$B + \delta B$

In the absence of seasonality, both types of donors possess a baseline motivation of B. During summer, i.e., $t = 1$ and $t = 3$, the increase in motivation for summer donors to donate is equal to δB, while their demotivated winter counterparts' motivation falls by a slightly larger magnitude of $\delta B + \eta$. This change in baseline motivation reverses during wintertime, i.e., $t = 2$ and $t = 4$.

With these baseline motivations along with Equation (F.1), we simulate the donor's persistence in the simplest case by setting $\delta = 1$ while also assuming no motivation-based persistence by setting $\alpha = 1$, which yields the following:

$$u_{it} = \beta_0 + \beta_{call} \cdot P_{it} + \beta_{adv} \cdot R_{it} + B + I_{on} \cdot B - I_{off} \cdot (B + \eta) + \gamma \cdot d_{it-1} + \varepsilon_{it} \tag{F.2}$$

$$\varepsilon_{it} \sim U(0, 1)$$

where:

β_{call}, β_{adv}, d_{it-1}, P_{it}, R_{it}, and γ are as defined previously

I_{on} is an indicator of *"on-cycle"* donation

e.g. summer donors during summertime, vice versa

I_{off} is an indicator of *"off-cycle"* donation

e.g. summer donors during wintertime, vice versa

The results of the simulation are plotted in Figure F.4. The circle markers are the donation rates conditional on the donors having donated in period 1 (red) or period 2 (blue), while the crosses are conditional on the donors having not donated in period 1 (red) or period 2 (blue).

Figure F.4: Persistency in Donors with Cyclical Change in Baseline Motivation