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SOCIAL CONTAGION OF FERTILITY: AN AGENT-BASED SIMULATION STUDY

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ABSTRACT

Social contagion is a process in which the behavior of a social group spreads across a population. We model and analyze this phenomenon with respect to fertility in an agent-based social simulation study. Our model integrates personal motivations for having children and social influences to allow for quantifying their respective impact on fertile behavior. We use a real-world dataset for model calibration and compare three hypothetical scenarios with the reference setting. Our results show that each of the social influences has a statistically significant impact on birth rates in the agent population. Moreover, we identify differences in the effects of social pressure and social support depending on a person's age. This shows that our simulation is not only capable of realistically reflecting family development, but can also provide further insights into the functioning of social mechanisms in a controlled experiment setting.

1 INTRODUCTION

Fertility is contagious (Lois and Becker 2014). Not only individual preferences but also social influences play a major role for partners in a relationship when deciding whether or not to have children. The latter depend on a couple's social context which it shares with others. Thus, people mutually influence each other in their decisions. This leads to similar decisions within groups of people who share the same context, i.e., who have overlapping social networks. That phenomenon is known as social contagion (Levy and Nail 1993). It can be observed in various situations, e.g., information spread and opinion formation, voting behavior, marriage decisions, and, in fact, fertile behavior (Iyengar et al. 2011; Braha and De Aguiar 2017).

Social contagion is an empirically well-studied phenomenon (Levy and Nail 1993; Lois and Becker 2014). These empirical studies are descriptive in character. They focus on situations as they appear in reality and identify correlations between different aspects of these situations, e.g., birth rates and perceived social influence. Agent-based social simulation can complement these findings by modeling individual persons as agents for experimenting with different decision contexts and mechanisms (Davidsson 2002). This is especially useful in the presence of several social mechanisms which produce a phenomenon together. In such a situation, social simulation allows for analyzing hypothetical scenarios that remove one or more of these mechanisms or place agents in different social contexts. These studies can then be used to assist sociological theory development or to forecast effects of public policy (Malsch 2001).

In this paper, we present an agent-based simulation study on social contagion of fertility. The goal of that study is to quantify the impact of personal motivations and social influences on fertile behavior. To that end, we identify the individual *value of children* as well as *social pressure* and *social support* as relevant decision-making factors from sociological foundations and discuss related approaches to model social contagion in Sec. 2. Subsequently, we present a novel and extensible agent decision-making algorithm based on these foundations in Sec. 3. In contrast to existing simulation models of social contagion, our approach integrates individual as well as social influence factors to allow for comparing their respective

impacts on fertility. We calibrate our model using the German Family Panel *pairfam* as a real-world reference dataset in Sec. 4. In our simulation study, we show that each of the social influences has a statistically significant impact on fertility rates in the agent population. Moreover, we quantify that impact to provide insights into the importance of the social context on fertile behavior. Finally, Sec. 5 provides a concluding summary of these findings.

2 SOCIAL MECHANISMS OF CONTAGION

Social contagion is a process in which a certain behavior or property of a social group spreads across a population. Much like the spreading of a disease, this effect on a macro-social level is the result of micro-social interactions. People interact with each other in social networks of friendship and family relations. They influence each other by encouraging or enforcing socially accepted behaviors. These influencing factors are known as social mechanisms (Hedström and Ylikoski 2010). The following sections introduce relevant mechanisms for social contagion of fertility and discuss related approaches to model them for computer simulation studies.

2.1 Social Contagion of Fertility

There are numerous consideration factors for couples when deciding whether to have (additional) children. They can be subdivided into mainly *intrinsic motivations* and predominantly *extrinsic influences*. Intrinsic motivations depend on a person's age, their already existing number of children, their economic situation, their socialization, and other individual properties. By contrast, extrinsic influences originate from a person's social context, particularly from their parents and friends. If a couple is situated in a social network that values children very highly, their own motivations for having children can become amplified by influences from that network. In that case, social influences emphasize positive aspects of family life and understate negative consequences. Vice versa, negative opinions toward children within a social network can dampen individual motivations for having children by accentuating disadvantageous implications. As a couples' own opinion feeds back into the network, this leads to reciprocal influences that either support or discourage fertile behavior. Thus, fertility is (partially) an effect of a social contagion process (Lois and Becker 2014).

To describe those influences on fertility, individual motivations can be subsumed under the *Value of Children* (VoC) to potential parents (Hoffmann 1975). This concept covers personal expectations and concerns toward children with respect to subjective well-being as well as social recognition (Ormel et al. 1999). These decision factors are often culturally determined and result from a specific socialization. Thus, while they can be exaggerated or downplayed in different social contexts, they remain comparatively stable for each individual. For instance, religious moral and social norms are not easily changed. Also, in some cultures, children are a factor of power and influence that is hard to neglect. Hence, children can provide identity and give their parents a competitive advantage in social comparisons. On the other hand, they can also be perceived to delimit the parents' freedom as well as to induce financial costs and risks. The concept of VoC describes these competing influence factors on a person's decision-making (Hoffmann 1975).

Contrastingly, extrinsic influences are context-specific for the social network a couple is situated in. They can be further subdivided into two mechanisms out of which the first one is *social pressure*. That mechanism describes normative pressure a person receives from their immediate social environment. This pressure results from anticipated rewards for complying with social expectations (e.g., of having children at a specific age) and from potential sanctions for deviating from them (Bernardi 2003). These rewards and sanctions do not need to be uttered explicitly; mere comparisons within a group can lead to an urge for reducing cognitive dissonance between of a person's own opinion and that of the majority (Festinger 1954). Such a reduction can be achieved by individually adopting the socially desired behavior or by changing the target group of that comparison. Therefore, social pressure results both in a person's perceived obligation to comply with others' expectations (if physically possible) and to the formation of homophilic social network compositions in which individual attitudes and the resulting behavioral intentions are similar (Ajzen 1991).

The second mechanism of extrinsic influences is *social support*. It covers the availability of measures to counterbalance perceived limitations and costs that result from having children. A person can gain social capital in their social network by supporting others (Bourdieu 1985). In turn, that capital may be utilized in the form of financial, instrumental, or emotional support from others in the network (particularly within families; i.e., from a person's parents). The amount of support depends on the network composition which can vary between traditional and individualized ones. The former tend to be more homogeneous and supportive than the latter (Keim 2011). Consequently, that composition of the social context plays an important role in a person's perceived ability to mitigate potential disadvantages related to having children.

The aforementioned mechanisms of social contagion have been empirically analyzed using panel data (Lois and Becker 2014; Richter 2015). For such a purpose, the German Family Panel *pairfam* (Panel Analysis of Intimate Relationships and Family Dynamics) provides a longitudinal study on partnership and family dynamics of more than 12,000 persons (Huinink et al. 2011). This dataset comprises *anchor persons* from three birth cohorts (1971-73, 1981-83, 1991-93) who have been questioned in annual waves since 2008 (the latest release includes nine waves). This makes it ideal for identifying correlations between fertile behavior and social influences (Lois and Becker 2014). However, empirical analyses can only provide insights into social contagion of fertility as it is reported in the data. Computer simulation can complement these studies by providing a controlled test-bed for theories of social contagion in which the impact of particular mechanisms can be compared in different experiment settings. The *pairfam* dataset provides a real-world reference for calibrating social simulation models and for evaluating their ability to produce realistic results. Thus, we use it for that purpose in this paper.

2.2 Related Work: Models of Social Contagion Mechanisms

Contagion processes have been frequently simulated using agent-based models. Agent-based approaches allow for modeling interactions of individuals that lead to a spreading of the represented phenomenon across a population. In this context, the so-called SIR and SIS models have been applied for simulating biological as well as social contagion processes (Dodds and Watts 2005). These models differentiate between three possible states for each agent: *susceptible* agents (S) can become infected, *infected* agents (I) can infect susceptible ones, and infected agents will eventually *recover* (R), so they will no longer infect others. Transitions between the states are modeled by means of probabilities or thresholds representing a minimum number of contacts between susceptible and infected agents for the infection to spread.

SIR models are well-suited for simulating contagion phenomena in which different states of activity can be clearly distinguished. However, this is not the case for social contagion of fertility. Even network members without children can put social pressure on others or offer social support to them. In addition, the VoC represents processes of "self-infection"; i.e., individual decisions to have a child without any influence from other agents or even against their opposition. These effects are better represented by sociologically inspired agent decision-making mechanisms developed in distributed artificial intelligence, namely normative multiagent systems and approaches to reciprocal exchanges between agents.

Normative multiagent systems control the behavior of their member agents by means of rewards and sanctions for norm-compliant or deviating activities (Savarimuthu et al. 2009). This can even lead to agents acting against their own motivations due to normative pressure (Centola et al. 2005; Dechesne et al. 2013). Hence, these approaches incorporate interactions of intrinsic and extrinsic influences similar to the interplay between VoC and social pressure. To that end, agents have first to become aware of that pressure, for which a filter function can be used to model selective norm perception (Hashimoto and Egashira 2001). Subsequently, an agent must deliberate between individual desires and emotions as well as social goals and obligations to decide for appropriate action in a given situation (López y López et al. 2002). By these means, agents can decide to adopt accepted behaviors from their social network environment. They can also attempt to change the opinions of others or exclude them from their network (De Pinninck et al. 2010).

While normative multiagent systems aim at enforcing desirable behaviors, there are also approaches to model social capital among agents which encourage reciprocally supportive action. These agents accumulate

trust within a social network by supporting other agents and they receive support according to the trust others put in them (Sutcliffe et al. 2015; Wang et al. 2016). This leads to stable relationships where agents either trust or distrust each other. These relationships reflect the differences between traditional and individualized social networks where the strength of bonds between agents determines the amount of support they can receive (Lin and Zhang 2017). While these networks lack representations of different relationship types (i.e., family bonds or friendships), methods for generating trust are well-suited to model and explain their emergence in social simulations.

Nonetheless, incorporating intrinsic motivations and extrinsic influences in an agent-based model requires an agent architecture that can represent these decision-making factors. Agents must be capable of reasoning about their current situation, their own motivations and goals, as well as the influences they receive from others. To that end, there are various decision-making methods available for social simulations (Balke and Gilbert 2014). In particular, the *Belief-Desire-Intention* (BDI) model of practical reasoning (Rao and Georgeff 1995) provides a good starting point for modeling decisions in the context of fertility. It organizes individual goals (desires), situation-related information (beliefs), and action-oriented measures (intentions) into the respective mental states. In addition, it provides a decision-making algorithm (deliberation) that generates intentions from desires and beliefs. This makes it particularly suitable for integrating the mechanisms of VoC, social pressure, and social support into an extensible agent-based model of social contagion as we demonstrate in the following.

3 AN AGENT-BASED MODEL OF SOCIAL CONTAGION

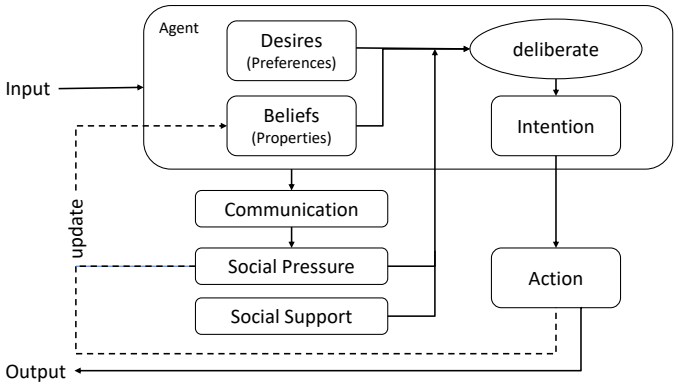
To simulate social contagion of fertility, we consider each couple to be represented as an agent. Since we are interested in the interplay of individual preferences and influences from social networks, we abstract from interactions between partners in a relationship. This is in line with our reference dataset which focuses on individual *anchor persons*. These persons have individual properties like their age and number of children as well as preferences such as expectations and concerns about having children. Our agent-based model must be capable of representing these and provide an algorithm which transfers them into a decision for or against having an (additional) child. Moreover, that decision has to factor in extrinsic influences from an agent's simulated social network.

To that end, our agent architecture is based on a simplified variant of the aforementioned BDI mechanism for practical reasoning (Rao and Georgeff 1995) as depicted in Fig. 1. Each agent has three sets of mental states: The beliefs (B) contain dynamically changing information about its current state and any extrinsic influences on its decisions. The desires (D) contain static information about its personal preferences. From these two sets, an agent derives the current intention (I) in a decision-making process called *deliberation* (Eqn. 1). With respect to fertility, an agent either intends to have another child or not. Subsequently, it performs an *action* as the observable result of its decision. Because the mere intention of having a child does not necessarily result in actually having one, an agent selects its action stochastically, depending on its modeled current $age \in \mathbb{N}$ and existing number of children $numChildren \in \mathbb{N}$ (Eqn. 2). Those variables are part of the agent's beliefs whereas the probability distribution for $P(child|age, numChildren)$ is empirically derived from our dataset (cf. Sec. 4).

$$deliberate : \{B\} \times \{D\} \rightarrow \{I\} \qquad \text{where} \qquad I \in \{child, \neg child\} \tag{1}$$

$$P(action = child) = \begin{cases} P(child|age, numChildren) & \text{if } I = child \\ 0 & \text{otherwise} \end{cases} \tag{2}$$

To represent an agent's personal preferences, we model the value of children $VoC \in [-1, 1]$ as one of its desires which can range from entirely child-averse (-1) to completely child-oriented (1). Without any extrinsic influences, this value would be the sole driving force for generating the agent's intention. However, as Fig. 1 shows, agents also interact with each other, resulting in their perception of social support



Algorithm: **BDI Control Cycle**

```
1   for each agent
2      B := initializeBeliefs(input)
3      D := initializeDesires(input)
4      I := {}
5      action := none
6      initializeSocialNetwork(input)
7   end for
8   repeat
9      for each agent
10        s := perceiveSupport()
11        p := communicatePressure()
12        B := updateBeliefs(B,s,p,action)
13        I := deliberate(B,D)
14        action := selectAction (I)
15        perform(action)
16     end for
17  end repeat
```

Figure 1: Agent decision-making architecture (left) and algorithm (right).

and social pressure. The former is a belief $s \in [0,1]$ about the amount of expected support, ranging from none (0) to full (1). The higher this perceived support, the more likely an agent will consider having a child. Moreover, social pressure is a belief $p \in \{-1,1\}$ which models the predominant normative opinion in an agent's social network either for (1) or against (-1) having children. Whether an agent is susceptible to comply with that norm depends on its emotional autonomy $EA \in [0,1]$. Similar to the work by López y López et al. (2002), a high *EA* enables an agent to resist normative pressure by others. Again, an agent's initial beliefs and its desires as summarized in Eqn. 3 are derived from empirical data (cf. Sec. 4).

Each time an agent makes a decision, it first updates its beliefs about extrinsic influences. The belief s is updated by perceiving the currently expectable amount of support directly derived from the *pairfam* dataset. By contrast, the social pressure p depends on its interaction with other agents. Each agent has an individual number n of other agents that form its social network. Being a subset of the set of all agents, this results in a sparsely connected graph of influence relations. For each neighboring agent j in that graph, an agent perceives their current pressure p_j for or against children and updates its own normative belief accordingly. If the mean social pressure exceeds the strength of an agent's own opinion, it changes its belief p as shown in Eqn. 4 which has been adopted from Centola et al. (2005).

$$B = \{age, numChildren, p, s\} \qquad \text{and} \qquad D = \{VoC, EA\} \tag{3}$$

$$p \leftarrow \begin{cases} 1 & \text{if } pressure > 0 \wedge |VoC| < |pressure| \\ -1 & \text{if } pressure < 0 \wedge |VoC| < |pressure| \\ p & \text{otherwise} \end{cases} \qquad \text{with} \qquad pressure = \frac{1}{n}\sum_{j=1}^{n} p_j \tag{4}$$

Using its updated beliefs and its desires as input, an agent then deliberates about its next intention. Combining its intrinsic motivation with extrinsic influences, there are six cases in which the agent will intend to have an (additional) child as shown in Eqn. 5. If its VoC is at the maximum, the agent will always intend to have a child without further consideration (a). If that value is in the center (i.e., the agent is indifferent about children), it will comply with the group opinion of its social network (b). Otherwise, the agent is either positively or negatively undecided concerning children. Thus, it must make up its mind and decide whether it is *willing* to have a child. The value of this stochastic variable is renewed for each decision according to an empirically determined probability distribution $P(child|VoC)$. The value *weightVoC* scales the impact of the agent's personal desire on its decision. If the agent is willing, it will intend to have a child as long as its social network either has a positive opinion toward children or the agent's emotional autonomy is high enough to resist child-averse social pressure (c). To determine the

latter, there is a threshold *thldEA* for the minimal required *EA* for resistance. Hence, an unwilling agent can be convinced by others to have children if its *EA* is low (d). Nonetheless, the reverse case is also possible: If an agent is willing to have children but successfully pressured against that decision, it requires social support as an additional incentive to counterbalance that pressure (e). To that end, the agent compares the offered support with another threshold *thldS* which denotes the minimal amount of support required. Finally, an agent can be unwilling and resist pressure toward having children (or perceive pressure against it). Then, social support can also act as an incentive for intending to have a child (f).

$$deliberate(B,D) = \begin{cases} I \leftarrow child & \text{if a) } VoC = 1 \\ & \quad \text{b) } VoC = 0 \wedge p = 1 \\ & \quad \text{c) } VoC > 0 \wedge willing \wedge (p = 1 \vee EA \geq thldEA) \\ & \quad \text{d) } VoC > -1 \wedge \neg willing \wedge p = 1 \wedge EA < thldEA \\ & \quad \text{e) } VoC > 0 \wedge willing \wedge p = -1 \wedge EA < thldEA \wedge supported \\ & \quad \text{f) } VoC > 0 \wedge \neg willing \wedge (p = -1 \vee EA \geq thldEA) \wedge supported \\ I \leftarrow \neg child & \text{otherwise} \end{cases} \tag{5}$$

$$\text{with} \quad P(willing) = P(child|weightVoC \times VoC) \quad \text{and} \quad supported \Leftrightarrow s \geq thldS$$

As described above, each agent's initial beliefs and desires are derived from the *pairfam* dataset. Thus, every agent in our simulation represents a specific real couple with their individual opinions, preferences, and concerns about family life and children. In addition, the probability distributions $P(child|age, numChildren)$ and $P(child|VoC)$ represent the entire population contained in the data. They are the same for all agents. Furthermore, the parameters *weightVoC*, *thldEA*, and *thldS* are also uniform across the entire agent population. However, these are variables specific to our model which control the agents' decision-making algorithm. Since there is no empirical data for them, it is necessary to calibrate them to a combination of values that accurately represents fertile behavior of humans. Subsequently, the agent-based model can be used to dynamically analyze the impact of social contagion mechanisms on fertility as Sec. 4 demonstrates.

4 SIMULATING SOCIAL CONTAGION OF FERTILITY: EXPERIMENTS AND RESULTS

In this section we evaluate the agent-based social contagion model in a hypothesis-driven simulation study (Lorig et al. 2017). Firstly, we define the simulation setup as well as the model input and calibration using the panel study *pairfam*. Subsequently, we explain the experimental design and introduce three hypotheses to be tested. Finally, we present the findings and a discussion on the importance of different variables to quantify the impact of social contagion on fertility in our model.

4.1 Simulation Setup and Calibration

The goal of our simulation study is to quantify the impact of different social contagion mechanisms in the agent-based model on fertility. To evaluate the conceptual model according to that goal, it was implemented in Repast Simphony (North et al. 2013). From the *pairfam* dataset, we identified 2333 persons that are relevant for our study. In a simulation run, the $m = 2333$ agents perform fertile behavior based upon their own *VoC* and the social network influences throughout 7 steps, whereas each step represents a wave of the panel study lasting one year. Because the model uses random number generators, each of the following settings, including model calibration, is repeated 50 times.

For each agent, the model input consists of its *beliefs* and *desires*. In the *pairfam* study, a person's individual *age* is in the range of $[24,..,45]$ and their *numChildren* in $[0,..,10]$. We take these values directly from the dataset. For the initially perceived social pressure p and the social support s an agent can obtain, we aggregate several related answers to the *pairfam* questionnaire in a combined value. The same holds

for the desires *VoC* and *EA*. For that aggregation we take the arithmetic mean of all relevant answers (from a five-item scale) and normalize it to the respective value range of the input variables. Moreover, the social network of influence relations (i.e., close friendships) is randomly generated by connecting each agent with a number of others as provided in the dataset. The mean number of friends n an agent is connected to is about three. Finally, we obtain the probability distributions $P(child|age, numChildren)$ and $P(child|VoC)$ by counting the ratios of the respective events in the dataset for the entire population.

In addition to the empirical inputs, the impact of social contagion in the model is controlled by the variables *weightVoC*, *thldEA*, and *thldS*. Since there is no data directly available for these variables, they must be calibrated to simulate realistic birth rates. To that end, we test all combinations of their values in a full factorial design (varied in steps of 0.1) and identify the optimal setting. That optimum approximates real fertility most accurately, i.e., it minimize the distance *totalDist* between the overall numbers of children in the simulation nC_{sim} and the dataset nC_{real} in wave w across all 50 replications (Eqn. 6).

$$totalDist = \sum_{w=1}^{8} dist_w \qquad \text{with} \qquad dist_w = \sum_{r=1}^{50} |nC_{real,r}(w) - nC_{sim,r}(w)| \tag{6}$$

Fig. 2 shows the calibration results as cut-planes through the three-dimensional parameter space. That is, for each varied parameter, the other two are kept at their identified value for the overall optimum. The figure demonstrates that the model is capable of simulating realistic fertility under specific conditions. However, each social mechanism has a potentially large impact on these birth rates. In particular, the relationship between *weightVoC* and the simulation output is largely linear. While low values lead to underrated fertility in the model, the distance to real-world data reduces as the value of *weightVoC* is increased to reach its minimum at *weightVoC* = 0.7. For higher values, the model overestimates fertility.

Varying *thldEA* leads to a different output behavior. Within the value range of 0.2 to 0.5 the curve rapidly drops. Subsequently, the distance slowly decreases to its minimum value at 0.8 when it starts to increase again slightly. This shows that emotional autonomy must be comparatively high to resist the influence of social pressure. Agents in our simulation with low to average *EA* must be prone to be influenced by others in order to obtain realistic birth rates.

A similar effect is observable for social support. Manipulating *thldS* leads to a relatively small decrease in the distance of the simulated data to the real data in a range from 0.1 to 0.6. Followed by this, the curve drops fast from approximately 3500 ($thldS = 0.6$) to under 500 at value 0.9 of *thldS*. The minimal distance is found at the extreme value of $thldS = 1.0$. Consequently, social support amplifies or counterbalances social pressure and *VoC* most effectively when it is perceived as strongly as possible.

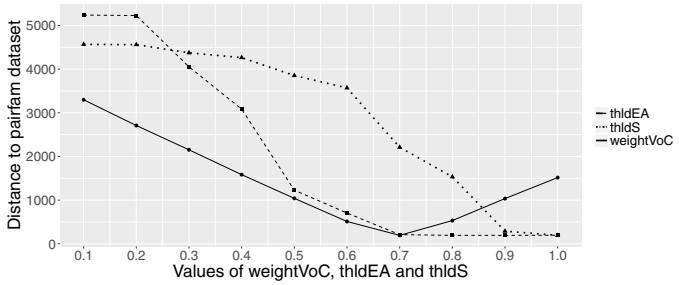


Figure 2: Calibration of the variables *weightVoC*, *thldEA* and *thldS*.

4.2 Hypotheses and Experiment Design

To evaluate the agent-based model, we define three hypotheses for comparing different scenarios of social contagion with each other. These hypotheses cover the impact of each social mechanism as well as their combination on fertility, namely the influence of *weightVoC*, *thldEA*, and *thldS*. In the calibrated setting, all social mechanisms are active which represents real situations of social contagion. To test and quantify their respective impacts, we compare that full scenario with modified ones in which one or two mechanisms are deactivated in the model. This results in the following four distinct simulation runs.

1. Full scenario with VoC, social pressure and social support
2. Only VoC and social pressure; without social support
3. Only VoC and social support; without social pressure
4. Only VoC; without social pressure and social support

Using the hypothesis definition language FITS (Lorig et al. 2017), run 1 is described by the full parameter set $ParSet1(weightVoC(0.7) \wedge thldEA(0.8) \wedge thldS(1.0))$. We compare that setting with the others and hypothesize on their differences with respect to the aggregated number of children of all $m = 2333$ agents as follows.

$$ParSet1(thldS(1.0)) \wedge ParSet2(thldS(-)) \wedge \#$$
$$\Rightarrow \mu_1(\sum_{i=1}^{m} numChildren_i) \wedge \mu_2(\sum_{i=1}^{m} numChildren_i) \wedge (H_{0_{Support}}(\mu_1 \leq \mu_2) \vee H_{1_{Support}}(\mu_1 > \mu_2))$$

$$ParSet1(thldEA(0.8)) \wedge ParSet2(thldEA(-)) \wedge \#$$
$$\Rightarrow \mu_1(\sum_{i=1}^{m} numChildren_i) \wedge \mu_2(\sum_{i=1}^{m} numChildren_i) \wedge (H_{0_{Pressure}}(\mu_1 \leq \mu_2) \vee H_{1_{Pressure}}(\mu_1 > \mu_2))$$

$$ParSet1(thldEA(0.8) \wedge thldS(1.0)) \wedge ParSet2(thldEA(-) \wedge thldS(-)) \wedge \#$$
$$\Rightarrow \mu_1(\sum_{i=1}^{m} numChildren_i) \wedge \mu_2(\sum_{i=1}^{m} numChildren_i) \wedge (H_{0_{VoC}}(\mu_1 \leq \mu_2) \vee H_{1_{VoC}}(\mu_1 > \mu_2))$$

Null hypothesis $H_{0_{Support}}$ states that by removing social support (*thldS*) the mean number of children will not decrease. $H_{0_{Pressure}}$ claims that neglecting social pressure (*thldEA*) does not lead to a decrease in the mean numer of children. $H_{0_{VoC}}$ states that by removing both social pressure and social support, the mean number of children will not decrease. Contrastingly, the respective alternative hypotheses state that social pressure and support on their own as well as in combination do have a significant supportive impact on fertility. To test the importance and impact of the different variables and to accept or decline the hypotheses, our four experimental settings are tested in the following section.

4.3 Simulation Results and Discussion

Fig. 3 depicts the results of all four experiment runs. The left diagram shows the mean number of children per simulated wave compared to the real-world data. The error bars represent the standard deviation between experiment repetitions. That deviation varies in a range of about 13 in wave two and 32 in wave eight. The line plot to the right shows the relative developments of fertility by depicting the mean differences in the number of children between each experiment setting and the *pairfam* data over all eight waves. In all experiment runs, the simulation approximates the original birth rates. For the full setting of social contagion mechanisms (run 1), the total distance in the mean number of children between simulation and real-world data across all waves is 143 with the last wave having the maximal distance of 44. The simulation shows

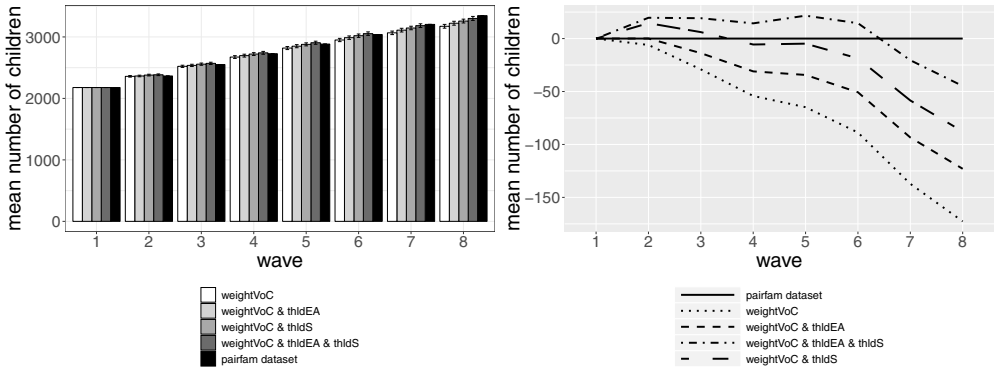


Figure 3: Number of children per wave (left) and relative differences between simulation settings (right).

a slight overproduction of children in the first six waves. This relationship reverses in the last two waves. This behavior can be explained by the fact, that the agents' age increases throughout the simulation. The possibiliy to have a first or additional child decreases with a growing age. The growth rate of the number of children thus decreases, too. At the transition between wave one and two the growth rate is at about 9.5 %. From wave seven to eight this rate only reaches a value of approximately 3.7 %. In the *pairfam* data set the growth rate between wave one and two lies at 8.6 %, at the transition from wave seven to eight this rate still takes a value of 4.4 %. Thus, the simulation slightly overestimates the importance of age which can be corrected by tweaking the probability distribution $P(child|age, numChildren)$.

When removing social support (run 2), the growth rate from wave one to two takes the same value of 8.6 % as in the real-world data. This rate then drops to a value of 3.6 % at the intersection of wave seven to eight, which is 1.3 percentage points below the *pairfam* data. In fact, the mean number of children in the simulation output consistently lies below the original data. The maximal distance is in wave eight with a value of 123 children. The total aggregated distance to the data set throughout the eight waves is approximately 346. This is a difference of 203 births less than in run 1. As Tab. 1 shows, that difference is statistically significant which leads to a rejection of hypothesis $H_{0_{Support}}$. That is, the presence of social support significantly increases fertility.

In the absence of social pressure (run 3), the growth rate starts at 9.3 % and decreases to a value of 3.5 % from wave seven to eight. The aggregated distance to the original data over all waves is 196, whereas the maximal distance of 87 children is present in the last wave. Consequently, social pressure is less effective than social support in earlier waves and gains a similar impact later. In other words, young persons are more reliant on support and with increasing age, social pressure takes effect. This shows that the type of social networks in our model changes over time. In wave one, most networks tend to be individualized due to their random generation. Over the course of time, these networks homogenize as particular norms become predominant. This reflects the process of re-traditionalization in social networks that is known to have an impact on fertility (Keim 2011). As Tab. 1 depicts, this effect is statistically significant in our model (i.e., null hypothesis $H_{0_{Pressure}}$ must be rejected).

When removing both social support and social pressure (run 4), there is a major difference in the mean number of children in comparison to the *pairfam* data. This distance increases over the course of the eight waves from initially 6 children to a maximum of 173 in the same manner as for the previously discussed runs. The total aggregated distance between simulation and real-world data is 553. Compared to run 1, this amounts to a combined loss of 410 births across all eight years. Compared with the other two runs, this loss is still 207 (run 2) and 357 (run 3), respectively. Combining the effects of both other experiment scenarios, this effect is statistically significant, as well. Therefore, null hypothesis $H_{0_{VoC}}$ is rejected since individual preferences alone lead to a severe underestimation of fertility.

In summary, the experiment results show that our model of social contagion is capable of simulating the effect of social mechanisms on fertility. Each mechanism has a significant impact on the number of children

Table 1: Variable combinations, simulation results, and hypothesis tests.

Run	**Variables**	**Simulation results**		**Hypothesis test**	
		cumulative mean	standard deviation	Two sample t-test (vs. run 1)	rejection
1	weightVoC, thldEA, thldS	22317.08	153.5802		
2	weightVoC, thldEA	21946.76	150.7841	t = 12.166 df = 98	t >1.98 $H_{0_{Support}}$ rejected
3	weightVoC, thldS	22138.2	146.29	t = 5.9635 df = 98	t >1.98 $H_{0_{Pressure}}$ rejected
4	weightVoC	21740.26	143.0658	t = 19.433 df = 98	t >1.98 $H_{0_{VoC}}$ rejected

over the course of the simulation. As the effects are strongest at different times, their combination leads to realistic results. However, even removing social pressure and social support still leads to fertility rates in the same order of magnitude as in the full setting and in the real-world data. Thus, the most important influence factors on fertility are a person's age and their individual attitude toward having children. Nevertheless, there is a strong tendency to underestimate fertile behavior if social contagion is neglected.

Despite these successful results, our model still tends to underrate fertile behavior in later waves, particularly of persons in their late thirties and in their forties (cf. waves seven and eight in Fig. 3). In these situations, it is likely that other mechanisms than initial preferences and social influence take effect. To represent those, it is possible to extend the model by a perceived pressure of age, dynamically changing VoC, or dynamically changing thresholds for support and social pressure. This would allow for representing an increasing urge to have children as a person grows older. The BDI-based agent architecture makes it simple to represent such additional influence factors for including them into an agent's decision-making.

Another method of addressing that kind of effect is to model interdependencies between several social contagion mechanisms. For instance, an agent's attitude toward children represented by its VoC is likely to also influence its emotional autonomy. A person with strong individual opinions will be less likely to be influenceable by family and friends exerting pressure on them. While this is already given in the dataset by correlating VoC and emotional autonomy, an individual threshold for social pressure instead of a population-wide one can reflect finer details of their interplay. The same holds for social support which correlates with social pressure. If parents expect their children to show fertile behavior, they are likely to give a large amount of support to foster this decision. For the sake of simplicity and to be able to compare the impacts of individual contagion mechanisms, our model includes these interdependencies only as they appear in the input data while keeping the mechanisms algorithmically independent from each other.

5 CONCLUSIONS

In this paper we have presented an agent-based social simulation study on contagion of fertility. We have introduced a novel decision-making algorithm that integrates individual motivations and social influences, namely *value of children*, *social pressure*, and *social support*. In addition, we have shown that our model is capable of producing realistic simulation results by calibrating and validating it using real-world reference data from the panel study *pairfam*. Moreover, we have conducted a simulation study to analyze the respective impacts of social contagion mechanisms on fertility. To that end, we have compared three hypothetical scenarios with the reference setting. Our results show that both social pressure and social support significantly increase fertility (individually and in combination). Without social contagion, the 2333 simulated couples have 410 children less than in the reference scenario over the course of eight years. Additionally, our results reveal varying impacts of different contagion mechanisms depending on a person's age. While social support is most relevant for young couples, social pressure manifests more strongly after a few simulated years. This is an interesting emergent effect of the agents' interactions within their

respective social networks. Our study shows that social simulation is particularly useful for identifying such effects in a controlled experimental environment.

Nevertheless, there is still potential for improvement and future research. As discussed, further interdependencies between mechanisms of social contagions can be included in the model to improve its long-term behavior and potential predictive capabilities. Furthermore, dynamically changing calibration parameters are an alternative method for achieving this. A driving force for such dynamic changes over time can be *social learning* by observing and imitating others (Bandura 1986). That mechanism of contagion can potentially amplify social dynamics even further. Finally, we also apply similar agent decision approaches to other application domains like social media analysis (Rodermund et al. 2017).

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