Reply to DellaPosta and Davoodi

American Sociological Association

American Sociological Review
2021, Vol. 86(6) 1205–1210

DOI:10.1177/00031224211057150
journals.sagepub.com/home/asr

SAGE

Associative Diffusion and the Pitfalls of Structural Reductionism

Amir Goldberg[a]

Abstract

In their insightful comment, DellaPosta and Davoodi argue that our finding (Goldberg and Stein 2018) that segmented networks inhibit cultural differentiation does not generalize to large networks. However, their demonstration rests on an incorrect implementation of the preference updating process in the associative diffusion model. We show that once this discrepancy is corrected, cultural differentiation is more pronounced in fully connected networks, irrespective of network size and even under extreme assumptions about cognitive decay. We use this as an opportunity to discuss the associative diffusion model's assumptions and scope conditions, as well as to critically reassess prevailing contagion-based diffusion models.

Sarah Stein and I thank DellaPosta and Davoodi (henceforth DD) for their thoughtful and gracious comment.[1] They push back against our finding (Goldberg and Stein 2018), challenging received wisdom in the sociology of diffusion, that segmented networks inhibit rather than promote cultural differentiation. Although they reproduce this dynamic—which we refer to as *connected differentiation*—they argue that it does not generalize to large-scale networks. Whether the associative diffusion (AD) model's predictions hold at all portions of the parameter space is, ultimately, a question at a more granular level of detail than the general theoretical argument put forth in our original article. Nevertheless, DD conclude that the AD model's predictions do not apply in "real world settings."

Unfortunately, DD's reversal hinges on an incorrect implementation of the original AD model. When an agent (the "observer") observes another agent (the "actor") display behaviors, she updates her preference for one of these behaviors (the one for which her preference is weaker). The magnitude of the update is drawn from a random distribution. In DD's implementation this distribution is uniform. In the original AD model, however, this distribution is normal.[2] As we will show, once DD's preference updating process is corrected, we find that connected differentiation extends to networks of size 500 or greater across the parameter space.[3] Contra DD's claims, no reversal occurs.

DD's decision to use a uniform distribution implies that the magnitude of agents' updates is equally likely to be large as it is to be moderate. A normal distribution, in contrast, implies that only a handful of agents dramatically update their preferences. DD's assumption is not categorically incorrect. Nevertheless, it is an inherently different assumption than the one informing the

[a]Stanford University

Corresponding Author:
Amir Goldberg, Knight Management Center, Stanford University, 655 Knight Way, Stanford, CA 94305
Email: amirgo@stanford.edu

original AD model. We conjecture that our approach is a more reasonable general model of preference updating, one that is consistent with what we know about human cognition.

But there is more to this exchange than a mere comparison of assumptions. DD's and our discrepant implementations provide a fortuitous opportunity to probe the potentially significant implications of these seemingly marginal assumptions. Let us therefore assume, for the sake of the argument, that DD's decision to rely on a uniform distribution has higher fidelity to how human agents behave in real life. As we will discuss, even if that were the case, a reversal of connected differentiation only occurs when DD introduce additional and unreasonably extreme assumptions into the AD model. These assumptions, we contend, echo the limitations of conventional contagion-based approaches in sociological studies of diffusion.

STRUCTURAL REDUCTIONISM REVISITED

Before delving into DD's results, let us first note the assumptions informing canonical social contagion models. Sociological work on diffusion, especially that which uses agent-based models, has been dominated by *structural reductionism*. To paraphrase Garfinkel (1967), this literature often treats agents as cognition-free "structural dopes," operating like relay stations whose only purpose is to automatically respond to external stimuli. The true actor in these models is therefore the network, as it is the network's structure that ultimately determines how the model reaches an equilibrium. For all its virtues, DellaPosta, Shi, and Macy's (2015) elegant and influential study demonstrates the limitations of this approach. Without the precondition of a connected caveman network, the model does not produce polarization. Indeed, as we show in Figure 7A of our original article, cultural differentiation emerges in homophily-based models (of the DellaPosta and colleagues' [2015] kind) only if the underlying network structure is segmented. Cultural differentiation, in essence, needs to be "baked in" structurally for conventional contagion models to produce polarization.

We believe our model's main contribution inheres in the introduction of an alternative that considers agents as more cognitively complex than simple human relay stations. Taking our inspiration from recent work in cultural sociology and cognition, we showed, in the original article, that the emergence of cultural differentiation can be fully attributable to the complexity of human cognition and does not necessitate a segmented network structure. Of course, we modeled a very simplified version of this cognitive complexity. Yet, as we showed, it is enough to assume agents do not blindly imitate one another, but rather learn associations from one another, to produce the emergence of cultural differentiation. That, we believe, is our model's main innovation. DD show that this result is reliably reproduced as the model's scale is expanded, even under their different updating regime. As they illustrate in Figure 1 of their comment, the model equilibrates at full preference congruence (the signature of complete cultural bifurcation) regardless of network size.

WHAT DOES THE DECAY RATE DO?

Why, then, do DD challenge our conclusions? They find that when the network size is 500 and the decay rate is very high (.7 to .8), a fully connected topology does not reach perfect preference congruence. In one of these conditions, when the decay rate equals .8, the emergent preference congruence in a fully connected topology is significantly (but only slightly) lower than that which emerges in a small-world network. In other words, of the 30 conditions they evaluate in the parameter space, only one produces an outcome that "reverses" (albeit slightly) the pattern we reported in the original article. In all but one other condition (where the decay rate equals .7 and $N = 500$), the fully connected topology consistently results in more pronounced

cultural differentiation than does the small-world topology.

Why might the pattern of connected differentiation suddenly "reverse" when the decay rate is set to .8? DD, we argue, make an unreasonable assumption about agents' cognition when they assign such a high decay rate. The decay rate controls the extent to which the associations represented by the agent in her association matrix weaken between model iterations.[4] It models a cognitive process of knowledge retention. In the original model we assumed a universal decay rate of .05 ($\lambda = .95$), which implies agents' representations of the associative order they observe attenuate at a slow pace.[5] A decay rate of .8, in contrast, implies agents very quickly forget what they have recently observed; an association learned at time t would effectively decay to 0 by time $t + 3$.[6] Like the protagonist in Christopher Nolan's movie *Memento*, agents in DD's world with a decay rate of .8 suffer from acute amnesia.

The rate at which cognitive associations decay is, ultimately, an empirical question. We do not purport to know the answer to that question. Nevertheless, we conjecture that, in the real world, humans' associative decay rate is far closer to .05 than it is to .8. Otherwise, like the protagonist in *Memento*, we would all have to walk around taking photos of our experiences.

DD are correct in noting that nondeclarative culture of the kind modeled by the AD model is learned slowly (Lizardo 2017). But the decay rate models agents' ability to retain knowledge, not the speed with which they acquire it. An AD model with a decay rate of .8 is, effectively, a world in which agents cannot learn the cultural order. It produces a model dynamic in which the mechanisms of associative learning and constraint satisfaction (i.e., the process by which the association matrix constrains an agent's preferences) are mostly short-circuited. DD's different implementation of the preference updating process amplifies this circumvention.[7] What we are left with is, in essence, a world dominated by conventional social influence. The thing that matters most in such a world is network structure.

Given that DD fine-tune the model to behave like a traditional contagion model, it is not all that surprising that they reproduce a canonical social contagion finding. As we noted in the original article (p. 921), "contagion models require structural complexity to explain cultural variation because they conceptualize interpersonal transmission as a simple epidemiological process." DD's exercise, effectively suppressing the associative diffusion apparatus, simply provides additional proof for that observation.

Why is connected differentiation only reversed when the network size is 500, not when it is smaller? DD do not provide a compelling answer. Rather, they repeat the canonical structuralist narrative about actors' attentions to "local contexts." When we corrected the preference updating process in their implementation of the AD model and ran it with a network size of 500, in contrast, we found that connected differentiation persists at scale whether the decay rate is high or low. As the upper row of Figure 1 illustrates, whereas a fully connected topology reaches full preference congruence at equilibrium, a small-world topology does not, irrespective of decay rate. In fact, we found this pattern holds when we extend the model to larger networks of size 1,000. As Figure 2 illustrates, the level of preference congruence at equilibrium in such large networks declines as the number of caves in the small-world topology increases. Consistent with DD's findings, the more segmented the network, the less pronounced the emergent cultural differentiation. Network segmentation, in other words, disrupts rather than promotes cultural differentiation. Unsurprisingly, it takes the model longer to reach an equilibrium when the decay rate is high. But even a decay rate as high as .8, unrealistic as it may be, does not upend the associative diffusion process, regardless of scale.

Although they sought to highlight the effects of network size on diffusion, DD, perhaps unintentionally, ended up producing

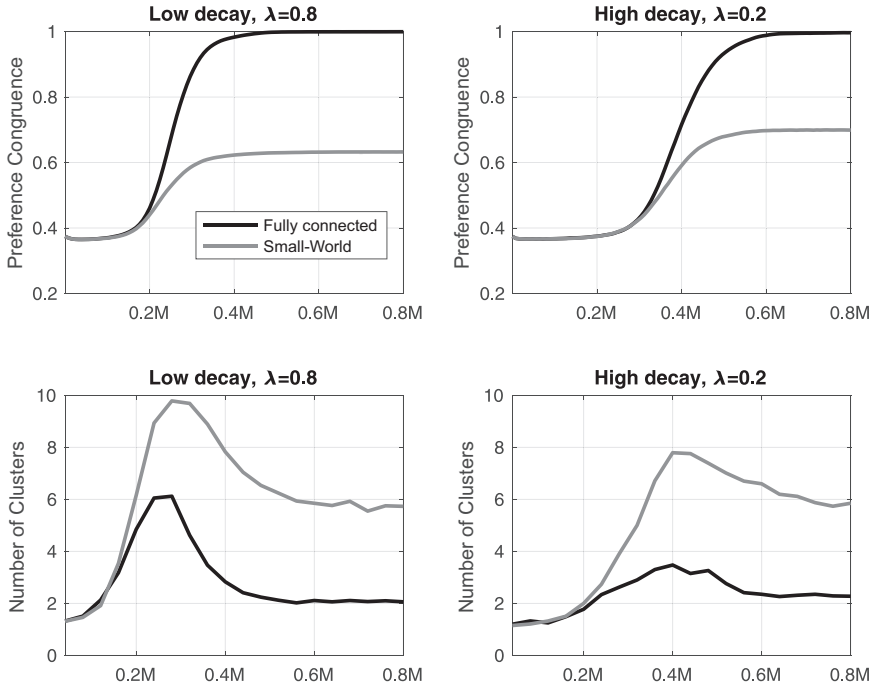

Figure 1. Associative Diffusion Models with 500 Agents, by Network Topology (Fully Connected in Black; Small-World with Five Caves in Gray) and Decay Rate

Note: Results are averaged across 100 simulations per topology/decay combination. The upper two panels present preference congruence (average absolute pairwise preference correlation between agents), and the bottom two panels the average number of clusters identified by the gap statistic (excluding clusters of size 1, see Goldberg and Stein [2018] for details). Note that the decay rate equals $1 - \lambda$.

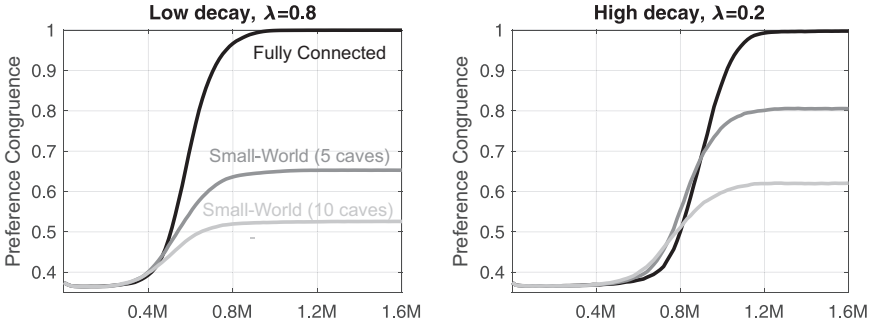

Figure 2. Associative Diffusion Models with 1,000 Agents, by Network Topology and Decay Rate

Note: Network topologies include fully connected (black), small-world with five caves (gray), and small-world with 10 caves (light gray). Results are averaged across 50 simulations per topology/decay combination. Each panel plots preference congruence (average absolute pairwise preference correlation between agents) by number of simulation iterations. Note that the decay rate equals $1 - \lambda$.

an analysis that mostly sheds light on the ways cognitive retention affects cultural differentiation. In fact, DD seem to overlook their most interesting finding, which was also reproduced in our analyses. As Figure 1 of their comment illustrates, when retention is

perfect (the decay rate is 0), cultural differentiation is attenuated regardless of network size or topology. In other words, agents' ability to forget appears to be an important precondition for pronounced cultural bifurcation.

We further found that, although the patterns of preference congruence are fairly consistent irrespective of decay rate, the trajectories of cultural differentiation are quite different across the decay rate spectrum. As the bottom panels in Figure 1 illustrate, when the decay rate is low and the network is fully connected, an early period of high interpretative ambiguity (as reflected in the number of clusters) is followed by a convergence toward two stable preference groups. These two groups have different preferences but are in interpretative consensus about which practices are associated with another.[8] When the decay rate is high, in contrast, convergence is slower to emerge but the number of cultural clusters peaks at a lower total. This is the case irrespective of network topology. Higher decay, in other words, results in less initial interpretative heterogeneity. At the same time, however, the number of clusters is significantly higher in the small-world topology regardless of decay rate. Consistent with connected differentiation, when diffusion is associative, a segmented small-world network structure impedes the emergence of full interpretative consensus even at a large scale.

CONCLUSION

DD's defense of the canonical findings of social contagion models rests, we believe, on the misconception that associative diffusion necessarily negates these models. It does not. As we discussed in the original article, the dynamics of associative diffusion do not apply to all forms of interpersonal cultural transmission. By the same token, however, not all forms of diffusion can be explained by simple or complex contagion. This does not make complex contagion theory wrong; rather, it implies it is incomplete. The assumption that cultural differentiation is merely epiphenomenal to network segmentation, as contagion models implicitly assume, is empirically indefensible. We hope DD's comment will spur further work investigating how the combined assumptions of associative diffusion and complex contagion—for example, if associative learning were to require a complex contagion-like threshold—can explain the interplay between network structure and cultural diffusion.

So what have we learned from DD's comment? As we showed, contra DD's argument, the dynamics of connected differentiation are robust to network size. DD achieve a reversal of this dynamic only when they fundamentally alter both how agents update their preferences and how they retain associative knowledge. These changes correspond to unreasonable assumptions about agents' cognition. We are nevertheless thankful to DD for highlighting the importance of probing the AD model's assumptions about agents' cognitive retention. We originally built the model with low-dimensionality realism (Bruch and Atwell 2015) to facilitate an understanding of the implications of associative learning. DD's results clearly highlight the need for expanding this dimensionality. Yet, because they are too focused on defending contagion models, they fail to see how the theories of associative diffusion and complex contagion are complementary, rather than mutually exclusive. Perhaps the greatest value of this exercise is in demonstrating why it is high time the sociology of diffusion moved beyond the constraints of structural reductionism.

Acknowledgments

We thank Bart Bonikowski and Michael Hannan for feedback on earlier versions.

ORCID iD

Amir Goldberg https://orcid.org/0000-0002-0858-3058

Notes

1. My co-author on the original article, Sarah Stein, was unavailable for writing this reply. It is written on her behalf as well.
2. Table 1 of our original article (as well as the article's main text on p. 909) explicitly states that the

updated values are drawn from a normal distribution with a mean of 0 and a variance of 1. Note 1 of DD's comment incorrectly states that the original article claims this distribution is uniformly bounded by 0 and 1.

3. Our model is implemented in Matlab. The code is publicly available at https://github.com/amirgo/associative_diffusion. Identical results are obtained whether using our original implementation or DD's implementation in R, once preference updating is corrected to follow a normal distribution.
4. We thank DD for pointing out that the parameter we label λ and call the decay rate is, in fact, the retention rate, which is the complement of the decay rate. Formally, the decay rate is $1 - \lambda$.
5. We apologize for omitting this information from the original article.
6. An association that equals 1 at time t will decay to $.2^3 = .008$ by time $t + 3$ unless it is observed again during that time. This level of decay rate means agents can only retain associations they consistently observe all the time. Given that the model works such that at any given moment the actor performs only two practices, it is extremely unlikely an agent will consistently observe the same association.
7. Unlike associations, preferences do not decay in the AD model. When the magnitude of a preference update is randomly drawn from a uniform distribution, it becomes more likely the agent will significantly increase or decrease her preference for the practice just enacted by the actor (relative to that likelihood when the random distribution is normal). This is analogous to a process of strong positive or negative influence.
8. As we explain in detail in the original article, interpretative heterogeneity exists when agents' association matrices are incongruent. A steady state of two preference groups emerges when all agents have similar association matrices—that is, they are in interpretative consensus—but diverge in their preferences.

References

Bruch, Elizabeth, and Jon Atwell. 2015. "Agent-Based Models in Empirical Social Research." *Sociological Methods & Research* 44(2):186–221.

DellaPosta, Daniel, Yongren Shi, and Michael Macy. 2015. "Why Do Liberals Drink Lattes?" *American Journal of Sociology* 120(5):1473–511.

Garfinkel, Harold. 1967. *Studies in Ethnomethodology*. Englewood Cliffs, NJ: Prentice-Hall.

Goldberg, Amir, and Sarah K. Stein. 2018. "Beyond Social Contagion: Associative Diffusion and the Emergence of Cultural Variation." *American Sociological Review* 83(5):897–932.

Lizardo, Omar. 2017. "Improving Cultural Analysis: Considering Personal Culture in Its Declarative and Nondeclarative Modes." *American Sociological Review* 82(1):88–115.

Amir Goldberg is an Associate Professor of Organizational Behavior and (by courtesy) Sociology at the Stanford Graduate School of Business, where he co-directs the Computational Culture Lab. His work uses computational methods to understand how culture evolves and how it affects individual and group-level outcomes.