



# Individual and collective encoding of risk in animal groups

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**The need to make fast decisions under risky and uncertain conditions is a widespread problem in the natural world. While there has been extensive work on how individual organisms dynamically modify their behavior to respond appropriately to changing environmental conditions (and how this is encoded in the brain), we know remarkably little about the corresponding aspects of collective information processing in animal groups. For example, many groups appear to show increased “sensitivity” in the presence of perceived threat, as evidenced by the increased frequency and magnitude of repeated cascading waves of behavioral change often observed in fish schools and bird flocks under such circumstances. How such context-dependent changes in collective sensitivity are mediated, however, is unknown. Here we address this question using schooling fish as a model system, focusing on 2 nonexclusive hypotheses: 1) that changes in collective responsiveness result from changes in how individuals respond to social cues (i.e., changes to the properties of the “nodes” in the social network), and 2) that they result from changes made to the structural connectivity of the network itself (i.e., the computation is encoded in the “edges” of the network). We find that despite the fact that perceived risk increases the probability for individuals to initiate an alarm, the context-dependent change in collective sensitivity predominantly results not from changes in how individuals respond to social cues, but instead from how individuals modify the spatial structure, and correspondingly the topology of the network of interactions, within the group. Risk is thus encoded as a collective property, emphasizing that in group-living species individual fitness can depend strongly on coupling between scales of behavioral organization.**

group structure | antipredator behavior | social contagion

**A** key challenge faced by animals is to appropriately adjust their behavioral responses to changing environmental contexts (1). To do so, organisms must make probabilistic decisions based on often imperfect or conflicting sensory information. Longer-term states such as fear or hunger can be considered as a persistent (but updatable) memory stored by the animal that modulates the mapping from sensory input to behavioral change. The mechanisms by which individual organisms achieve effective context-dependent behavior have been well studied (2–4), but what has been comparatively rarely explored is how such behavioral plasticity is encoded by organisms that live in groups. In highly coordinated animal groups, such as many species of schooling fish, flocking birds, or herding ungulates, individual reproductive success is often intimately linked with the functional complexity of collective behavior (5, 6). This introduces a coupling between individual (“microscopic”) properties and collective (“macroscopic”) behavior, and it is reasonable to expect that this coupling will impact how evolution has shaped the mechanisms by which individuals sense and respond to changing environmental conditions.

For example, if we consider an individual in isolation, it must base its decisions on sensory inputs and previous experience, which may also be modulated by physiological state. However, it is clearly the individual that is “responsible” for the decision. If we consider instead individuals embedded in a social network, another possibility is introduced: As in other information-processing networks, such as neural circuits, computation may be affected by changes in the individual components themselves (network “nodes”) and/or by changes in the structural connectivity (topology) among the components (network “edges”). In animal groups, individuals often exhibit a highly dynamic group structure, with individuals’ spatial positions, orientations, and sensory neighborhoods changing rapidly (5, 7–9). Yet nonetheless, individuals exhibit the capacity to change, consistently and repeatedly, the topology of their social connectivity by switching between what is often a relatively small number of group structural states (e.g., ref. 9). This presents an additional nuance to understanding collective cognition (10–12), as while individuals may be influenced by the topology of their network, they are also able to modify this topology through their movements and perception of the environment.

## Significance

Many biological systems exhibit an emergent ability to process information about their environment. This collective cognition emerges as a result of both the behavior of system components and their interactions, yet the relative importance of the two is often hard to disentangle. Here, we combined experiments and modeling to examine how fish schools collectively encode information about the external environment. We demonstrate that risk is predominantly encoded in the physical structure of groups, which individuals modulate in a way that augments or dampens behavioral cascades. We show that this modulation is necessary for behavioral cascades to spread and that it allows collective systems to be responsive to their environments even without changes in individual computation.

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Here we explore the possibility that information processing may be facilitated not only by individuals changing their internal behavioral rules/states, as is typically considered in animal behavior, but that, by forming a networked system, individuals can facilitate collective computation by changing the structural topology of the network (their social connectivity), without necessarily adjusting the way they respond to sensory information. We refer to changes in individual behavioral rules and states as individuals changing their responsiveness and to changes in group structure as individuals changing their spatial positioning.

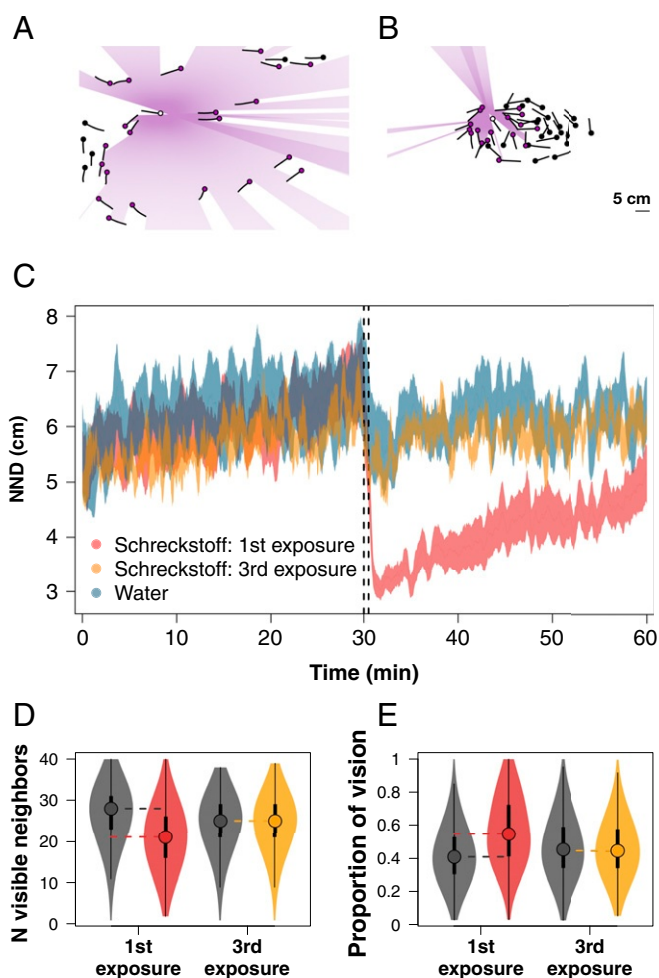
Across many animal taxa, group structure is known to be highly sensitive to group members' perceptions of risk and resources (13–19). These changes have generally been attributed to simple game theoretic considerations (20, 21), where structure is merely a byproduct of individuals acting to maximize their survival (5). But overlooked is the possibility that group structure, as an emergent encoding of the external environment, could itself be an important mechanism by which organisms effectively process information in a changing world. In this way, the group's structure could act as a collective memory that modifies future decisions, similar to how an individual's memory guides its own behavior (22, 23).

To test the relative contributions of group members' responsiveness vs. spatial positioning to collective information processing, here we present results from experiments with schooling fish (golden shiners, *Notemigonus crysoleucas*), known to have highly dynamic and self-regulating group structure (9, 16, 19), and use these data to investigate context-dependent changes in individual and collective responses to perceived risk. Like many fish species (3, 24, 25), predation is a source of extremely high mortality in the wild (26) and juveniles form coordinated schools in response to this risk. Shiners also exhibit startle responses as an escape behavior (27) that is socially contagious (28). Startles in this species occur even in the absence of an external stimulus, and these spontaneous false alarms propagate through the group in the same manner as triggered true alarms (28). In nature, false alarms account for a high proportion of overall alarms (29–32), very likely because there are such considerable costs to not responding to true threats relative to false alarms (33).

In our experiments, we manipulate the magnitude of perceived risk (individuals' priors that an immediate threat is present) by introducing, remotely, the natural alarm substance Schreckstoff. Schreckstoff is a family of chondroitins released from fish skin when punctured or torn, such as in the vicinity of a successful predation event (34–37), that induces a “fear response” in fish, increasing group cohesion and startling behavior (37–39). However, while response to Schreckstoff is innate (37), fish will habituate to Schreckstoff if repeatedly exposed with no paired stimulus (39–41). As will be shown, these changes in group structure and collective responsiveness (the increased spread of alarms) allow us to ask whether this context-dependent change in collective behavior results from individuals modulating their responsiveness to neighbors and/or whether risk is encoded by changes in the groups' internal spatial structure. Our analyses, involving automated tracking, computational visual field reconstruction, and determination of the functional mapping between socially generated sensory input and individual and collective response, allow us to not only distinguish between these alternative mechanisms, but also demonstrate the relative importance of each.

## Results and Discussion

**Group-Level Changes under Perceived Risk.** Schreckstoff changed group structure upon first exposure, but not upon third exposure (Fig. 1). In agreement with theoretical predictions on investment in antipredator behavior (4, 5), as well as previous empirical results (19, 35, 42–44), average nearest-neighbor distance sharply



**Fig. 1.** Effect of perceived predation risk on group structure. (A) A subset of the school prior to Schreckstoff. Rays (purple) represent a visualization of the field of view of the focal individual (colored white). (B) The entire school after receiving Schreckstoff. (C) Median nearest-neighbor distance upon exposure to Schreckstoff or water (dashed lines). Shaded regions indicate mean of the group medians  $\pm$  1 SE. (D) Distributions of number of visible neighbors before (black) or after (red and orange) exposure to Schreckstoff. Left and right pairs of violin plots correspond to first and third exposures to Schreckstoff, respectively. Dashed lines demonstrate difference in medians. (E) Distributions of the proportion of individuals' visual field occupied by other fish.

dropped when individuals were first administered Schreckstoff (permutation test, test statistic [t.s.] =  $-2.04$  cm,  $P < 0.0001$ ,  $n = 7$  groups; Fig. 1A–C; similar results for density, *SI Appendix*, Fig. S1). In contrast, individuals did not move closer to one another upon third exposure to Schreckstoff (t.s. =  $0.073$  cm,  $P = 0.14$ ,  $n = 6$  groups), in agreement with previous observations of groups of habituated fish (39–41).

Such dramatic changes in the spacing among individuals are associated with corresponding changes to the visual fields of the fish. Because golden shiners rely primarily on vision for schooling (28, 45, 46), we set out to quantify the visual properties of the group structure. Upon first exposure, group members on average saw fewer neighbors (Fig. 1D; permutation test, t.s. =  $-4.47$  neighbors,  $P < 0.0001$ ) and more of their vision was taken up by neighbors (Fig. 1E; t.s. =  $15.59\%$ ,  $P < 0.0001$ ). On the third exposure to Schreckstoff, however, there were no significant changes in the average number of visible neighbors (t.s. =  $-0.04$  neighbors,  $P = 0.37$ ) or proportion of visual field occupied by other fish (t.s. =  $0.00\%$ ,  $P = 0.30$ ).

**Changes to Cascade Frequencies and Sizes.** Because golden shiners frequently startle even in the absence of any apparent external stimulus (28), we can discern between 2 forms of individual-level responses to predation risk: changes in intrinsic alarming (the onset of an alarm cascade) and changes in alarm propagation (participation in an alarm cascade). We chose not to manually trigger startles, as a global stimulus makes it impossible to distinguish between the social (response to neighbors) and asocial (response to stimulus) progression of a cascade, and locally triggering individuals is experimentally challenging. In addition, our previous work has shown that startles triggered by an aversive stimulus are indistinguishable from, and propagate in the same way as, spontaneous startles (28). We developed a way of categorizing startles based on the product of speed and acceleration, which corresponds to the change in kinetic energy (work rate) of the startling individual (*SI Appendix, section 3*), allowing us to reliably identify startling fish by thresholding (*SI Appendix, Fig. S3*).

As suggested by previous studies (34, 40, 47), Schreckstoff increased the intrinsic frequency of startling (Fig. 2A). The number of cascades significantly increased on both the first and third exposures to Schreckstoff (1-way Wilcoxon signed-rank test on groups' difference in number of cascades: first exposure,  $V = 28$ ,  $P = 0.011$ ; third exposure,  $V = 15$ ,  $P = 0.029$ ). Exposure to water did not increase the frequency of alarms ( $V = 11$ ,  $P = 0.219$ ).

In addition to cascades being initiated more frequently under the first exposure to Schreckstoff, the average cascade size also increased (Fig. 2B; permutation test,  $t.s. = 0.954$  individuals,  $P < 0.0001$ ). On the third exposure, however, despite cascades occurring more frequently, the average number of participants did not increase ( $t.s. = 0.060$  individuals,  $P = 0.313$ ) and was

comparable to the water control (*SI Appendix, section 4.2*,  $t.s. = -0.117$  individuals,  $P = 0.221$ ).

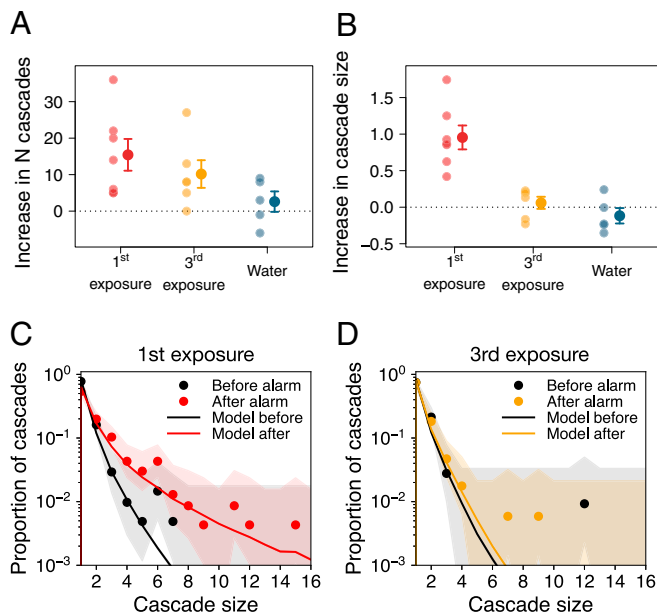
**Predictors of Startle Response.** The increase in cascade sizes under the first exposure to Schreckstoff can occur by 2 nonexclusive mechanisms: a change in internal rules or probabilities of response to neighboring alarms (individual responsiveness) and/or a change in group structure that enhances alarm propagation (spatial positioning). To tease apart these mechanisms, we set out to determine 1) the top predictors of response to a neighboring startle before and after Schreckstoff, 2) whether the sensitivity to these predictors changes with Schreckstoff, and 3) whether including information on when startles occurred (before or after Schreckstoff) improves our ability to predict whether a fish will respond.

To examine what is predictive of response to a neighboring startle, as in ref. 28, we focused on the first responder to an initiator in cascades featuring at least 1 responder (baseline,  $n = 46$  events; alarmed,  $n = 108$  events), since here we have the clearest causal relationship between alarm initiation and response. (Because startles are relatively rare, we combined prestimulus data from first-exposure Schreckstoff and water trials, i.e., prior to either treatment being given.) We then used  $L_1$ -penalized logistic regression (48, 49) to determine the features that are most predictive of response to an initiator. We included a set of features that can be broadly categorized into the measurable properties about the stimulus itself (e.g., distance, relative spatial orientation) and associated visual information. (Full details are in *SI Appendix, section 5.1*.)

In agreement with previous work on this species (28), the features best predictive of response to an initiating startle were the logarithm of the metric distance to, and the ranked angular area subtended by, visible neighbors (Fig. 3). These features emerged as the most predictive under both baseline and alarmed conditions. Similar features were found for predicting startle responses upon third exposure to Schreckstoff (*SI Appendix, section 5.1*). Our data also do not provide evidence of a change in the functional form for responding to a neighbor given these top 2 predictors after first exposure to Schreckstoff (see *SI Appendix, Tables S3 and S4* for logistic regression model coefficients and 95% confidence intervals for before and after Schreckstoff model fits).

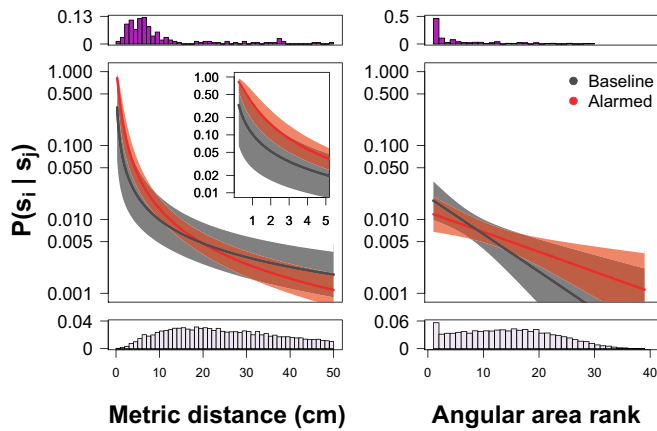
We then took 2 approaches to determine whether including information on when startles occurred (before vs. after Schreckstoff) improved our ability to predict whether a fish will respond. First, we fitted a mixed-effects generalized linear model with a logistic link function on all startles in the first exposure to Schreckstoff (*SI Appendix, section 5.2*). We included log metric distance and ranked angular area as fixed effects, as well as their interactions with time (before vs. after Schreckstoff). Cascade ID nested within group ID was included as a random effect. We did not find support for statistical significance for time or its interactions with log metric distance and ranked angular area (*SI Appendix, Table S5*). Then, we performed a likelihood-ratio test comparing this model to an identical model that did not include time or its interactions. Model fit did not significantly improve when including information on when startles occurred ( $\chi^2 = 3.925$ ,  $P = 0.270$ ).

Taken together, these results suggest that individuals follow the same rules for responding to neighbors, the sensitivity to these rules does not change with Schreckstoff, and information on when startles occurred does not improve our ability to predict responses. While this indicates that changes in individual responsiveness with Schreckstoff are either negligible or small, these results alone are insufficient to conclude that changes in responsiveness do not contribute to changes in collective sensitivity. Below we employ a behavioral contagion model that builds on these results and explicitly compares the relative contributions



**Fig. 2.** Perceived predation risk changes alarm propagation. (A) Schreckstoff increases the intrinsic frequency of alarms: The number of startle cascades increased upon first and third exposure. Raw data are plotted alongside the mean  $\pm 1$  SE. (B) Schreckstoff increases average cascade size following first exposure, but not upon third exposure. Exposure to water does not increase frequency of alarms or average cascade size. (C) Distribution of cascade sizes before and after the first exposure to Schreckstoff. Lines represent contagion model fits to data, with shaded regions representing 95% confidence intervals. Model is described in main text. (D) Cascade size distributions before and after the third exposure to Schreckstoff.





**Fig. 3.** Probability of an individual startling in response to an initiator as a function of the top 2 predictors, log metric distance (*Left*) and ranked angular area (*Right*), holding the other predictor constant at its mean value. Gray corresponds to the first-exposure data prior to receiving Schreckstoff; red corresponds to after receiving Schreckstoff. Solid lines are the fit of the model with the top 2 predictors to the first-responder data; shaded regions represent 95% confidence intervals. *Top* and *Bottom* histograms correspond to first responders and nonresponders, respectively.

of individual responsiveness and spatial positioning on a common scale.

**Changes to Cascade Size Distribution.** Are changes in the spatial positioning of group members sufficient to account for larger cascades? Or are there changes in individual responsiveness that do not play a role at the onset of cascades but still contribute to their spread? Answering these questions requires that we consider the entire behavioral contagion process, as the decision of whether or not to startle likely depends on the decisions of all of an individual’s observable neighbors (28).

Thus, to understand the origin of the change in average cascade size, we investigated a generic model of behavioral contagion that incorporates 2 key components: the sensitivity of individuals to available social cues and the structure of the interaction network. The latter is given by a network of weighted edges  $w_{ij}$  that represent the probability of individual  $i$  to be a first responder given that individual  $j$  initially startled. The interaction network for each trial is parameterized directly by fish positions and orientations via a logistic regression on sensory features detailed in the previous section (*SI Appendix, Eq. S3*). In this way, the interaction network captures the relevant differences in pre- and postexposure sensory features caused by changes to spatial positioning (Fig. 1). Once the interaction network is fixed, the complex contagion model contains a single free parameter that specifies the social sensitivity of individuals. This sensitivity parameter (“dose threshold”) (50) determines how much perceived risk an individual tolerates before startling. An internal state (the “cumulative dose,” Fig. 4A) tracks an individual’s perceived risk based on the time course of startle responses of its network neighbors, causing a startle once reaching the threshold. See *Materials and Methods* and *SI Appendix, section 6* for a complete description of the model. We fit this individual-level sensitivity parameter to the observed cascade size distributions in 4 cases: before and after Schreckstoff in both the first and third exposure treatments (Fig. 4B and C).

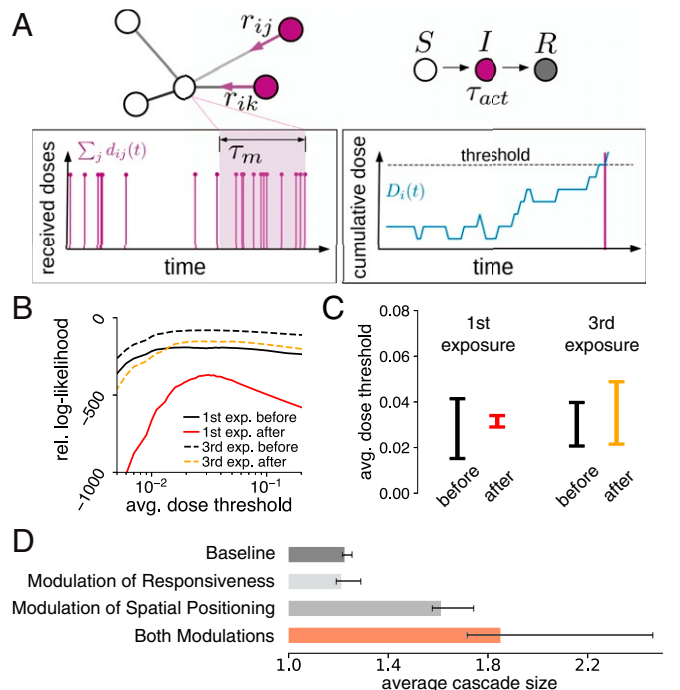
For both experimental treatments (first and third exposure), the 95% credible intervals of the maximum-likelihood estimated dose thresholds before and after Schreckstoff overlap (Fig. 4C), indicating that a change in individual responsiveness to the startles of neighbors is not required to explain the observed increase in average cascade size under perceived risk. Changes in spa-

tial positioning, however, are sufficient to explain this increase. Moreover, the lack of a change in spatial positioning in the third exposure case, resulting in no difference in average cascade size pre- and post-Schreckstoff, indicates that a change in spatial positioning is also necessary to account for a change in average cascade size.

Finally, we quantified the relative contributions of responsiveness and spatial positioning to average cascade size with a full factorial design (*SI Appendix, section 6.3*). We trained a regression model that allows weights of the contagion network to depend both on the presence of Schreckstoff (capturing changes due to individual responsiveness) and on distances and orientation (capturing changes due to spatial positioning). In the resulting behavioral contagion model, the increase in the average size of cascades post-Schreckstoff (Fig. 4D) could not be accounted for by changes to individual responsiveness alone. Instead, the increase in cascade sizes required the observed change in spatial positioning. Thus we find that changes to spatial positioning are essential for the increase of group responsiveness post-Schreckstoff.

### Conclusions

The central question of our paper is whether collective sensitivity is modulated by changes in individuals’ responsiveness (rules for



**Fig. 4.** SIR-type behavioral contagion model explains alarm propagation and indicates that changes in spatial positioning are necessary to explain increased cascade sizes. (A) Model schematic. The focal individual in the susceptible state ( $S$ , white) receives doses from active individuals ( $I$ , “infected,” magenta) that it integrates over timescale  $\tau_m$ . When the cumulative dose reaches the individual’s threshold, it startles and remains in the active state for time  $\tau_{act}$ , after which it enters the recovered state ( $R$ , gray). (B) The model is simulated starting with the observed initial starter and dose rates that correspond to the first-responder probability functions for first and third exposure to Schreckstoff. The relative log-likelihood of the model producing the observed cascade sizes is plotted as a function of the single free parameter that modulates individual responsiveness, the average dose threshold  $\bar{\theta}$ . (C) Best-fit parameter values controlling responsiveness are similar pre- and postexposure, with overlapping 95% credible intervals. (D) Comparing average cascade sizes after modulating responsiveness and spatial positioning separately reveals that a change in spatial positioning is essential for the increase of group responsiveness post-Schreckstoff.

translating sensory input into alarms), their spatial positioning (the physical spacing and sensory network of group members), or some combination of them. In solitary animals, the only option for responding to changing environmental conditions is to modify responsiveness. For social animals such as golden shiners, either option (or a combination) is possible. Our approach allows us to separate the relative contributions of spatial positioning and individual responsiveness, and we find that any changes in collective responsiveness are predominantly encoded in spatial positioning.

Using a combination of experiments and modeling, we demonstrate that individual-level changes in responsiveness do not contribute meaningfully to the augmented spread of startle cascades under perceived risk. Risk did not change the sensory features predictive of responding to neighboring alarms or the sensitivity to these features. Information on whether a startle occurred under baseline or alarmed conditions did not improve the ability to predict startle responses. In our behavioral contagion simulations where we explicitly vary individual responsiveness, we found that changes in responsiveness are not necessary to generate the observed changes in cascade sizes. Finally, when simulating cascades under solely changes in responsiveness, changes in spatial positioning, or both, we find that average cascades did not change with changes in responsiveness but did with changes in spatial positioning.

In contrast to typical conceptualizations of collective cognition, in which individuals interact on a relatively fixed network structure (51, 52), the fish schools in our experiment can change their group structure on the same timescale as relevant changes in the environment. The fact that this group structure encodes relevant environmental features suggests that the fish could actively control and make adaptive use of their emergent group features, a concept with growing theoretical support (53–57). The work we have presented here indicates the potential for self-organized animal groups to reveal additional insights into how dynamical networks may play an important role in collective intelligence emerging from simple interacting components.

## Materials and Methods

**Experiments.** Groups of 40 golden shiners were filmed freely swimming in a  $1.06 \times 1.98$ -m tank filled to 4.0 cm depth. One hour after being transferred to the tank, an automated sprayer released either Schreckstoff or water into the tank. The group was then filmed for an additional 0.5 h. No experimenter was present in the room for the duration of the trial. Details on data extraction, processing, and analysis are available in *SI Appendix, section 1*. All experiments were conducted in accordance with Princeton University's Institutional Animal Care and Use Committee.

**Behavioral Contagion Model.** Our model is based on a generalized model of contagion proposed by Dodds and Watts (50, 58). Here, we have reformulated the original model in terms of activation rates to describe behavioral contagion dynamics in continuous time. This allows us to more easily constrain parameters based on experimentally determined timescales and networks of influence, derived from the logistic regression's predictions for response probabilities given fish positions at the time of the initial startle. We then simulate the model using a standard Euler discretization.

Individual fish, as nodes in a network, are connected by weighted directed edges  $w_{ij} \in [0, 1]$  that define the rate of signaling doses received by individual  $i$  when individual  $j$  startles. Each individual  $i$  can be in 1 of 3 states  $s_i$  that we call susceptible, active, and recovered. Susceptible nodes

may become activated due to inputs received from active neighbors. After a fixed activation time  $\tau_{act}$ , activated individuals transition into the recovered state. The activation time is set to  $\tau_{act} = 0.5$  s, matching the experimentally observed average startle duration. For simplicity, we consider the recovered state as an absorbing state with no outward transitions, which restricts the model dynamics to single, nonrecurrent cascades. A simulation run is terminated when no active individuals remain.

As an initial condition we set all individuals as susceptible, and at time  $t = 0$  a single individual is activated (spontaneous startle). A susceptible individual  $i$  receives from an active neighbor  $j$  stochastic doses of activation signal of size  $d_a$  at a rate  $r_{ij} = \rho_{max} w_{ij}$ , with  $\rho_{max}$  being the maximal rate of sending activation doses for  $w_{ij} = 1$ . The maximal activation rate is bounded by limits on response times due to physiological constraints and neuronal processing of sensory cues which trigger a startling response in fish (59). The fastest startling responses to artificial stimuli were reported to be of the order of few milliseconds. Therefore, we assume  $\rho_{max} = 10^3 \text{ s}^{-1}$ , which allows in our model for fastest response times of the order of 1 ms (for  $w_{ij} \approx 1$ ). To be able to resolve this timescale, we choose the numerical time step accordingly to  $\Delta t = 1$  ms ( $\rho_{max} = 1/\Delta t$ ).

Thus, with small  $\Delta t$ , the activation signal received from individual  $j$  is a stochastic time series  $d_{ij}(t)$  with 2 possible values,  $d_a$  and 0, whereby the probability of receiving an activation dose per simulation time step  $\Delta t$  is  $p_a = r_{ij} \Delta t$ . Each agent integrates all inputs over a finite memory  $\tau_m = 2$  s. The agent becomes activated if the cumulative dose

$$D_i(t) = \frac{1}{K_i} \sum_j \int_{t-\tau_m}^t d_{ij}(t') dt' \quad [1]$$

received by a susceptible agent  $i$  within its memory time exceeds its internal threshold  $\theta_i$ . Here,  $K_i$  is the in degree of the focal individual, such that the doses received by the focal individual are rescaled by the number of its network neighbors, a form supported by prior work in a similar system (28). The individual thresholds are drawn from a uniform distribution with minimum 0 and maximum  $2\bar{\theta}$ , producing an average threshold of  $\bar{\theta}$ . This accounts for stochasticity due to inaccessible internal states of individuals at the time of initial startle.

The expected value of the cumulative activation dose received by agent  $i$  due to the activation of a single neighbor  $j$  ( $K_i = 1$ ) over the activation time  $\tau_{act}$  is thus  $\langle D_i \rangle = d_a \rho_{max} w_{ij} \tau_{act}$ . We choose the weights  $w_{ij}$  to be equal to the probability that  $i$  responds and is the first responder to an initial startle of  $j$ , inferred using the logistic regression model depicted in Fig. 3. The linear relationship between the cumulative dose  $\langle D_i \rangle$  and the weights  $w_{ij}$ , along with the uniform distribution of thresholds across fish, guarantees that the complex contagion process produces the correct relative initial response probabilities in the limit of small  $\Delta t$  and  $w_{ij}$  (*SI Appendix*). Without loss of generality, we can set  $d_a \rho_{max} = 1$ . Thus, based on the maximal rate  $\rho_{max} = 10^3 \text{ s}^{-1}$ , we set the activation dose  $d_a = 10^{-3}$ . This leaves us with a single free parameter, the average dose threshold  $\bar{\theta}$ , which we fit via maximum likelihood. A total of  $10^4$  independent runs were performed for each threshold value to estimate corresponding cascade size probability distributions.

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