Collective motion conceals fitness differences in crowded cellular populations

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Many cellular populations are tightly packed, such as microbial colonies and biofilms, or tissues and tumours in multicellular organisms. The movement of one cell in these crowded assemblages requires motion of others, so that cell displacements are correlated over many cell diameters. Whenever movement is important for survival or growth, these correlated rearrangements could couple the evolutionary fate of different lineages. However, little is known about the interplay between mechanical forces and evolution in dense cellular populations. Here, by tracking slower-growing clones at the expanding edge of yeast colonies, we show that the collective motion of cells prevents costly mutations from being weeded out rapidly. Joint pushing by neighbouring cells generates correlated movements that suppress the differential displacements required for selection to act. This mechanical screening of fitness differences allows slower-growing mutants to leave more descendants than expected under non-mechanical models, thereby increasing their chance for evolutionary rescue. Our work suggests that, in crowded populations, cells cooperate with surrounding neighbours through inevitable mechanical interactions. This effect has to be considered when predicting evolutionary outcomes, such as the emergence of drug resistance or cancer evolution.

The growth and division of non-motile cells tends to produce densely packed assemblages. The tight packing of cells is the natural consequence of population growth against a visco-elastic extracellular environment, and can be further promoted by attractive cell–cell interactions4,2. Common examples range from microbial communities, in the form of colonies or biofilms, to multicellular structures, such as developing tissues or tumours.

An important consequence of high packing density is that any cellular growth and division increases the spatial extent of the population. Statistical approaches to describe the ensuing growth dynamics have therefore been based predominantly on the framework of range expansions3–6. This analogy is particularly apt when most of the growth occurs in a ‘growth layer’ at the population margins5,7–12. In this case, ‘pioneer’ cells near the periphery have a twofold advantage: they enjoy high growth rates and place their offspring near the periphery so that their descendants can become the pioneer cells of the next generation.

The positional advantage of pioneer cells has important population genetic consequences. Mutant clones can reach high frequencies just by chance, when their descendants happen to keep up with the advancing edge of the expanding population. This process, termed ‘gene surfing’, is highly stochastic, but can be biased by mutations that increase or decrease the expansion velocity13,14. Successfully surfing clones grow in well-segregated sectors, which have a spatial structure that reflects the competition between genetic drift and selection4,15,16. These population genetic phenomena have been directly verified in microbial evolution experiments and shown to strongly influence evolutionary outcomes, including drug resistance3,10,11, the emergence of cheater types14,17, mutualism18 and adaptation19. Even though spatially resolved experiments in higher eukaryotic systems are much more challenging, hallmarks of gene surfing, including the formation and coarsening of clonal sectors, were recently identified in cancer20.

Despite this progress, we still lack an understanding of how evolutionary dynamics arise from the interactions between individual cells. In particular, conventional models of range expansions, based broadly on pulled or pushed reaction–diffusion waves, do not capture cell–cell forces resulting from the tight packings of growing cells. These forces have been seen to matter for the structure and function of biofilms21–23, and they might also influence natural selection. For example, slower-growing cells might be pushed along by the forces generated by faster-growing cells, leading to a form of mechanical cooperation.

To explore the interplay between mechanical forces and evolution, we tracked the evolutionary fate of slower-growing clones in dense colonies of the budding yeast Saccharomyces cerevisiae. We find that high population density inherently results in a screening of fitness differences on small length scales due to the collective motion of nearby cells. Our results can be described in the framework of an effective surface tension originating from cumulative cell–cell forces.

**Results**

As a model system for crowded cellular populations, we focused on colonies of the budding yeast S. cerevisiae24. Since yeast cells lack motility, colony expansion is fuelled purely by the pushing forces generated by cellular growth in a peripheral growth layer (see Supplementary Fig. 7)3,10,11. To explore how these pushing forces affect the strength of natural selection, we competed a strain carrying a growth-rate deficit s with a faster-growing strain in expanding colonies (Fig. 1a)4. The behaviour of the slower-growing strain is equivalent to that of a clone carrying a deleterious mutation—a ubiquitous scenario in population genetics. We therefore refer to the slower-growing strain as ‘mutant’ and the faster-growing strain as ‘wild type’ throughout this work (see also Table 1). During colony expansion, mutant and wild-type cells remained in well-segregated monoclonal sectors separated by sharp boundaries5.

We could thus monitor the gradual decay of mutant clones by measuring the width w of mutant clones as a function of the front propagation distance y.

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We found that mutant clones were purged from the population front in two stages. In the first stage, the clone width decreased at a constant rate, \( \frac{dw}{dt} \sim \text{constant} \). We observed this behaviour both in circular (Fig. 1b,c) and linear (Fig. 1d) inoculations. To disentangle evolutionary dynamics from radial expansion effects, we focus on linear inoculations in this work (see Supplementary Fig. 1 for an example of a linear front). The constant rate of mutant decline is in line with a minimal null model where local front expansion velocities depend only on the cell-type-specific growth rates of the furthest-forward pioneer cells at the leading edge, or ‘front cells’\(^{13} \) (dashed black lines in Fig. 1d; see Supplementary Information section 1 for model details). The null model predicts that the width of the mutant clone decreases at a constant rate until all mutant cells are expelled from the front. This form of abrupt clonal ‘extinction’ is in agreement with commonly used non-mechanical simulations (Supplementary Fig. 19b–d). In contrast, we observed a second stage of mutant decline as the clone width fell below a characteristic width of \( w_c \sim 230 \mu m \) (~46 cell diameters). Then, clones progressively slowed down narrowing, and gradually tapered to form elongated streaks. As a result, clones persisted at the growing front over distances that far exceed the null expectation (Fig. 1e,f). The consequences of this persistence became apparent on the colony level: even after eight days of colony growth, none of the slower-growing sectors shown in Fig. 1c (no matter how narrow initially) had been completely expelled from the front.

### Table 1 | \textit{S. cerevisiae} strains used for this work

| Name   | Type      | Colour | Resistance                  | Genotype                                               |
|--------|-----------|--------|-----------------------------|--------------------------------------------------------|
| yJK22  | Mutant    | Yellow | Hygromycin B                | his3:prACT1-ymCitrine-tADH1:HygMX                     |
| yMM9   | Wild type | Blue   | Cycloheximide               | his3:prACT1-ymCherry-tADH1:His3MX6 CYH2:cyh2-Q37E     |
| yMM8   | Wild type | Yellow | Cycloheximide               | his3:prACT1-ymCitrine-tADH1:His3MX6 CYH2:cyh2-Q37E     |
| JHK111 | Ancestor  | –      | –                           | his3:prACT1-ymCitrine-tADH1:His3MX6                   |
| JHK102 | Ancestor  | –      | –                           | W303 MATa bud4Δ::BUD4(S288C) can1-100                  |

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Fig. 1 | Slower-growing clones persist at the front of expanding yeast colonies. a. Schematic of the experimental set-up. Competition experiments were conducted with a faster-growing yeast strain (\( \text{yMM9, blue} \)) and a slower-growing yeast strain (\( \text{yJK22, yellow} \)) with the growth-rate deficit \( s = -0.06 \). Images in all figures are shown in false colour. Circular colonies were inoculated from a mixture of both strains (the initial fraction of faster-growing cells was 10%). In linear colonies, sectors expanded from flat fronts composed of extended regions with alternating cell types. b. Schematic of a circular colony grown for 8 days from a mixture of both cell types (the dotted circle indicates the area of cell deposition). Monoclonal ‘sectors’ remain well separated and expand outwards at different rates (blue and yellow arrows). c. Sectors of the slower-growing strain form an inverted funnel shape with elongated tips (yellow arrowheads) that persist at the front before being completely expelled. d. Schematic of a mutant sector (yellow) being expelled from a linear colony (see Supplementary Video 1 for a complete time lapse, and Supplementary Fig. 1 for the experimental set-up). The strains and colours are as in a–c. Solid white lines mark the front position at the indicated times after inoculation. Dashed black lines delineate the sector shape predicted by the null model (the local front speed only depends on the cell type. \( w_c \sim 230 \mu m \) is the characteristic clone width below which observations deviate from null model predictions (see Supplementary Fig. 3 for an assessment of \( w_c \)). Sector extension (yellow arrow) is the difference between the observed sector length (blue arrow) and the null model expectation (orange arrow). Positions along the axis of sector expansion, \( y \), are measured relative to \( y_c \), the position at which \( w_c = w(y_c) \). e. Observed clone lengths (blue dots) as a function of the expected length (also indicated as orange dots) for \( n = 41 \) sectors. \( y_{\text{extinct}} \) indicates the position of clone expulsion from the front. Yellow arrows indicate sector extension (see d). Histograms show binned distributions of expected and observed lengths. \( c_{\text{corr}} \) is the Pearson correlation coefficient of both lengths. f. Histogram of the sector length extensions (yellow arrows in e). Sectors are significantly \( (P < 10^{-22}) \) extended by an average of 1,150 ± 340 \( \mu m \).
Thus, purifying selection is inefficient in removing small mutant clones in our experiments. Next, we aimed to show that a strongly reduced efficacy of selection is not specific to yeast colonies but, in fact, is a predictable consequence of growth in crowded cellular populations, and that this effect can dramatically alter probabilities of important evolutionary outcomes.

The fate of clones in crowded populations fundamentally depends on the relative motion of mutant and wild-type front cells at clonal boundaries. In the null model, the direction of cell motion abruptly changes at the wild-type/mutant boundary (Fig. 2a). A delay of the extinction of mutant clones could arise from a suppression of such discontinuities. Indeed, while front cells much further apart than $w_c$ can have substantially different velocity vectors, their direction of motion becomes highly correlated at smaller distances (Fig. 2bc and Supplementary Videos 3 and 4). Front cells, including those at the mutant/wild-type boundary, tend to move perpendicular to the local front line, with very little lateral motion. This normal motion is also reflected in the trajectories of clone cells at clonal boundaries. In the null model, the direction of motion abruptly changes at the wild-type/mutant boundary, tend to move perpendicular to the local front line. This model corresponds to a multi-type extension of the Kardar–Parisi–Zhang model—a well-studied model of surface growth (see Supplementary Information section 3). The surface tension model reproduces the observed clone width dynamics very well on tuning the surface tension, our single fitting parameter (see Fig. 2f). The condition that motion is perpendicular to the local front line is an essential ingredient for the delayed extinction of the mutant clone. Even a small tilt

Curvature suppression is an active, growth-driven process: Fig. 2d shows how a region of high curvature in a purely wild-type front, deliberately formed by initial cell deposition, is smoothed out by subsequent growth. Cells in front regions exhibiting a higher degree of curvature move faster than those in flatter regions (Fig. 2e and Supplementary Videos 3 and 4).

Based on these observations, we hypothesized that the dynamic suppression of high front curvatures, and thus the directional alignment of nearby cells, could be described by an effective surface tension. To test this, we set up a phenomenological ‘surface tension model’ that represents the expanding population as a moving one-directional front line whose velocity depends not only on cell type but also on local front curvature (Fig. 2f). The curvature dependence is controlled by an effective surface tension term that gives a velocity boost to indented divots in the front, as observed in our experiments (Fig. 2d,e). Mutant and wild-type lineages occupy contiguous portions of the front, separated by sharp boundaries that move perpendicular to the front line. This model corresponds to a multi-type extension of the Kardar–Parisi–Zhang model—a well-studied model of surface growth (see Supplementary Information section 3). The surface tension model reproduces the observed clone width dynamics very well on tuning the surface tension, our single fitting parameter (see Fig. 2f). The condition that motion is perpendicular to the local front line is an essential ingredient for the delayed extinction of the mutant clone. Even a small tilt
results in abrupt extinction rather than extended clonal dynamics (Supplementary Fig. 17).

So far, we have shown that the delayed extinction of slower-growing clones can be traced back to the active suppression of high front curvature. This effective surface tension leads to the directional alignment of the motion of mutant front cells with that of the flanking wild-type population, thereby concealing the fitness difference between the two types. We can describe this screening effect by an effective fitness difference \( s_m \) between mutant and wild-type cells that is sharply diminished for clones of small width (Fig. 2g).

Which mechanism could generate an effective surface tension capable of driving the observed directional alignment? Expecting cell–cell pushing forces within a crowded growth layer to be the root cause for the collective cellular motion (see Fig. 3a), we tested in silico whether surface tension can be generated purely through the mechanical interactions between cells in a dense population. To this end, we simulated growing and dividing cells as proliferating elastic objects that move via overdamped dynamics (Fig. 3b). Similar to the expansion of yeast colonies (Supplementary Fig. 7), only cells within a peripheral growth layer grow and divide. In this simplified model, we approximated the complex growth-rate profile observed in experiments by assuming an effective growth layer of constant width \( \lambda \) and a uniform growth rate within the layer (see Methods for model parameterization and simplifying assumptions). These
simulations allowed us to directly quantify the force exerted on each cell in the growth layer. Figure 3c shows that cell–cell forces in an undulating monoclonal front are larger in divots than in flat regions or bulges. Consequently, front cells in divots move faster than those in bulges. The resulting velocities are consistent with a surface tension proportional to the growth layer depth (Fig. 3d).

In two dimensions, the picture subtly changes in that the front velocity also depends on the local curvature of the growth layer (Fig. 3d,h): if the front line is indented and the growth layer depth is constant, there are more growing cells per unit length of curved front compared with the flat-front case. This leads to an increased rate of biomass production. Incompressibility of the cell packing requires that additional space needs to be made for the excess biomass. As a result, the front advances at a slightly higher rate, which allows for the excess biomass to be added to the growth layer. Conversely, we observe a local slow-down if the front exhibits a bulge, so that fewer cells per unit length of front contribute to pushing. Thus, a negative feedback on front curvature, which is equivalent to a surface tension, spontaneously arises from purely passive, mechanical effects generated by cell proliferation within a growth layer. Note that the link between excess biomass production and forward motion

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Fig. 4 | Slow purging of costly drug-resistant mutations can lead to resurgent growth on drug application. a, Fluorescent micrographs of thin sectors originating from single mutant cells after four days of growth. Colonies were grown in a linear geometry from a mixture of strains yJK22 (5%, yellow; s = −0.06) and yMM9 (95%, blue) (see Supplementary Fig. 6 for a wider field of view). b, Corresponding surface tension model simulations (s = −0.06 and T = 25 µm, as in Fig. 2f), including stochastic boundary drift (see Methods for a description and parameterization). c, Null model (s = −0.06), including boundary drift. d, Probability of a clone persisting at the front, P_{front}, as a function of the front position, y, relative to inoculation. Experimental data represent the average of n = 12 fronts (~3,500 initial sectors; see Supplementary Fig. 6 for an example). The shaded grey area indicates one s.d. For neutral experiments (s = 0), mutant cells (strain yJK22) were substituted for cells with a growth rate identical to that of the wild type (strain yMM8). The surface tension model and null model results are averages of n = 10^3 independent sectors for both models. e, Image of persisting and expelled mutant sectors (yellow, yJK22; s = −0.06) in a wild-type background (blue, yMM9) after 4 days of growth. Mutant cells are resistant to the antimicrobial hygromycin B, while wild-type cells are susceptible. f, Application of hygromycin B to the entire colony after 6 days of growth completely arrested wild-type growth. Mutant clones still present at the front at the time of drug application (right) expanded quickly (see Supplementary Video 8), while those inside the colony (left) remained confined.
is based on the mechanical incompressibility constraint of the cell packing, which is a key feature of crowded cellular populations.

In addition to a sufficiently strong effective surface tension, our cell-based model produces two additional essential ingredients for fitness screening: sharp wild-type/mutant domain boundaries (Supplementary Fig. 21) that move perpendicular to the front line (Supplementary Fig. 16). This condition also arises in a continuum description of colony growth, such as diffusive waves, where domain boundaries are generically tilted with respect to the front line (Supplementary Fig. 16). Our cell-based simulations thus generate all of the key ingredients of the surface tension model: a population front with a natural surface tension, and sharp domain boundaries that move normal to that front. Consequently, on simulating a slower-growing mutant clone in a wild-type background, we reproduce the experimentally observed funnel-like sector shapes with extended tips (Fig. 3i).

Our results so far suggest that slower-growing mutant clones at the advancing frontier are mechanically screened from competition with wild-type cells—an effect that is strongest for clones of small width. Clones emanating from single mutant front cells should therefore benefit the most and exhibit nearly neutral lineage dynamics. To test this prediction, we investigated the fate of clones originating from single mutant cells interspersed into a front of wild-type cells (see Supplementary Fig. 6). These experiments indeed showed that mutant clones form elongated streaks (Fig. 4a) similar to a stochastic version of the surface tension model (Fig. 4b; see Methods).

Even though slower-growing mutant clones can still be expelled from the expanding front due to stochastic fluctuations in clone width, their probability of persisting at the progressing front decays several orders of magnitude more slowly than the null model expectation (Fig. 4c,d). In addition, these extended lifetimes allow slower-growing mutant clones to produce a larger total number of offspring than expected from our null model (Supplementary Fig. 20).

The inefficient purging of slower-growing mutants can have crucial evolutionary consequences when the selection pressure suddenly shifts in favour of the mutants. For instance, costly drug-resistant mutants may persist long enough to trigger resurgent growth on drug application16,25. To test whether the screening of fitness effects promotes such an evolutionary rescue26,27, we first grew a colony interspersed with a small fraction of single mutant cells, as described in Fig. 4a. These mutants are resistant to the antimicrobial hygromycin B while having a fitness cost in the absence of the drug. Despite their growth disadvantage, some mutant clones persisted at the front even after four days of colony expansion (Fig. 4c,e). We then subjected the entire colony to high levels of hygromycin B, completely starving wild-type growth (see Methods). While mutant clones trapped inside the colony remained effectively confined by surrounding wild-type cells, those clones persisting at the leading edge radiated out, establishing an all-mutant population expansion (Fig. 4f).

Although our study focuses on deleterious mutations, the mechanism proposed here acts on less fit and more fit cells alike. Consequently, the screening of fitness differences also affects beneficial mutations: sectors of faster-growing clones initially widen more slowly than predicted by non-mechanical models (Fig. 5a)—a feature that had previously been noted in yeast colonies but not...
traced back to its mechanical origin\textsuperscript{34,41,45}. As a result of this fitness screening, we expect faster-growing clones to take longer to establish, thus slowing down adaptation\textsuperscript{62,69}.

In addition to our findings in \textit{S. cerevisiae}, we also observed fitness screening in colonies grown from the rod-shaped bacterium \textit{Escherichia coli} (Fig. 5b). \textit{E. coli} differs from \textit{S. cerevisiae} in a number of characteristics, such as cell size, shape and modes of proliferation (budding versus cytoskeletal cell division).

The robustness of fitness screening is further demonstrated by cell-based simulations that model symmetrically dividing ellipsoidal cells (Fig. 5c). All investigated cell shapes and division types exhibit the hallmarks of a crowding-mediated fitness screening. For a fixed growth layer depth, even the magnitude of the effect is conserved in cell-based simulations (Fig. 5d). This finding corroborates our notion that the mesoscopic features of fitness screening are independent of microscopic cell properties.

Discussion

Our results demonstrate that evolutionary processes have a mechanical basis in crowded populations. Growth-induced pushing forces generate long-range correlations in cell motion, thereby screening fitness differences. Consequently, selection is less efficient in weeding out less fit mutants from the expanding population front than predicted by non-mechanical models. Less fit mutant clones thereby produce many more offspring than expected. These persisting mutants can contribute to population resilience in the face of environmental deteriorations, such as the application of a drug. In addition, the larger total number of mutant offspring might increase the chance of mutant survival after a population bottleneck, or the acquisition of subsequent mutations compensating the fitness cost\textsuperscript{34,41}.

Fitness screening has three necessary ingredients: the front has to exhibit an effective surface tension; the average motion of all cells has to be perpendicular to the local front line; and domain boundaries have to be sharp compared with the characteristic length scale of boundary interaction. Whereas conventional non-mechanical models fail to generate this combination of ingredients, all ingredients inherently result from the collective nature of mechanically driven population expansion generated by the growth and division of cells within a growth layer near the population frontier. In addition, the models presented in this work, fitness screening can also be described via an overdamped continuum mechanics model. Using this approach, a parallel study by Giometto et al.\textsuperscript{31} corroborates our results and demonstrates its implications for adaptation in oscillating environments.

Fitness screening may be altered beyond the simple scenario presented in this paper. For example, a modulation of nutrient penetration into the colony could reduce the growth layer depth\textsuperscript{34,41,44}, decreasing the effective surface tension and strength of fitness screening. An inherent surface tension can also arise in cellular populations independent of cell growth, originating from forces such as cell–cell adhesion\textsuperscript{10,43} and cell contractility\textsuperscript{46–48}.

Fitness screening can be viewed as a form of cooperation\textsuperscript{51,49,50}; in crowded populations, faster-growing cells tend to push along nearby slower-growing cells, as dictated by the laws of mechanics. This mode of cooperation merely requires physical cell–cell forces near by slower-growing cells, as dictated by the laws of mechanics.

In experiments with \textit{S. cerevisiae}, we expect faster-growing clones to take longer to establish, thus slowing down adaptation\textsuperscript{62,69}. Extensive experimental evidence from the laboratory of Andrew Murray (Harvard University). yMM8 and yMM9 both have a point mutation, \textit{CYH2}:\textit{cyh2-Q37E}, causing resistance to the translational inhibitor cycloheximide (Table 1). yJK22 is similar to yMM8, but featuring a \textit{HogmX} cassette conveying resistance to the drug hypomycin B, in addition to the \textit{CYH2} mutation. yJK22 was constructed from yJK111 via integration of the plasmid pAG32 (see https://www.addgene.org/35122/ for details). Note that the ‘mutant’ (yJK22) and ‘wild-type’ (yMM9) designations used in this paper refer to the mimicked scenario of a costly drug resistance mutation and not to the genotypes of the involved strains.

\textbf{Yeast competition assays.} Unless otherwise specified, experiments were based on linear inoculations competing the strains yMM9 (‘wild-type’) and yJK22 (‘mutant’). Of these strains, only yJK22 is susceptible to the translational inhibitor cycloheximide, allowing us to impose a growth-rate deficit on yJK22 cells. All experiments were conducted in the presence of 90 nM cycloheximide, resulting in a fitness difference of \(s = -0.06\), as quantified previously\textsuperscript{7}.

\(s\) is defined via the ratio of strain-specific doubling times, \(k\), as \(s = k_{\text{mutant}}/k_{\text{wild-type}} - 1\). Neutral controls were conducted by substituting yJK22 with yMM8, with yMM8 exhibiting growth rates equal to yMM9.

Linear colonies for the wide-sector assays were inoculated by running a gravity-pulled droplet (10 \(\mu\)L of concentrated yJK22 culture (\(OD_{600} > 10\)) across an inclined agar plate (YPD; 1% (w/v) agar). Subsequent air drying produced a dense, continuous layer of cells at the periphery of the wetted area (a ‘coffee stain effect’). To produce mutant (yJK22) sectors flanked by wild-type (yMM9) regions (see Supplementary Fig. 1), sections of the linear yJK22 inoculum, several hundreds of micrometres apart, were removed by rolling a sterile glass bead across the front, lifting cells from the surface. The resulting gaps were filled by concentrated yMM9 culture. We conducted competition assays in replicate on a total of eight linear colonies on four plates, each of which had an average number of five sectors. Generally, colonies were grown in an incubator and temporarily transferred to the microscope for imaging. Time-lapse measurements were made, and spatial and temporal resolution were conducted in a temperature-controlled chamber (ibidi).

\textbf{Imaging and analysis.} Sector boundaries serve as a record of past front compositions, allowing us to infer the spatiotemporal progression of a clone from a single fluorescence microscopy image\textsuperscript{7}.

Mutant sectors were imaged on a total of eight linear colonies on four plates, each of which had an average number of five sectors. Generally, colonies were grown in an incubator and temporally transferred to the microscope for imaging. Time-lapse measurements were made, and spatial and temporal resolution were conducted in a temperature-controlled chamber (ibidi).

\textbf{Evaluation of }\(w\)\textbf{ and }\(w_c\). The width of a sector \(w(y)\) was measured perpendicular to the axis of colony expansion and as a function of position \(y\) along the same axis. Measurements were taken from the complete sector at the end of the experiment by tracing the sector boundaries via a custom-built, semi-automated algorithm developed in MATLAB (R2015a). To compare the lengths and widths of individual sectors, it is necessary to align them to a common, globally defined reference width. Throughout this work, sectors in assays starting with a wide section of the slower-growing cell type were aligned to the characteristic width \(w_c\), below which the observed sector shape departed from its anticipated linear behaviour. To evaluate \(w_c\), we applied a linear fit to the initial straight boundary trajectory and calculated the difference \(\delta(w)\) between the observed sector width and the linear fit for the sectors shown in Supplementary Fig. 2. This value is independent of sector alignment and can be averaged over all high spatial resolution measurements. Subsequently, \(w_c\) can be inferred via the choice of a sector at which \(\delta(w)\) exceeds a predefined threshold \(\delta_s\). Here, we chose \(\delta_s = 5\mu\text{m}\). The so-obtained alignment of sectors is robust against variations of \(\delta_s\) by a factor of 2 (Supplementary Fig. 3b).

\begin{equation}
\frac{\mu c}{\delta w}\quad\nabla\frac{\nabla}{\nabla}
\end{equation}
Single-cell sector assays. Experiments tracking the fate of sectors originating from single cells (Fig. 4) were set up in a fashion similar to the wide-sector assays described above. Here, colonies were inoculated from a mixture of YM9M containing a small fraction (5%) of yJK22. Cell separation was ensured by briefly sonicating the mixed culture before deposition. For the neutral control, we ran parallel experiments substituting yJK22 with YM8H. To verify neutrality, we repeated these experiments, inverting the ratio of YM8 to YM9M. We initially observed a total number of approximately 3,500 sectors (in a total of 12 colonies), which, finally, declined to 0 within the course of the experiment. Front persistence probabilities (P_	ext{persistence}) were obtained by identifying fluorescent regions via a custom segmentation algorithm, based on local intensity gradient peaking, developed in MATLAB (R2015a).

Evolutionary rescue assays. In a subset of colonies of the single-cell sector assay, we halted wild-type growth by the application of hygromycin B. To this end, a line of 50 mM hygromycin B in medium (70 μl total volume) was pipetted approximately 5 mm in front of the leading colony edge. The drug then dispersed via diffusion and local concentrations were sufficient to completely halt wild-type propagation while allowing for continued growth of the mutant. The colony depicted in Fig. 4c,e exhibited a total of eight sectors still at the front after four days of growth (the time point of hygromycin B application), all of which exhibited resurgent growth at day 6.

High-curvature inoculations. Experiments with high initial curvature (see Fig. 2d) were initialized by first placing a small droplet (1 μl) of culture on an agar substrate followed by air drying. A second droplet of equal volume and from the same culture was subsequently placed adjacent to the first droplet, such that its peripheral ring of cell deposition intersected that of the first droplet. This series of cell depositions created an initial front with a high radius of curvature at the intersection points.

Cell tracking. In single-cell-scale time-lapse videos, individual cells were tracked visually using custom MATLAB (R2015a) scripts. For the images in Fig. 2b (and accompanying Supplementary Video 9), t = 0 min is defined as approximately 3 days after inoculation. To assess the relationship between cell velocity and local curvature, i, in curbed inoculations, the instantaneous velocities were computed from individual cell tracks as a function of k, which was obtained from the smoothed front position y(x) of the colony as:

\[ x = \frac{dy}{dx^2} \times (1 + |dy/dx|)^{-\frac{1}{2}} \]  

(1)

Particle image velocimetry (PIV). To find the local speed inside yeast colonies, Supplementary Video 5 of growing S. cerevisiae fronts was analysed using PIV with the ImageJ PIV plugin. Seven pairs of images, each taken one minute apart, were analysed with a window size of ~8μm. To find the local speed as a function of the distance from the (moving) colony front, images of the front at each time point were binarized to find the position of the front. Displacements in the growth direction were averaged along the direction perpendicular to the growth direction, shifted to account for the moving front position and then averaged across image pairs to produce Fig. 3f. Supplementary Video 5 shows the expanding colony front approximately 2 h after the start of expansion when the colony was still very thin. This allowed for the tracking of features deep within the colony. Note that at this time point the growth layer had not yet reached its final profile. Refer to Supplementary Fig. 7 for data on the fully established growth layer.

E. coli colonies. Colonies of E. coli were grown on plates of M9 minimal media containing 2% agar from a 1:1 mixture of strain MG1655 and the derivative S102 (fluorescently labelled; t = 0.04 ± 0.03, as measured from n = 17 colony collision events), which are non-motile under the given conditions. The colonies were imaged after five days.

Verification of constant relative fitness. The relative fitness between strains with the wild-type and resistant alleles of CHY2 for the group of strains used in this study (with YHK11 as the direct ancestor of yJK22) has been previously quantified as a function of the cycloheximide concentration using liquid culture competition and colony collision assays. The concentration of 90 nM used in this study corresponds to a fitness effect of 8 = C_{cyh2r}/C_{cyh2h} = 1 = 0.06, with C_{cyh2h} and C_{cyh2r} indicating the doubling rates of the susceptible and resistant strains, respectively. To ensure that the position of the front remained constant over the course of our experiments, we measured the front velocities of colonies composed of the individual strains (as calculated from the width increase of linear monoclonal colonies). Supplementary Fig. 8a-c shows how front speeds generally decline over time as nutrients become depleted. However, the ratio of expansion velocities (Supplementary Fig. 8d)—the basis for competition—remains constant. Even though expansion speed assays might not be equivalent to measurements in liquid culture (see ref. 13), here, they serve to verify the consistency of relative fitness in conditions equivalent to those applied in our competition assays.

Description of the surface tension model. Here, we describe a phenomenological surface growth model, which we refer to as the ‘surface tension model,’ where the population is modelled as a one-directional surface with height h(x) that moves forwards at velocities that are normal to the surface (Supplementary Fig. 11a). Each point on the surface moves at a velocity, v, that is modulated by the local front curvature, k, via an effective ‘surface tension’, T:

\[ v = v_\text{tip}(1 + T x) \]  

(2)

where \( v_\text{tip} \) is the cell-type-specific velocity of a flat front.

Assuming that flat-front velocities are proportional to the cellular growth rate of cells at the surface (Supplementary Fig. 11b), forward velocities of a front composed purely of wild-type or mutant cells have separable selection (\( v_\text{tip} = v \) for the wild type; \( v_\text{tip} = (1 + \sigma) v \) for the mutant) and surface tension (1 + T x) contributions:

\[ v = v_\text{tip}(1 + T x) \]  

(3)

where \( v_\text{tip} \) is the velocity of a flat front composed of wild-type cells. Equation (3) describes the competition of adjacent phenotypes, in which case \( v_\text{tip} \) changes discontinuously from \( v_\text{tip} = v \) to \( v_\text{tip} = (1 + \sigma) v \) across the wild-type/mutant boundary. The wild-type/mutant boundary evolves in time, following the surface normal of the propagating front.

Parameterization of the surface tension model. To parameterize the surface tension model, we compared the decrease in width of initially wide deleterious clones obtained from the model with the experimental data shown in Fig. 2L. Initializing the surface tension model with a slanted front designed to be consistent with the null model (Supplementary Fig. 9), we found a funnel-like tapering of the deleterious clone, as for the experiments. The spatial extent of this deleterious tapering varied with surface tension, T, and we found that the model best matched the experiments with a surface tension of T = 25 μm (Supplementary Fig. 13).

Stochasticity in interface models. To incorporate stochasticity into the surface tension and null models, we added diffusive boundary fluctuations along the front with a diffusion coefficient D. The mean-square displacement of boundaries was therefore \( \Delta x^2 = \Delta D x \) in a neutrally growing boundary. Resultants were matched experimentally observed persistence best for \( D = 0.025 \mu m^2/s \) (see Fig. 4D), which is significantly lower than what we had previously measured from single-cell traces (\( D = 0.3 \mu m^2/s \) calculated from a diffusive fit to the data in Fig. 3f of ref. 4). One potential reason for this difference is that the persistence times (the basis for our fit) are governed by the fluctuations of clone width rather than those of the individual boundary position in real space. It is conceivable that the assumption of independent diffusive behaviour of the sector boundaries breaks down at small length scales due to the granularity of the system. Indeed, the single-cell traces shown in Fig. 2b suggest a certain degree of correlation in the positional noise of neighbouring cells, resulting in a width dependence of sector width fluctuations.

Description of the cell-based model. The cell-based model was based on simulations developed in ref. 42, where budding cells are modelled as two-directional frictionless disks (mother and bud) that are fused together. In this model, cells increase in area at a rate \( \gamma \), by bud expansion (equation 6 of ref. 42), move according to overdamped dynamics with a repulsive spring (equations 7 and 8 of ref. 42) and interact via repulsive spring forces with elastic modulus \( k \) (equation 9 of ref. 42), as described in ref. 42:

\[ \dot{a}_i = \gamma_i a_i \]  

(4)

\[ r_i = \mu_i \]  

(5)

\[ \theta_i = \frac{m_i}{I_i} \]  

(6)

\[ V = \sum_{i,j} k_i j \delta_{ij}(\theta_i - \theta_j) \]  

(7)

where \( a_i, \gamma_i, \mu_i \) are the area of cell i, \( \gamma_i \) and \( \sigma_i \) are the diameter of the mother and bud, \( r_i \) is the cell position, \( \theta_i \) is the cell orientation, \( m_i \) is the cell mass, \( I_i = \frac{1}{2} m_i \) is the moment of inertia, \( \delta_{ij} \) is the Kronecker delta, \( \delta(\theta_i - \theta_j) \) is the overlap between lobes k of cell i and l of cell j, and \( \theta_i \) is the unit step function. Each mother lobe has the diameter \( \sigma_i \), and \( \mu_i \) is the number of mother lobes. Equations of motions are integrated using a third-order Gear predictor-corrector algorithm. Growth progresses while \( a_i, \sigma_i \) and\( \sigma_i \) culminates in division. After division, both new cells are oriented inwards (axial division), with buds facing each other.
In this study, we further developed the simulations described in ref. 42 so that cell populations grew in two-directional colonies, where only cells in a ‘growth layer’ of depth $\lambda$ near the edge actively grew. To calculate the growth layer depth, we first found all cells in the front. We then classified cells as being in the growth layer if they were within $\lambda$ of any cell in the front. The resulting dynamics is shown in Supplementary Video 6 for the neutral case ($\sigma=0$) and Supplementary Video 2 for deleterious clones ($\sigma=-0.06$), both with $\lambda=45\mu m$.

**Boundary conditions in the cell-based model.** We used three different boundary conditions, as illustrated in Supplementary Fig. 12. In Fig. 3h, we used open boundary conditions so that the colony grew radially in all directions. In Fig. 3c,f, we used periodic boundary conditions in the horizontal direction. In Fig. 3i, we used ‘wedge’ boundary conditions, in which mutants occupied a large, flat portion of the front and were bordered by regions composed of wild-type cells that were rotated at a tilt angle consistent with the non-mechanical model.

**Parameterization of the cell-based model.** To obtain a value for $\lambda$, we compared the decrease in width for the initially wide deleterious clone with that from the *S. cerevisiae* experiments (shown in Fig. 1b). Initializing the cell-based model with a slanted front, we found a funnel-like tapering of the clone where the tapering length-scale increased with growth layer depth $\lambda$. Using a cell diameter of $\sigma=5\mu m$, the cell-based model best matched the experiments, with $\lambda=45\mu m$ (Supplementary Fig. 14). To constrain the number of cells included in time integration, we fixed the position of cells further behind the front than 80$\mu m$. Note that the growth layer used for cell-based simulations refers to an idealized ‘effective’ growth layer. It might therefore not directly reflect the depth up to which cell movement was observed in experimental colonies where additional factors, such as out-of-plane growth, nutrient absorption and diffusion, or cell elasticity, influenced the details of growth-layer dynamics.

We used a mobility of $\mu=4\times10^{-5}k\cdot m/s$ and a time step of $\Delta t=5\times10^{-7}$s for time integration. In Supplementary Fig. 14, we used an initial box width of 4 mm and initial clone mutant width of 2 mm. We used averaged growth rates for mutant time integration. In Supplementary Fig. 14, we used an initial box width of 4 mm and initial clone mutant width of 2 mm. We used averaged growth rates for mutant

\[
\frac{dx}{dt} = \lambda_x \frac{x}{x+1} - \lambda \nu \frac{x}{(x+1)^2}
\]

where $\nu$ represents the neutral-type and mutant concentrations, respectively, $D$ is the diffusion constant, $\lambda$ is the replication rate of the wild type and $s$ is the selective coefficient associated with the mutant.

The simulations were initialized with a slanted profile, as depicted in Supplementary Fig. 9b. Boundary conditions were implemented to maintain the angle $\alpha$ of the wild-type profile at the edge of the simulation box.

**Reporting summary.** Further information on research design is available in the Nature Research Reporting Summary linked to this article.

**Code availability.** Custom codes used in this study are available at https://github.com/Hallatscheklab/collectiveemotion.

**Data availability.** Imaging data used in this study are available at https://figshare.com/projects/Kayser2018_NatEE/55727.

Received: 6 March 2018; Accepted: 23 October 2018; Published online: 3 December 2018

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Reporting Summary

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| ☑   | An indication of whether measurements were taken from distinct samples or whether the same sample was measured repeatedly |
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| ☑   | For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes |
| ☑   | Estimates of effect sizes (e.g. Cohen’s \( d \), Pearson’s \( r \)), indicating how they were calculated |
| ☑   | Clearly defined error bars |
| ☑   | State explicitly what error bars represent (e.g. SD, SE, CI) |

Software and code

Policy information about availability of computer code

Data collection  
Microscopy images were collected using Zeiss ZEN (2012, blue edition).

Data analysis  
Data was analyzed using custom algorithms in MATLAB (R2015a and R2018a) from Mathworks. Image processing was performed using Fiji.

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors/reviewers upon request. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Research guidelines for submitting code & software for further information.

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Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

| Sample size | Number of individual sectors imaged was limited by the maximum number processable in the given time frame (12h imaging intervals). |
|-------------|-------------------------------------------------------------------------------------------------------------------------------|
| Data exclusions | Sectors not exhibiting a phase of straight boundary trajectories (due to insufficient initial size) were excluded from the analysis. |
| Replication | We conducted competitions assays in replicate on a total of 8 linear colonies on 4 plates, with each having an average number of 5 sectors. For 9 sectors from 2 separate colonies we further evaluated the position dependent width w(y). |
| Randomization | This was not relevant in this work since randomization can have no effect on observables. |
| Blinding | Blinding was not relevant in this work since no group allocation was performed. |

Reporting for specific materials, systems and methods

Materials & experimental systems

| Materials & experimental systems | n/a | Involved in the study |
|----------------------------------|-----|-----------------------|
| Unique biological materials      | ☒   | ☒                     |
| Antibodies                       | ☒   | ☒                     |
| Eukaryotic cell lines            | ☒   | ☒                     |
| Palaeontology                    | ☒   | ☒                     |
| Animals and other organisms      | ☒   | ☒                     |
| Human research participants      | ☒   | ☒                     |

Methods

| Methods | n/a | Involved in the study |
|---------|-----|-----------------------|
| ChIP-seq | ☒   | ☒                     |
| Flow cytometry | ☒   | ☒                     |
| MRI-based neuroimaging | ☒   | ☒                     |

Unique biological materials

Policy information about: availability of materials

Obtaining unique materials

Unique yeast strains used in this study are readily available from the author