Learning is a fundamental source of behavioural individuality

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ABSTRACT

Learning and memory are essential components of our individuality and sense of self[1]. While it is established that behaviour can vary between genotypes and across genetically identical individuals, it remains unknown how much of this variation stems from learning compared to environmental stochasticity or genetics[2–12]. To answer this fundamental question, we measured individual visual behaviour in thousands of flies in the presence and absence of learning. Even when the flies were genetically identical, raised under the same conditions and exposed to the same environment, we found that the flies which learned were more individually diverse than the non-learning flies. We could quantify, model and then recreate this phenomenon in computer simulations. The emergence of behavioural individuality in simulated agents was most consistent with the individuality of real flies only once we enabled them to learn through reinforcement. Our study establishes that learning is a third fundamental source of individuality, besides genetic and environmental variation.

Significance statement

It is well-established that differences in spontaneous behaviour between individuals stem from genetic (*Nature*) and environmental factors (*Nurture*). In studies of individuality, ability to learn has been so far considered as a phenotype, a direct product of Nature and Nurture. In this study, we show experimentally that despite genetics and environment, individuality in behaviour can be shaped directly by learning. We thus establish that the process of learning is itself a third fundamental diversifying force, sufficient to induce divergence in individual behaviour.

Introduction

Behavioural individuality is an inevitable, fundamental aspect of life. Even identical twins who share the genome and life history eventually diverge into distinct individuals[12–14]. In every individual, behaviour is initially shaped by deterministic, genetic factors[15–18] followed by stochastic, environmental or molecular events that occur during development and early life[6,9,18–21]. These classical sources of individuality have been studied extensively[22]. Divergence of individuality after development is less well understood. Behaviour can change during adult life due to life experience, either through learning and memory, or learning-independent process[10,22–27]. Traditionally, variability in learning has been attributed

to differences in genotype and environment. As such, it is considered important but not necessarily fundamental to individuality. An alternative view is that the learning process itself—if kept consistent across individuals—can directly drive behavioural divergence, regardless of genetic or environmental factors. The challenge in understanding the role of learning in individual behaviour lies in its entanglement with classical sources of individuality (1A). The ability to learn and remember is known to vary across individuals[1,22,26,28,29], genotypes[30,31], environments[32–34], and even maternal status[35–37]. Because of that, studying the emergence of individuality in the context of learning requires precise, trans-generational experimental control over genetic and environmental factors, and over learning experience. While essential, achieving this level of control is technically extremely challenging. As a result, most previous studies have focused on individuality in spontaneous behaviours that do not depend on learning.[5,7,9,12,25,27,38–41]. In the few studies that do address learning, the influence of traditionally studied sources of individuality is rarely (if ever) fully accounted for—mainly due to technical limitations and necessary complexity of the experimental design.[4,11,26,31,42–46]. Consequently, the role of learning as a putative fundamental source of individuality remains unexplored.

In this study, we address this question. We take on the technological challenge by leveraging the utility of the Drosophila model system. Using flies we could achieve extreme uniformity in experienced environments across thousands of individuals (Table S1) and in 90 different genetic backgrounds. We then developed a highly parallelized learning behaviour assay that scales up test throughput by hundred-fold compared to the current state-of-the-art, while maintaining the uniformity of environmental momentary experience across individuals even during the behaviour test. We analysed, modelled, and computationally recreated the time-resolved experiences of individual flies as they made nearly half a million decisions, with or without the influence of learning. This allowed us to disentangle and quantify the independent contributions of all measurable sources of individuality—genetics, developmental and life experience, momentary experience, and learning (Figure 1.A). Our findings showed that behavioural diversity can arise solely from learning, even when genetic and environmental factors are held constant, demonstrating that the process of learning is a third, fundamental driver of individuality.

Results

Distributions of individual behaviours are altered by learning

Both genotype and environment affect the behaviour. However, even in experimentally controlled environments, a genotype will interact with the inevitable environmental and molecular stochasticity. This interaction is experienced individually, mostly during the development, resulting in behavioural measurements that are normally distributed within genotype[15,47,48]. Gaussian variance has thus been reliably used as a read-out of the extent of individuality expressed by genetically identical individuals raised in identical environments[7,15]. We asked if the behavioural variance within genotype is larger in the presence of learning. This required testing learning-dependent behaviours in all individuals at the same time, to avoid confounding effects of individual learning experience.

To do so, we designed a platform that implements operant conditioning assay that can be parallelized across 64 freely behaving individual *Drosophila melanogaster* (Figure 1.E). The system takes input from real-time video tracking of individuals (Supplementary Video S1-2). This is then used for closed-loop coupling of visual stimuli and delivery of a mild foot shock to individual flies upon an undesired behaviour (Figure 1.B-E, Supplementary Figure 1.A). Operant conditioning paradigms are based on colour and place avoidance learning in a Y-shaped arena ("Y-maze"; Figure 1B-D; see Methods). The conditioned task

was to spend less time in the arm or colour that was associated with shock (*task performance* or $T_{shocked}$, Figure 1D,F, Supplementary Figure 1.A; see Methods). In the control group, the shock was not applied, allowing the flies to behave spontaneously, without a need to learn (Figure 1D).

We used this assay to test the performance of known learning- and sensory-deficient flies (N=128) as well as 88 genetically diverse wild-type genotypes (N= 5632)[16,49]. We ensured strict multi-generational control over their life-history (Table S1) and across two independently reared replicates of each genotype (Figure 2.B,G, Supplementary Figure 3). Individuals from each replicate were then split into **conditioned** and **control** group (Figure 1.A) and we tracked their momentary task-relevant behaviours, including ∼half a million individual decisions (i.e., switches from one arm in the Y-maze to another arm; N = 497450; Video S2). The difference between the genotype-averaged control and conditioned task performance successfully emulated the traditionally used group-average measures of learning performance Figure (1.G, Supplementary Figure 1.C). Interestingly, individual measurements revealed a reduced inter-individual variability of $T_{shocked}$ among flies that are unable to learn and form memories (Supplementary Figure 1.A) and a diverse range of expressed individuality across the wild-type genotypes[16,50,51] (Figure 1.H, Supplementary Figure 2.A). Despite the strict experimental control (Table S1, Supplementary Figure 3.A-H, Table S4), behavioural variability within groups of genetically identical individuals was non-negligible (Figure 1.H, Figure 2.A-B), pointing to at the first glance to likely high level of developmental stochasticity. Curiously though, not only the means and variances, but also the shapes of the distributions of individual behaviours varied reproducibly between genotypes, particularly in the conditioned group (Figure 2.B-C, Supplementary Figure 3.A). Although they shared developmental experience and genetics with the control flies, conditioned flies expressed within-genotype individuality in a non-Gaussian and multi-modal fashion (Figure 1.H, Figure 2.B-C). These findings indicated that both genotype and learning play a role in shaping individual behaviour. We asked whether this modified expressed individuality in conditioned flies is sourced stochastically.

Task performance (Figure 2.A,E, Supplementary Figure 4.A,F,K; Supplementary Text) and other task-relevant behaviours were significantly different between control and conditioned flies (Supplementary Figure 4.A-E). They also varied consistently over time (Supplementary Figure 4.G-J), across conditioning paradigms (Supplementary Figure 1.C, Table S3) and sexes (ANOVA $p = 0.537$, Sum Sq. = 0.004, Table S2). The observed shapes of their distributions were also consistent across replicates (Exact general independence test, $p > 0.05$ for 83 out of 88 genotypes; see Methods; Figure 2.B, Supplementary Figure 3.A). Altogether, this indicated that the shapes of the distributions were altered through a deterministic, likely genetic control over expressed individuality.

Learning exposes residual individuality

To dissect their contribution to the unusual non-normal display of individuality, classical characterization of expressed individuality using genotypes' mean and variance was not appropriate. Instead, we introduce a measure of *differential entropy* (H) to capture the extent of expressed individuality within a group of flies (see Methods for details). To compare the expressed individuality between groups we use Hellinger distance. This way we need not assume Gaussian probability distributions of individual behaviour.

The entropy of $T_{shocked}$ was highly genotype-dependent (Resampling test, parametric $p < 2.2e^{-16}$ resampled $p = 1e^{-06}$, Supplementary Figure 5.A). It also correlated between conditioned and control environments (Pearson's product-moment correlation between $H_{control}$ and $H_{conditioned}$ $r = 0.232$, $p = 0.03$, n= 88 genotypes, Supplementary Figure 5.A). This indicated that most of the observed individuality is

sourced from processes that preceded the behaviour assay, such as the development and life history. The difference in expressed individuality between replicates within genotype was consistently low in both conditioned and control flies (Mean $Hellinger_{\text{control}} = 0.06$, Mean $Hellinger_{\text{conditioned}} = 0.05$, T-test $p = 0.256$, $n = 88$, Supplementary Figure 5.C). This confirmed that the experimental control over environment was tight and unlikely to be the source of observed individuality. On the other hand, how much the expressed individuality changed between the conditioned and control flies varied substantially across genotypes (Resampling test, parametric $p < 2.2e^{-16}$, resampled $p = 1e^{-06}$, Figure 5.B). Individuality expressed within a genotype did not translate linearly from spontaneous to conditioned behaviour (Figure 1.H, Figure 2.A-B, Supplementary Figure 5.A). This pointed to genetic control over individuality, one that interacts with the process of learning.

Taken together, these results show that the observed behavioural variability is sourced from a mechanism that is neither random nor individual, and yet results in excess individuality in flies that learn. This putative mechanism must be i) systematically shared between all flies of the same genotype, ii) different between genotypes, and iii) expressed individually.

To capture this unusual, additional expression of individuality, we introduce a measure of *"residual individuality"* (H_{resid}), calculated as Shannon entropy of a probability distribution after scaling the distribution within its own range. This measure nullifies the variability induced by factors shared across all groups of flies, including stochasticity in rearing environments, genotype, and their interaction (Supplementary Figure 6; Methods). Then, we use three different measures of distribution shape divergence to dissect the source of the residual individuality. ΔH_{resid} *Learning* quantifies the change in expressed residual individuality (H_{resid}) of a genotype due to learning (Figure 2.D,H). ΔH_{resid} *Experimental* reflects the change in H_{resid} caused by experimental noise distributed systematically over individual flies during the behaviour assay (Figure 2.G). ΔH_{resid} *Genetics* captures the genetic contribution to the expressed residual individuality observed during the behaviour assay (Figure 2.F); see Methods for details on all three measures).

We found no difference in ΔH_{resid} *Experimental* between conditioned and control groups (ΔH_{resid} $Experimental_{\text{control}} = 2.08$, ΔH_{resid} $Experimental_{\text{conditioned}} = 1.85$, T-test $p = 0.206$, $n = 88$, Figure 2.G). Experimental noise during behaviour was therefore not causing the change in the distributions of behaviours in flies that learned. On the other hand, ΔH_{resid} *Learning* of T_{shocked} differed significantly across genotypes (Resampling test, parametric $p < 2.2e^{-16}$, resampled $p = 1e^{-06}$; Figure 2.D), and evolved with every made decision within a genotype (Figure 2.H). The presence of learning thus contributed to the change in residual individuality. This was unexpected. Genetics is usually expected to be the only deterministic variable that can shape the amount of expressed individuality in a controlled environment that is shared by all individuals. In isogenic populations, the effect of genotype on individuality is assumed to be absent except for its effect on phenotype variance that is realized during the development[12,15]. Yet, even after discounting mean and variance effects, we found that the shapes of the distributions of individual task performance were significantly more divergent between genotypes if the flies were conditioned (ΔH_{resid} $Learning_{\text{control}} = 0.70$, ΔH_{resid} $Learning_{\text{conditioned}} = 0.83$, T-test $p < 2.2e^{-16}$, $n = 88$ genotypes; Figure 2.D). This was true for other behaviours as well (Supplementary Figure 3D-H, Supplementary Figure 7.A) and could not be explained by residual heterozygosity[49] (Supplementary Figures 7.A, 8.A, 3.A, Figure 1.H) and individual or genotype-specific response to shock (Supplementary Figure 7.C, 8.A-B, Supplementary Text).
Residual individuality thus cannot be attributed solely to the effects of genotype without considering an

independent effect of learning. And yet, there was a lack of correlation of residual individuality in task performance with residual individuality in learning score. Similarly, there was no correlation between residual individuality in task performance with genotypes' mean learning score. Therefore, genetically encoded learning ability could not explain genotype-specific residual individuality in task performance (Supplementary Figure 8.A).

Learning amplifies the "butterfly effect" of individual experience

Despite the efforts to perfectly control and parallelize experienced environments across every individual, in practice, perfect control over biological, freely behaving agents, is rarely achievable. In a non-uniform environments, momentary experience of freely behaving individual depends on its previous decision (Figure 1.A). This is practically intractable in a natural environment, but the Y-maze arena minimizes the diversity of unique experiences and decisions a fly can make and allows us to track and enumerate them (Figure 1.A, Figure 3.A-C).

We aligned the discreet decisions across thousands of individuals, and asked do individual decisions and momentary experiences result in the divergence of individual behaviour. We found that despite our efforts to parallelize the environments, most minimal variations—such as the fly's initial position in the Y-maze when it made its first decisions—shaped unique learning trajectories that amplified individuality over time. Initial conditions are experienced seemingly stochastically by individuals, but their distribution is genotype specific. With more made decisions, learning, rather than innate bias, becomes the dominant factor influencing individual behaviour (Supplementary Figure 9.E-F). Learning, being a probabilistic process, continuously reshapes future experience through individual decisions, adding an additional layer of behavioural diversification, even in individuals that shared the same biases (3.A,C,E). We could directly observe the unravelling clash between learning and bias as it played out in the form of persistent colour-switching behaviour. This was further amplified in flies with high learning score and unfavourable bias (Figure 3.C,E).

Learning diversifies individual behaviours

While we did not track behaviour throughout the entire life of a fly, the design of our experiment allowed us to dissect the effects of life experience prior to the learning task from the effects of momentary experience during the task. We asked how extensive is the effect of the entire life's experience on individual momentary behaviour, compared to the effect of genetics or learning during the task?

To quantify the relative contributions of different sources of individuality, we turned to modelling of observed behaviours. We tested how well our models capture and represent the observed behaviours by using them to simulate behaving flies and then comparing their simulated behaviour to the behaviour of observed, real flies.

Simulated flies incorporate a range of behavioural parameters (Table S6). They decide how long they want to stay in the current arm of a simulated Y-maze, and whether they want to turn left or right when leaving the current arm. The left-right turns are sampled from a Bernoulli distribution with a time-dependent rate (Methods). The decision to leave the arm is made by sampling the escape duration (time to leave one arm) from a log-normal distribution with a time-dependent mean and stationary variance. The mean of their escape duration distribution depends on the colour of the arm and the presence of shock at the time point t. Importantly, the mean escape duration and the rate of turning left or right can change with a reinforcement learning rule, which depends on the presence of shock.

To assess the effects of genetics, learning, and individual life experience, we systematically exclude one or none of these factors in four different behaviour models. First, all individual flies of the same genotype

have identical parameters, but different genotypes have different parameters (*no individuality* model), thereby omitting the effect of any individual experience obtained prior to expressing a behaviour in the task. Second, the i-th behavioural parameter of an individual simulated fly is sampled from a normal distribution with genotype-dependent mean ($m_i^{(g)}$) and standard deviation ($s_i^{(g)}$). These simulated flies have individuality in the sense that their initial behavioural policies differ from one another, because no two flies have the exact same parameters (*individuality* model). In other words, simulated flies start the task as if in addition to their genetics and learning, their life experience prior to the task (including stochastic processes during development) had shaped their individual behaviour. In the third, *no learning* model, parameters are sampled as in the *individuality* model, but the learning rate is set to zero, such that the presence of shock or colour induce no lasting change on their behaviour beyond the immediate effect of changing the escape duration (effectively simulating only the shock response). Fourth and last is the *no genetics* model, where the parameters are sampled from a distribution per individual, but there is no genotype dependence of the means and standard deviations of the parameter distributions. This way the effect of genetics is omitted, including its effect on developmental variability.

Best fit was found for the *individuality model*, followed by *no learning* and *no genetics* model. *No individuality model* failed to recapitulate the observed behavioural individuality (Figure 4.A-B). Particularly striking was the mismatch in the distribution of the number of decisions made by flies simulated without individuality compared to the actually measured distributions of the number of decisions made by real flies (Supplementary Figure 10).
Compared to whole-life experience, the effect of momentary learning on individual behaviour was smaller, and for some genotypes seemed irrelevant (Figure 4.A). However, we found that the model without learning could not capture the temporal dynamics of many observed behaviours (Figure 4.C). Interestingly, the excess of observed non-Gaussian distributions of individual behaviour in observed conditioned flies was recreated in simulated flies only if they were allowed to learn (Figure 2.C) and despite all parameters of the model being drawn from normal distributions. This reaffirmed that the residual individuality emerges from the very process of learning.

Lastly, repeated simulations revealed that the genotype distribution of simulated task performance matches the observed behaviours better for the model with learning (Figure 4.D, Supplementary Figure 10, 11). Altogether, these results decisively demonstrate that learning is not just influential—it is a fundamental driver of behavioural individuality.

Genotype does not determine or predict individual behaviour

The fitted models allowed us to estimate how the momentary task-relevant behaviour of a given fly differs from other flies, or how it compares to other moments in time (Methods). We found that the individual behaviour had changed significantly during the experiment (Figure 4.E-F) and that this change was significantly bigger in the flies that learned (Figure 4.F, Supplementary Figure 12). A visualization of these differences with multidimensional scaling revealed that the behaviour of individual flies does not cluster according to genotype throughout the experiment (Figure 4.E). This lack of clustering was also confirmed with a classification analysis: using a long-short term memory (LSTM) classifier applied to the behavioural data of individual flies, we could predict the genotype in only 3.4% of the cases (Chance level: 1.1%, 4-fold cross-validation).
Although our models confirmed that variation in genotype is essential for behavioural diversity, they show that it determines practically a stochastic fraction of any given individual's behaviour.

Discussion

In the most comprehensive and powerful study to date on the sources of behavioural individuality, we show that learning—independent of genetics and life history—plays a direct role in shaping diverse individual behaviours. Our findings reaffirm the well-established importance of genetics and individual life experience, including stochastic developmental processes. But they also clearly challenge a widely held assumption: that genetics can reliably predict individual behaviour in a near-perfectly controlled environment.

Instead, we find that genetics only sets the stage—a behavioural landscape—but does not dictate what part of it will be inhabited by an individual. The path an individual takes to get to its place in that landscape depends largely on their unique experiences. In fact, we find that all genotype landscapes are largely overlapping, meaning that every genotype has the potential to produce nearly any possible behavioural phenotype, even when individuals are raised in the same environment (Figure 1.H, 4.E). Yet almost paradoxically, the variability in behaviour we observed cannot be understood without acknowledging the role of genetic diversity.

Previously, individual variability was attributed mainly to stochastic events during development and life history that interacts with genetics. We confirm that, but go further. Our study reveals that randomness isn't limited to developmental or environmental factors, or bound only by genetics. Seemingly trivial events—like the context of an individual's very first decision in a learning task—can set off a butterfly effect that shapes all future behaviour. These moments are often self-generated, underscoring the dynamic role of the agent in its own behavioural trajectory.

Crucially, learning is the main engine that drives this process. This makes individual cognitive behaviour fundamentally individual, time-sensitive, and non-ergodic. This, perhaps inconvenient, truth is something many researchers studying learning-dependent behaviour have long sensed: that even in controlled experimental conditions, and no genetic diversity, behavioural variability—especially in cognitive performance—is inevitable.

To truly understand the mechanisms—especially the genetic underpinnings—of cognition in autonomous agents, we will have to move beyond averaged metrics, linear models, and snapshot observations within a genotype or species. This will be particularly critical when studying complex animals like humans, whose lived experiences far exceed the simplicity of a fly choosing between two colours in a Y-maze.

Main figures

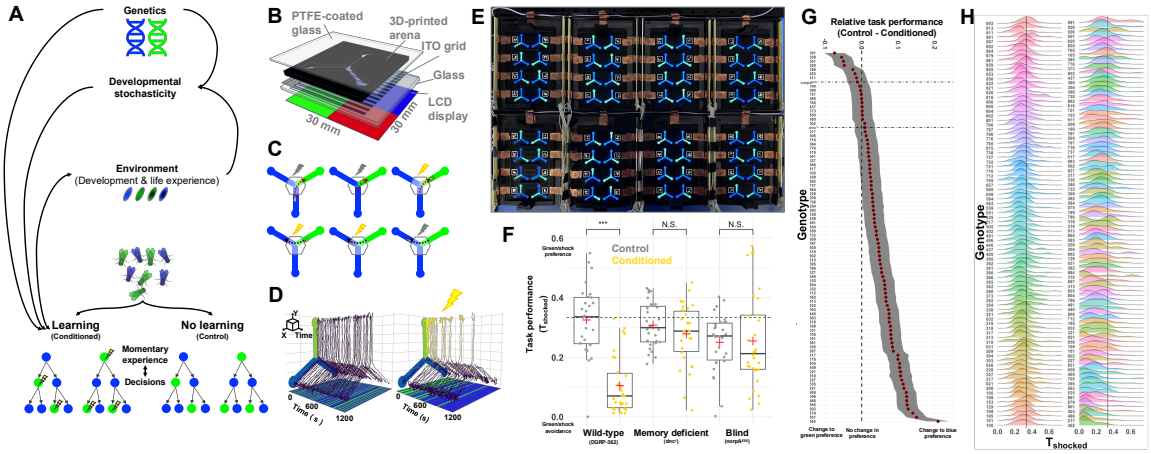

Figure 1. Capturing sources of variability in individual learning-dependent behaviour. (**A**) Sources of behavioural individuality. (**B**) Schematic of the Y-maze design. (**C**) Shock delivery and removal timing upon a decision in a visual place learning task. (**D**) Representative tracks of two flies traversing a Y-maze in the presence (right) or absence (left) of conditioning. Lines on the bottom represent colour occupancy. (**E**) Top-down view of the 64-maze multiplexed platform. (**F**) Task performance in green place learning task of a wild-type genotype compared to the memory- and learning-deficient mutant (*dnc*^{1}) and the visually impaired mutant (*norpA*^{EE5}). Points are individual fly measurements. Mean and medians are represented as bar and red plus. (**G**) Relative task performance in place learning task across 90 genotypes. Grey shading is SE. Interrupted line indicates no change in performance upon conditioning (vertical, 0) and relative task performance for memory- and vision-deficient genotypes (*dnc*^{1} and *norpA*^{EE5}, horizontal) . (**H**) Distributions of task performance (kernel density) across genotypes, ordered from the highest mean to the lowest mean $T_{shocked}$ (bottom) in control (N = 32 per genotype per treatment). Vertical line represents chance level $T_{shocked}$ = 1/3.

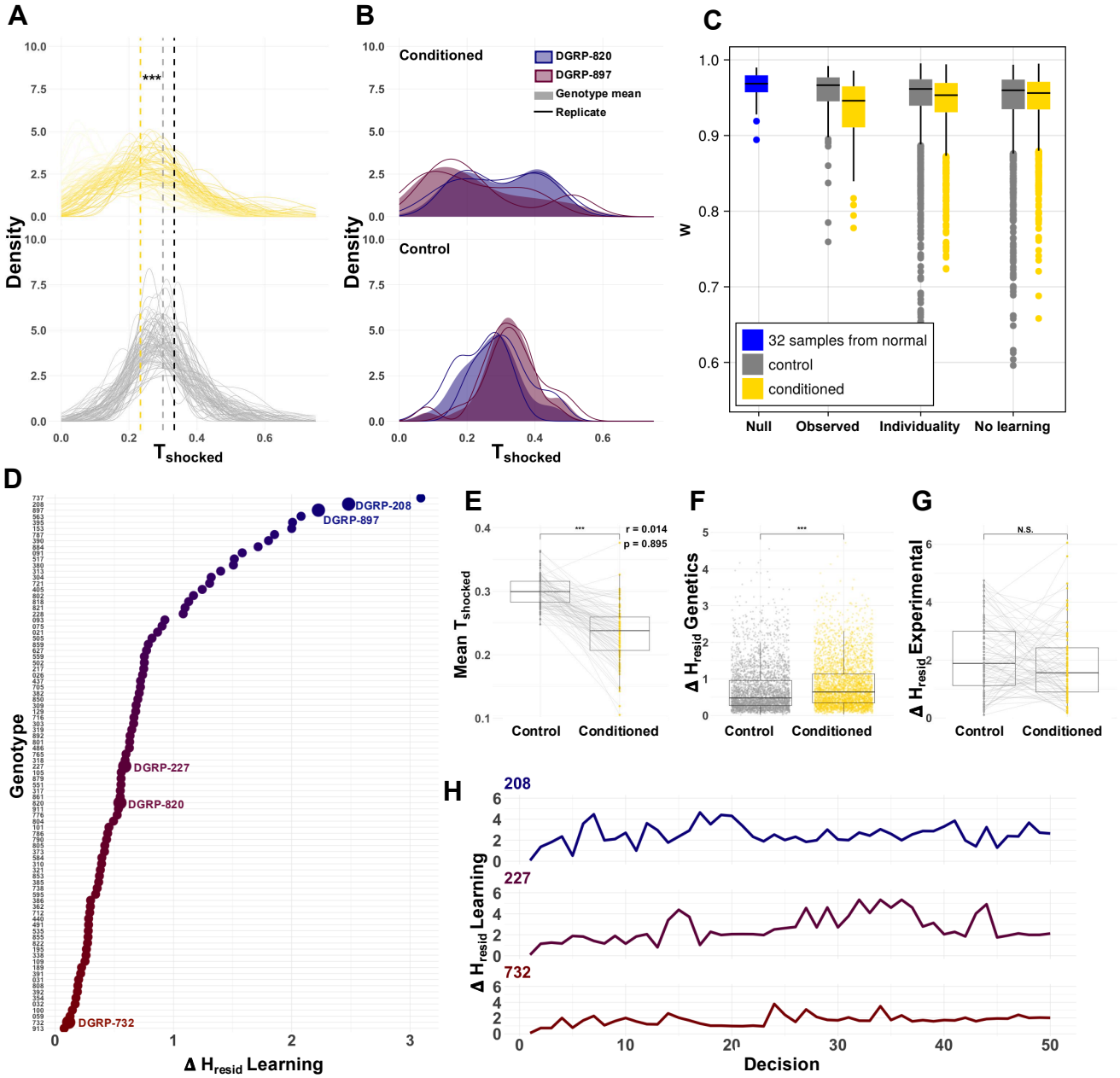

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Figure 2. Individuality and residual individuality in task performance. (**A**) Distributions of individual task performance for the 88 wild-type genotypes in conditioned (yellow) and control (gray) flies. Vertical lines show mean task performance. Chance expectation of $T_{shocked}$ at 1/3 is black. (**B**) An example of individual $T_{shocked}$ distributions in two genotypes (shaded red and blue) is shown. Replicates are shown as lines. (**C**) Shapiro-Wilk test for normality for task performance in observed flies and flies simulated under *Individuality* and *No learning model*, as well as for 90 random samples of N = 32 from a normal distribution (Null). The W-statistics of the control group (observed gray) does not differ from the null distribution (blue; Mann-Whitney U test, $p = 0.13$), but it clearly does from the distribution of conditioned flies (observed yellow; Mann-Whitney U test, $p < 10^{-4}$). Observed decrease in normality in conditioned flies is reproduced by the *Individuality* model (Control versus Conditioned Mann-Whitney U test, $p = 0.015$), and absent in the *No learning* model (Control versus Conditioned; Mann-Whitney U test, $p = 0.41$). The boxplots encompass the observed 90 genotypes (88 wild-type, dnc^{1} and $norpA^{EE5}$), and 25 independent simulation seeds for each genotype for the simulated data. (**D**) The change in residual individuality due to learning is genotype-specific. (**E**) Mean task performance across genotypes (points). Lines connect the same genotype's value across the two treatments. Inset text shows correlation and p-value for mean $T_{shocked}$ between two treatments. (**F,G**) Change in expressed residual individuality due to genetic and experimental causes. Each point represents pairwise divergence between two genotypes (F) or two replicates of a genotype (G). (**H**) Divergence in residual individuality due to learning evolves with every decision and increases over time.

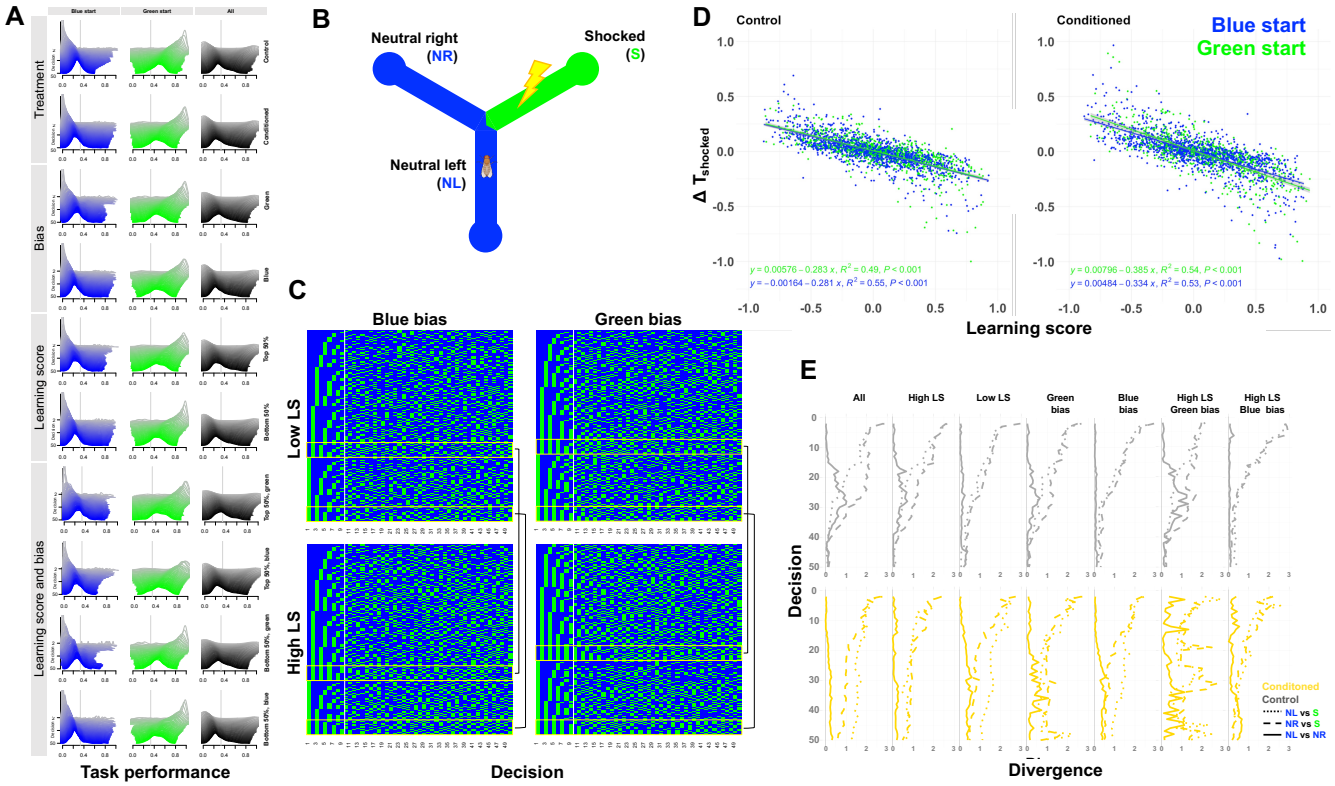
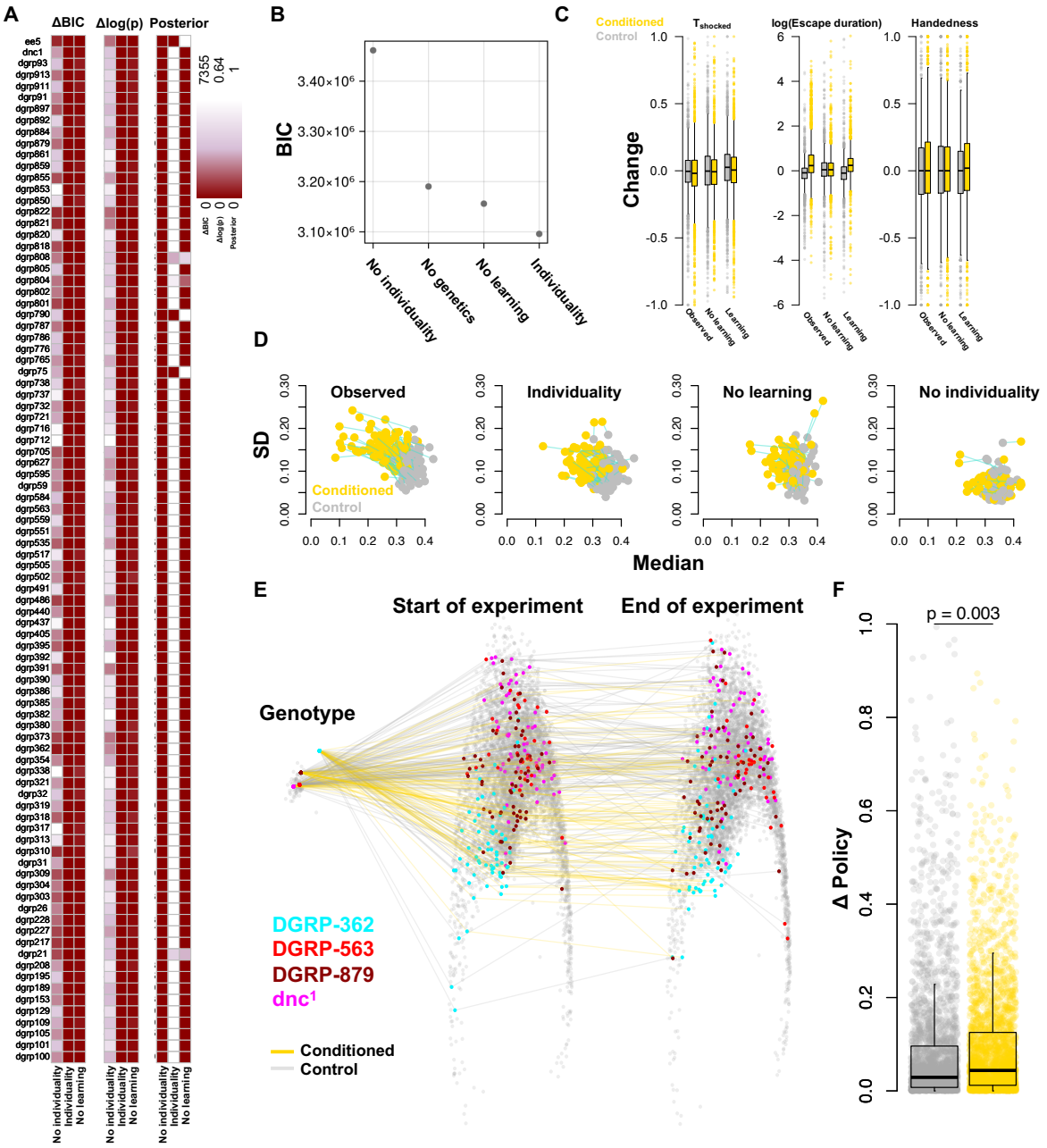


Figure 3. Divergence in individual task performance depends on learning through experience. (**A**) Evolution of individual task performance distributions over the first 50 decisions is dependent on starting colour in all flies, those who share the same colour bias, and those with above and below average learning score (LS). Two distributions of the left and right blue arm are overlaid(see B). Only flies that made at least 50 decisions are included. Distributions regardless of starting position are shown in black. (**B**) Schematic of the Y-maze and possible starting colour and shock experiences. For the conditioned flies, the green coloured arm (S) is associated with the shock, while for control flies it is not. (**C**) First 50 decisions made by flies with high and low learning score and different colour bias, are aligned across individual flies (rows) and sequentially sorted. The yellow rectangles bring attention to flies with most persistent colour choice switching behaviour. Black brackets indicate flies that share the same first decision for easier comparison across groups. (**D**) Change in individual task performance is more sensitive to experience at the first decision (blue or green colour) in the presence of learning. Each point is an individual fly. (**E**) Evolving change in H_{resid} between flies starting at different arms of the Y-maze, that made at least 50 decisions. Residual individuality is sensitive to initial experience and exacerbated by opposing forces of learning and unfavourable bias.


A
ΔBIC
Δlog(p)
Posterior
No individuality
Individuality
No learning
B
BIC
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SD
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Observed
Individuality
No learning
No individuality
Conditioned
Control
E
Start of experiment
End of experiment
Genotype
DGRP-362
DGRP-563
DGRP-879
dnc1
Conditioned
Control
F
p = 0.003
Δ Policy

289

Figure 4. Experience, genetics and learning shape individuality. (**A**) Variation in behaviour observed across genotypes is best explained by a model that includes genetically controlled but individually random prior life experience, and learning (*individuality*). (**B**) High Bayesian information criterion for models omitting variability in behaviour prior to the task, genetics, and learning demonstrate that they are necessary for close *in silico* recapitulation of observed individuality. (**C**) Change in individual behaviours during conditioning can be attributed to the effect of learning. Boxplots summarize the behaviours of observed, real flies and of flies simulated with and without learning. (**D**) Flies simulated under the assumption of genetic control over variability in life experience and with learning during task can faithfully reproduce observed mean task performance and individual variation in behaviour of real flies. Points are genotypes. (**E**) Genetic effect on behavioural policy projects to the entire individual behaviour space. Mean genotype policies and overlapping individual policies measured at the start and at the end of the experiment are projected on a multidimensional scaling plot. Points show genotypes and individuals. Highlighted points correspond to individuals from an outlier genotype (turquoise), two average genotypes (red and dark red) and a memory-deficient genotype (magenta). (**F**) Change from initial behavioural policy from the start until the end of the task changes more in flies that learn. Points are individuals.

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Author contributions statement

A.M.J. and R.M. conceptualized and designed the study. R.M., A.M.J. and I.T. conceptualized and designed the behavior platform. R.M. built the platform's software, R.M. and A.M.J. built the experimental hardware. R.M., G.V.B. and A.M.J. performed the experiments. R.M., J.B., A.M.J. and A.M. conceptualized the data analysis. R.M, J.B., G.V.B. and A.M.J. analyzed the data and produced the results. A.M.J., R.M. and J.B. wrote the manuscript. A.M.J. supervised the study. All authors read, edited and intellectually significantly contributed to the manuscript.

Additional information

Accession codes Data and code used to produce the main results, as well as PCB and 3D print designs are available on a Github repository https://github.com/jaksiclab/GeneticsOfLearningIndividual and on Zenodo.

Competing interests All authors declare no competing interests.