A canonical neural mechanism for behavioral variability

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The ability to generate variable movements is essential for learning and adjusting complex behaviours. This variability has been linked to the temporal irregularity of neuronal activity in the central nervous system. However, how neuronal irregularity actually translates into behavioural variability is unclear. Here we combine modelling, electrophysiological and behavioural studies to address this issue. We demonstrate that a model circuit comprising topographically organized and strongly recurrent neural networks can autonomously generate irregular motor behaviours. Simultaneous recordings of neurons in singing finches reveal that neural correlations increase across the circuit driving song variability, in agreement with the model predictions. Analysing behavioural data, we find remarkable similarities in the babbling statistics of 5–6-month-old human infants and juveniles from three songbird species and show that our model naturally accounts for these ‘universal’ statistics.

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Behavioural variability is a pivotal component of motor learning and adaptation\textsuperscript{1,2}. While young individuals can usually produce non-stereotyped disorganized behaviours, motor exploration is more often expressed as movement variability around a stereotyped motor pattern. Highly irregular patterns of activity, which are ubiquitous in the brain\textsuperscript{1}, are thought to underlie variable motor behaviours\textsuperscript{4,5}. Specifically in songbirds, a neural circuit necessary for song learning and adaptation\textsuperscript{6–9}. This includes two cortical-like areas: a premotor nucleus, the lateral magnocellular nucleus of the anterior nidopallium (LMAN), and its efferent motor nucleus, the robust nucleus of the arcopallium (RA). While RA is essential in driving the effectors (muscles or muscle synergies) producing the song\textsuperscript{1}, LMAN is not necessary for song production in adults\textsuperscript{6–9} but has a key role throughout development and in adults in driving variability in the song\textsuperscript{9,10} and in the activity of RA neurons\textsuperscript{11,12}.

The idea that temporally irregular activity of neurons in the central nervous system (CNS) is capable of generating behavioural variability may seem obvious. A careful examination, however, reveals that the link between irregularity in neural activity and behavioural variability is far from being straightforward. This is because to impact the behaviour, patterns of activity generated in the CNS must also be spatially correlated (that is, correlated across neurons). For example, consider the minimal model of a cortical network driving motor behaviour depicted in Fig. 1a. It consists of many neurons randomly connected recurrently, divided into $D$ functional groups; each group is composed of $M$ neurons (larger than $D$ by over an order of magnitude) that project to one effector of the motor behaviour. The collective dynamics of the network give rise to highly irregular firing patterns as a consequence of the interplay between excitation and inhibition\textsuperscript{13} (Fig. 1b, Supplementary Fig. 1a,b). Despite this large variability in their activity, unless the number of neurons in a group is very small, fluctuations in the effectors are negligible (the coefficient of variation of the input to the effector, $CV_{\text{eff}}$, see Methods section, is very small; Fig. 1a, Supplementary Fig. 1e). This stems from the fact that the network activity is only weakly correlated across neurons (on the order of $1/N$, where $N$ is the number of neurons in the network, Fig. 1b right, Supplementary Fig. 1d) and thus, by virtue of the law of large numbers, the fluctuations they induce in the net input to an effector ‘average out’. This example emphasizes the fact that, for the fluctuations to be transferred robustly from the CNS to the effectors, neuronal firing in the motor network must be sufficiently correlated within a neural population projecting to the same effector.

While the mechanism underlying asynchronous irregular spiking activity in recurrent networks of excitatory and inhibitory neurons is well understood\textsuperscript{13–16}, how the CNS autonomously generates patterns of activity, which are both temporally irregular and correlated across neurons, remains an open fundamental question\textsuperscript{16–19}. A key result of our theoretical work is that the activity of neurons in the motor network driving the effectors will be highly irregular and also spatially correlated if this network receives topographically organized excitatory projections from another upstream strongly recurrent network, hereafter premotor network. In the context of the circuit driving song variability in songbirds, our theory predicts that correlations emerge along the LMAN–RA circuit, namely, that correlations across neurons are very weak in LMAN but substantial in RA. We validate this prediction with simultaneous extracellular recordings of neurons in singing finches. Our theory also suggests that vocal variability in different species of juvenile vocal learners should exhibit very similar statistics, as a consequence of universal statistical properties of the circuit dynamics. We verify this prediction by comparing the statistics of the song produced during the babbling phase of three species of songbirds as well as of human infants. Preliminary report of this work previously appeared in an abstract form\textsuperscript{20}.

**Results**

We first show that temporally irregular and spatially correlated patterns of spiking activity can robustly emerge in a circuit of topographically organized and strongly recurrent networks. To this end, we consider the circuit depicted in Fig. 2a. Neurons in the motor network that project to the same effector share a fraction, $f$, of their premotor inputs, and this shared component is different from one group to the other (see Methods section for a detailed description of the architecture). With this architecture, the spiking activities of the neurons in the premotor, as well as in the motor network, are highly irregular, as a result of their recurrent dynamics. There is, however, an important difference between the networks in the spatial structure of their activities. In the premotor network, correlations across neurons are typically extremely weak (Figs 2b,c and 4a). In contrast, in the motor network pairs of neurons projecting to the same effector are substantially and positively correlated, whereas correlations are weak (and possibly negative) for neurons projecting to different effectors (Figs 2d–f and 4b and Supplementary Figs 3–5). These functional correlations are highly robust and only weakly influenced by the model parameters (Supplementary Figs 3d,e and 5d–f and Supplementary Note 1). As a result, fluctuations are amplified along the circuit (Fig. 2g) and the variability is robustly transferred to the effectors.

Importantly, the correlations in the motor network are substantial only if the footprint of the recurrent interactions in that network is sufficiently wider than the footprint of the premotor-to-motor projections (Fig. 2h). Indeed, when the recurrent interactions are too local, correlations in the motor network are weak (Fig. 2h,i). Thus temporally irregular and spatially correlated patterns of activity naturally emerge from the interplay between topographic feedforward (FF) projections from the premotor to the motor network and recurrent interactions within the motor network (Supplementary Fig. 10).

The emergence of spatial correlations in the motor network can be intuitively understood as follows. The FF input to a neuron in the motor network consists of one structured component, shared by all neurons belonging to the same functional group, and another one which is unstructured. Since the neurons in the premotor network are firing asynchronously, both components are the sum of a large number of uncorrelated contributions (on average $fK$ and $(1−f)K$, respectively; $K$ being the average number of synapses per neuron; see Supplementary Fig. 3i) and thus their temporal fluctuations are smaller than their temporal average by a factor on the order of $1/\sqrt{K}$ (Supplementary Fig. 3f: blue curve). Neural activity in the motor network will be spatially correlated if the amplitude of the fluctuations in the structured component to the network is on the order of the neuronal threshold. This implies that the temporally averaged FF input must be on the order of $\sqrt{K}$. This will happen if the strength of the FF connections are on the order of $1/\sqrt{K}$. To prevent the neurons in the motor network to fire regularly at a very high rate, the inhibitory recurrent inputs in the motor network must compensate for most of this averaged FF input. Such a compensation occurs naturally if the motor network is strongly recurrent and operates in the ‘balanced excitation–inhibition’ regime\textsuperscript{13} (see Supplementary Note 1 for more details on the mechanism).

The fluctuations in the component common to all neurons in the same functional group give rise to the correlations in the
activity in the motor network on a spatial scale on the order of the size of a group. Moreover, the groups also compete with each other provided that the recurrent interactions extend over a distance larger than the size of a group. As a result, the network dynamics self-organize such that the average instantaneous rates of the excitatory and inhibitory populations are essentially constant in time (Fig. 2d). This guarantees that the network operates in the balanced excitation-inhibition regime in a robust manner (see Supplementary Note 1).

**Correlation structure in circuit driving vocal variability.**

Songbirds, with their well-identified and segregated circuit devoted to song learning, including a minimal circuit driving song variability (see Introduction), offer an ideal opportunity to test predictions of our theory. In songbirds, LMAN controls the trial-to-trial fluctuations across repetitions of the temporally structured song. These fluctuations are important for adapting the song upon perturbation. Moreover, anatomical studies in the circuit driving the song indicate that the projections from LMAN to RA are topographically organized, as our model posits for the projections in the premotor-to-motor pathway. We therefore hypothesized that song variability stems from essentially uncorrelated fluctuations produced in LMAN, which by virtue of the topographic projections from LMAN to RA induce spatially correlated fluctuations in RA activity. To further test this hypothesis, we extended the two-area circuit considered above to take also into account the temporally structured inputs from nucleus HVC (used as a proper name) into RA neurons. To this end, we included an additional FF excitation to the motor network in our model, representing the latter input (Fig. 3b, see Methods section). The responses of the neurons in the motor network are then locked to this input in a way that is reminiscent to the locking of RA neurons to the song. By analysing the spatiotemporal patterns of these trial-to-trrial fluctuations, we found substantial noise correlations (see Methods section) for neurons in the motor network belonging to the same group but almost none in the upstream premotor network (Fig. 4). In the motor network, noise correlations were positive for pairs in the same group. They were typically weaker and negative for pairs in different groups. The averaged correlation over all pairs of excitatory neurons was very small due to the compensation between positive and negative correlations. (Fig. 4b and also Supplementary Fig. 4). Our model thus predicts a build-up of noise correlations along the circuit generating behavioural variability in singing birds.

To test this prediction, we recorded pairs of LMAN or RA neurons during singing in zebra finches. In LMAN, we found that spike-triggered average (STA) of the local field potential (LFP), as well as STA of the multi-unit activity were weak (Fig. 5b, Supplementary Fig. 6, see Methods section). We also found that noise crosscorrelograms were flat (Fig. 5c,d) and that correlation coefficients were tightly distributed around zero (Fig. 5e) in LMAN. By contrast, in RA neurons displayed substantial noise correlations during singing, as revealed by the shape of their crosscorrelograms (Fig. 5h,i; one-tailed two-sample t-test; $P<0.01$ for single-units pairs, $n=4$ pairs in LMAN and $n=5$ pairs in RA; $P<0.001$ for single- versus multi-units pairs, $n=6$ pairs in LMAN and $n=25$ pairs in RA; and $P<0.001$ for multi-units pairs, $n=21$ pairs in LMAN and $n=21$ pairs in RA; see Methods section) and large values of noise correlation coefficients (compare Fig. 5j with Fig. 5e). The fact that correlations in RA were stronger than in LMAN is consistent with the model prediction, since the recorded units were likely to be located in the same functional group given the small distance between electrodes compared to RA diameter (see also Supplementary Fig. 6 and Supplementary Note 2). Multi-unit-STA and LFP-STA also consistently displayed high noise-related activity around the recorded spikes in RA, in contrast to LMAN (compare Fig. 5g with Fig. 5b and Supplementary Fig. 6a). Finally, we found that noise crosscorrelations (CCs) between LFPs recorded from evenly spaced electrodes decreased with the distance between the electrodes and became negative when they

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**Figure 1 | Fluctuations in the inputs to the effectors are very weak when noise is generated autonomously in the motor network.** (a) The motor network projects in a topographic manner to $D$ effectors ($D=10$ effectors, 4 represented): each effector receives inputs from a different group of $M=1,000$ neurons. In spite of the large variability of the neuronal activity, the variability of the effectors (right) is extremely small (coefficient of variation of the effector averaged over the 10 effectors: $CV_{eff}=0.007$). (b) The neuronal activity in the motor network is highly irregular and the correlations across neurons are tightly distributed around zero. Left: Voltage traces for one excitatory (E, red) and one inhibitory (I, blue) neuron. Middle: Distributions of coefficient of variation of the inter-spike interval, $CV_{ISI}$. Right: probability density function (pdf) of Pearson correlation coefficients in the network.
were far apart (Supplementary Fig. 6b and Supplementary Note 2). Therefore, as predicted by our model, noise correlations during singing are strong in RA, while they are extremely weak in LMAN.

Our electrophysiological recordings also reveal that the decays of the autocorrelations (ACs) and of the CCs of the spiking activity last for hundred of milliseconds in RA neurons (Figs 5b,i and 6b,c) and that these decays are substantially faster in LMAN (Fig. 6a,c; two-sample t-test, \( P < 0.01 \) for \( n = 10 \) single units in LMAN and \( n = 14 \) single units in RA). What is the source of the relatively slow decorrelations in the activity of RA neurons? In our theory, synchronous temporal fluctuations in RA activity will be slow if the shared FF drive of the neurons in the motor network slowly fluctuates (see Supplementary Note 1 and Supplementary Fig. 7). If the synaptic dynamics in the premotor-to-motor pathway are slow, they give rise to autocorrelograms and crosscorrelograms in the motor network that can be as broad as in the data (compare Fig. 6a–c with Fig. 6d–f and Fig. 5h,i with Fig. 4b). This result suggests that the observed slowness of the fluctuations in RA activity stems from a low pass filtering of the fast fluctuations of LMAN outputs due to the large proportion of NMDA (N-methyl-D-aspartate) receptors in the LMAN-to-RA projections\(^29–31\).

Statistics of vocal variability in juvenile learners. Juvenile songbirds produce babbling-like vocalizations that are not stereotyped and highly variable\(^10,32\). At this early developmental
network of the model circuit in the presence of temporally structured FF input. (b) Extension of the model depicted in Fig. 2a. Neurons in the motor network receives also temporally structured FF inputs, representing HVC inputs in the adult zebra finch (see main text and Methods section). (c) Raster plot and corresponding average firing rate of a neuron in the motor network of the model circuit in the presence of temporally structured FF input.

Figure 3 | Single unit recordings in zebra finch RA nucleus and in the model motor network. (a) Top: song motif of a zebra finch. Bottom: recordings of RA single unit over 133 repetitions of song motif, aligned to one syllable in the motif (lower panel) and the corresponding average firing rate (upper panel; 5 ms bin size). (b) Temporal structure extension of the model depicted in Fig. 2a. Neurons in the motor network receives also temporally structured FF inputs, representing HVC inputs in the adult zebra finch (see main text and Methods section). (c) Raster plot and corresponding average firing rate of a neuron in the motor network of the model circuit in the presence of temporally structured FF input.

To what extent do these statistics depend on the details of the model architecture and connectivity, the neuronal dynamics and the nonlinearity in the input–output transduction by the effectors? Fluctuations in the FF input to the neurons in the motor network in our circuit consist of many uncorrelated fluctuating contributions and their statistics are thus close to Gaussian (Supplementary Fig. 8a). This is the case also for the net (FF + recurrent) input to these neurons. (Supplementary Fig. 8b). Hence, the fluctuating activity of the neurons in the motor network can be approximately described as a wideband Gaussian process that is rectified (Supplementary Fig. 8b–d), resulting in the tendency of the neurons to fire bursts of spikes with an approximately exponential distribution of durations (Supplementary Fig. 8f). Moreover, because neurons in the motor network are correlated, the temporal statistics of the input to an effector and of the neuron activity are similar. While the babbling behaviour generated by the circuit is a complex transformation. For example, for rectified power-law transformations the distribution of gesture durations is close to exponential (Supplementary Fig. 8g) and the ACE barely depends on the nonlinearity (Supplementary Fig. 8e). This is also true when combining the circuit with a mechanical model of the vocal production organ34. Therefore, these features reflect universal statistical properties of the circuit dynamics and are, to a large extent, insensitive to the circuit parameters and to the details of the transformation from the input to the effectors to the vocal behaviour.

Given the universality of the statistics of the features analysed above in our model, we then compared the ‘babbling behaviour’ generated by the model with babbling behaviour in different vocal learners. To this end, we analysed babbling vocalizations of juveniles from three different songbird species with completely different adult repertoires (zebra finches: single song of 3–8 syllables per individual; swamp sparrow: 2–5 stereotyped songs per individual gathering 5–10 syllable types; canaries: complex song sequences based on a repertoire of 20–40 syllables per individual), as well as vocalizations of 5–6-month-old human infants (adult repertoire: complex sentences based on 10–100 phonemes grouped in >10,000 words).

Remarkably, we found that the statistics of the vocalizations produced during the early period of babbling (but not later in development, Supplementary Fig. 9a–c,f) had a large degree of similarity in the four species we analysed. In all four species, as in the model, the distribution of vocal gesture durations could be well fitted with a single exponential (Fig. 7b–e, left and insets; see Methods section, see also30,36). In addition, the ACE lacks a clear temporal structure in all babbling vocalizations. The scale parameter of the gesture duration distributions, as well as the decorrelation times of ACE (that is, the typical time constant of the ACE) varied across individuals and species from several tens to a few hundreds of milliseconds (Fig. 7b–e). However, as the distributions were close to exponential and variability within species was small (Fig. 7f–i), interspecies and intraspecies differences in gesture duration distributions became comparable after normalizing each individual distribution by its species-averaged scale parameter (Fig. 7g–i; two-sample t-test, \( P = 0.92 \); only 9% of the total variance among distributions was attributed
to species differences compared to 87% before rescaling; one-way analysis of variance (ANOVA)). A similar uniformity was observed in the inter-gesture interval distributions after normalization by the species average (Supplementary Fig. 9d.; $P = 0.23$, only 10% of total variance among distributions was attributed to species differences compared to 66% before normalization; one-way ANOVA). Interspecies variations in ACE were mostly due to differences in the species-averaged decorrelation time ($P = 0.16$; only 17% of the total variance among ACEs was attributed to species differences, compared to 81% before normalization; one-way ANOVA). Finally, correlations between consecutive gestures and inter-gestures were small and comparable among species (Supplementary Fig. 9e). Together, these results show that in the four species we studied the statistics of the babbling-like vocalizations are very similar and can be naturally accounted by our minimal circuit.

**Discussion**

Our paper addresses the extent to which the intrinsic temporal irregularity of neuronal activity in the CNS can drive motor variability. This is a fundamental non-trivial question since, as to impact the behaviour, patterns of activity generated in the CNS must also be spatially correlated (that is, correlated across neurons; see also Supplementary Note 3. for alternative mechanisms). Although the emergence of asynchronous irregular activity in recurrent networks is well understood\(^1\), much less is known regarding the possible mechanisms giving rise to irregular spiking in which fluctuations of the activity are both temporally irregular and correlated across neurons. As a matter of fact, in virtually all network models of irregular spiking previously investigated, the activity is either asynchronous\(^2\) or the synchronous component of the temporal fluctuations in neuronal spiking is strongly rhythmic\(^3\).

In particular, previous theoretical studies\(^1\).\(^3\) concluded that correlations should be very weak in strongly recurrent cortical circuits (on the order of 1/N, where $N$ is the network size). However, these studies assumed a completely random connectivity, without structure (with an Erdős–Renyi graph). Here we showed that substantial correlations emerge naturally in a circuit with topography. With such an architecture, the dynamics self-organize in groups of neurons that are positively correlated within a group but negatively correlated between groups. In this spatial pattern of correlations, the balance between excitation and inhibition is maintained over the whole network. As a result, the circuit can eventually produce robust variable behaviour with ‘universal’ statistics. In fact, we showed that this mechanism does not require any fine-tuning of parameters. In particular, it is robust to the number of neurons and the average number of connections, as well as to the connectivity in the topographic pathway to the effectors (and the number of neurons projecting to an effector; see also Supplementary Note 1).

In songbirds, the organization of the LMAN-to-RA pathway becomes clearly topographic during the early sensory period of song learning\(^4\). Thus it is already present when juvenile finches start to babble (35–40 days post hatch (DPH)). Neurons in RA also send topographic projections to the hypoglossal nucleus (nXII) as well as to the respiratory motor nuclei\(^5\). The projections of the hypoglossal nucleus to syringeal muscles are also topographic\(^6\). Thus the pathway from RA to syringeal muscles (and likely similarly to respiratory muscles) is topographic, as required by our mechanism. Applied to the LMAN–RA circuit, this mechanism predicts that noise correlations are weak in LMAN but substantial in RA. We reported experimental evidence in line with this prediction in the adult zebra finch.

In juvenile and adult zebra finches, the inputs from LMAN to RA are dominated by NMDA receptors with slow kinetics of time
constant on the order of \( \sim 100 \text{ ms}^{29-31} \). Moreover, recurrent excitation in LMAN is largely dominated by NMDA receptors and the kinetics of these receptors is faster in adults than in young juveniles\(^{40}\), with typical time constants of \( \sim 30 \text{ ms} \) in adults and \( \sim 120 \text{ ms} \) in juveniles\(^{41}\). Therefore, slow synapses in LMAN as well in LMAN to RA projections can underlie the relative slowness of the dynamics of the babbling behaviour we reported in juvenile finches (see also Supplementary Fig. 7 and Supplementary Note 1). In agreement with this view, localized mild cooling of LMAN in zebra finches results in an increase in the time constant of the exponential gesture distribution during babbling-like behaviour and in a longer tail in the distribution in older juveniles\(^{36}\).

Our behavioural data show substantial differences in the timescale of the babbling behaviour between zebra finches, canaries, swamp sparrows and humans. Our model suggests that this may be due to differences in the kinetics of NMDA receptors in these species. Revealing a direct correlation between these differences and NMDA receptors kinetics requires data on the latter. To the best of our knowledge, there is no such data available for canaries, swamp sparrows or human infants. However, the range spanned by the babbling timescales in our behavioural data is compatible with the diversity of kinetics reported in NMDA receptors of different subunit composition\(^{42,43}\).

In adult subjects, motor variability is expressed as fluctuations around a stereotyped motor pattern, which despite their relatively small amplitude, can contribute significantly to motor learning\(^2\). At early stage of development, young animals, as well as human infants, produce spontaneous exploratory gestures referred to as 'motor babbling' that do not rely on any stereotyped or goal-oriented movement and rather appear to express pure motor variability\(^{10,44,45}\). Such exploratory movements may allow the self-organization\(^{46}\) and the adaptation of sensory-motor networks through correlation-based (Hebbian learning) and reinforcement learning mechanisms\(^{47-49}\). These mechanisms posit that synaptic neural correlates of exploratory behaviour must persist for tens of milliseconds in the learning circuit. Our work suggests that the wide presence of NMDA receptors in the LMAN-to-RA projections\(^{36,31}\) is a key component in the emergence of such eligibility trace in the overall dynamics of the circuit that generates behavioural variability in birds.

To conclude, we showed that a circuit comprising strongly recurrent neural networks, which is organized in a topographic manner, is capable of driving variable motor behaviours. This mechanism relies on only a few architectural constraints and is thus likely to be a general operating principle by which the brain acquires motor skills and adapt behaviour in a changing environment.
Methods

Subjects. Seven human infants (3 males and 4 females) were recorded in their natural environment. Their parents gave written informed consent for participation in this study. Nine zebra finches and five canaries were obtained from our breeding facilities (Paris Descartes and Paris Sud Universities). Seven swamp sparrows were collected as nestlings and hand-reared in the laboratory (see ref. 51 for details). Birds were housed under natural light/dark conditions and provided with food and water ad libitum. Animal care and experiments were performed in accordance with European directives (86/609/CEE and 2010-63-UE) and the French legislation. Experiments were approved by Paris Descartes University ethics committee.

Human infants. We recorded spontaneous vocalizations in six infants in their natural environment starting from 5 to 7 months after birth (denoted as the 'babbling period'). The parents were instructed to place a recorder (digital dictation machine with stereo microphone, ICD-IXM335M SONY) near the baby's head for ~30 min at least 5 days a week for several weeks (4–20 weeks). The data presented include babies which vocalizations were collected from least 20 days during this recording period. Additionally, one 10-month-old infant was recorded for repetitive babbling.

Zebra finches. Juvenile zebra finches were raised in single cages with their parents and siblings. At age 26–41 DPH ('babbling period'), 9 male zebra finches were collected as nesting and hand reared. Vocalizations were recorded continuously with Sound Analysis Pro, which was configured to ensure that recordings were triggered on all quiet vocalizations of young birds. Five of the nine birds were continuously recorded until song crystallization (~3 months), with episodic access to their father.

Swamp sparrows. Seven swamp sparrow males were recorded in individual sound isolation chambers (Industrial Acoustics AC-1) once per week, starting in February of their first year when they were about 250 DPH. The onset of song development was first detected at 262–296 DPH ('babbling period'), and recording continued up to 366–386 DPH, when the males were singing crystallized adult song. Subsong was sampled for 30 min (Marantz PMD221 cassette tape recorder, Realistic Omni-directional microphone, Yamaha Mike to Line Amplifier). An automated system was introduced to detect and record songs during late plastic and crystallized song using a voice-activated switch (modified UberAkosmat) and a Digital Delay System (Digittech).

Canaries. Juvenile canaries were raised in our breeding facility at Paris Sud University in single cages with their parents and siblings. At age 75–150 DPH ('babbling period'), they started to produce their first vocalizations, five male canaries were removed and placed in custom-made sound isolation chambers. Vocalizations were recorded continuously for 3 months (September–December) during the fall following their birth with Sound Analysis Pro, which was configured to ensure that recordings were triggered on all quiet vocalizations of young birds. Four of the five birds were also recorded 3 months later (early spring) for 5–10 more days.

Vocalizations. Songs and infant vocalizations were manually sorted. For subsongs, we took the first recorded song vocalizations of the bird. Recordings were from 1 day of vocalizations, except for zebra finches, where in some individuals subsongs from 1 to 3 recording days were combined to get enough gestures.

Spectrograms. Spectrograms were estimated using the multitaper method with two sleighan tapers.

Envelope signal. We extracted the envelope of the signal (termed also ‘amplitude’ in the literature) by band passing the signal in the frequency ranges of the vocalizations (from 800 Hz and up to 4,000–10,000 Hz, depending on the species, with order-80 linear-phase finite-duration impulse response filter), taking the absolute value of the signal and low passing it at 1–200 Hz with a linear filter of order-200 linear-phase filter finite-duration impulse response.

Averaged ACs of envelope (ACE). The ACE was estimated for each recording and then normalized to the zero lag. The ACE signal was then estimated by averaging this signal over 1 day of recording sessions.

Gusture and inter-gesture segmentation. We used a local method for gesture and inter-gesture detection. We calculated the peaks of the derivative of the log-envelope signal (after band passing the signal; see above) that was smoothed...
Figure 7 | Babbling statistics are similar across different vocal learners and in the model. (a) Statistics of the babbling behaviour generated by the model circuit depicted in Fig. 2a–e when coupled to a minimal model of the vocal organ (see Methods section). Top: spectrogram of the vocal output signal ($t_g^{\text{model}} = 100$ ms). Bottom: probability density function (pdf) of vocal gesture durations (left) and averaged autocovariance of the envelope (ACE; right). Inset: distribution of gesture durations when the $y$ axis is in log-scale. The distribution of gesture durations is well approximated by an exponential with a ‘scale parameter’, $\theta_{\text{ACE}}$ (see Methods section). ACE decorrelates over a time duration of $\tau_{\text{ACE}}^\text{model}$ · Slow synaptic dynamics in the premotor-to-motor projections (red: $\tau_{\text{ACE}}^\text{model} = 50$ ms; blue: $\tau_{\text{ACE}}^\text{model} = 100$ ms) results in slowly fluctuating vocal output (red: $\tau_{\text{ACE}}^\text{model} = 60$ ms and $\tau_{\text{ACE}}^\text{model} = 31$ ms; blue: $\tau_{\text{ACE}}^\text{model} = 120$ ms and $\tau_{\text{ACE}}^\text{model} = 64$ ms). (b–i) Statistics of the babbling behaviour in four species of vocal learners (ages of the subjects ‘babbling period’) are given in Methods section). Blue: Zebra finches (Zf); Red: Swamp sparrows (Sw); Green: Canaries (Ca); Black: Baby infant (Bab). Different lines of the same colour correspond to different subjects from the same species. (b–e) Same as in (a), but for the Zf (b) compare to the blue line in a), Sw (c) compare to the red line in a), Ca (d) and Bab (e). Gesture duration distributions lack any clear peak and are well fit with exponential decaying function with scale parameters (mean ± s.e.m.); $\tau_{\text{ACE}}^\text{model} = 80 \pm 7$ ms; $\tau_{\text{ACE}}^\text{model} = 23 \pm 2$ ms; $\tau_{\text{ACE}}^\text{model} = 42 \pm 7$ ms; $\tau_{\text{ACE}}^\text{model} = 258 \pm 23$ ms. (f,g) Cumulative distribution functions (cdf) of gesture duration for the four species before (f) and after (g) normalizing the gesture durations by $\theta_{\text{ACE}}$. (h) Top: Interspecies differences in cdfs are much smaller than intraspecies differences (Kolmogorov-Smirnov statistic as a distance measure between cdfs). Bottom: Differences of cdfs in pairs of learners within (left to right: Zf–Zf, Sw–Sw, Ca–Ca, Bab–Bab) and between species (left to right: Zf–Sw, Zf–Ca, Zf–Bab, Sw–Ca, Sw–Bab, Ca–Bab). (i) Most of the interspecies differences in (h) are accounted for by normalizing the gesture durations to the scale parameter of the exponential fit of their distributions (see Results and Methods sections for statistical comparisons).
by a 5–30 ms sliding window, depending on the noise level of the signal and the species (using spikes in Matlab and a filter of (−1 0 1)) and defined sound onsets and offsets as the closest points to these crossings where the envelope deviated from the noise by 4 s.d. Using a global method instead of a local one on all the juvenile recordings yielded similar results; however, we preferred to use the local method whenever possible. For both methods, sounds separated by a duration of <7 ms of silence were merged into a single gesture, and segments of overly long (ZE: > 800 ms; Sw: > 400 ms; Ca: > 900 ms; Bab: > 3,500 ms) or short durations (ZE; Sw, Ca: 7 ms; Bab: <30 ms) were eliminated.

Fitting exponential decay. We fit an exponential function to the gesture duration distribution using maximum-likelihood estimation on a finite interval16 (based on a median of 1,870 gestures per day for a songbird and 700 for about 1 month of human infant recordings; interval duration: ZE: 50–800 ms; Sw:10–200 ms; Ca: 50–600 ms; Bab: 50–1,500 ms). Distributions that were well fit by the exponential function usually had a high goodness-of-fit (adjusted R² = 0.7 and Lilliefors statistic < 3.5). To extract decorrelation timescales from the ACE, we fit an exponential decay to the ACE.

Microdrive implantation. For single- and multi-unit recordings, a custom-built motorized microdrive (RP Metrix) was modified to accept 2–4 tungsten micro-electrodes (8–20 MΩ, FHC), as well as lateral positioner. It was implanted in LMAN or RA as follows: Young adult male zebra finches (C. C0 50 ms after firing an action potential and the voltage reset to

$$V(t) = \frac{ab}{t}$$

where$$a = G(h)$$

$$b = \frac{1}{m}$$

$$m$$ is the neuron membrane time constant,$$I$$ is the total FF input into that neuron.

$$L$$ is the strength of the connection from presynaptic neuron$$J > a$$ due to its recurrent interactions yields:

$$I_{rec}(t) = \sum_{i=1}^{n} \frac{V_{rec}(t)}{E_{i}}$$

where$$E_{i}$$ is the strength of the connection from presynaptic neuron$$j$$ with$$i$$.

Statistics. Numerical values are given as mean ± s.d., unless stated otherwise. Whenever using a statistical test, we report the type of test applied and the associated$$P$$ value (probability of observing the given result, or one more extreme, by chance if the null hypothesis is true).

Histology. After the last recording session, subjects were killed by intramuscular injection of sodium pentobarbital (Nembutal) and perfused transcardially with 0.9% saline followed by 4% paraformaldehyde as fixative. The brain was then removed, postfixed in 4% paraformaldehyde for 24 h and cryoprotected in 30% sucrose. Sections (60 μm thick) were then cut in the parasagittal plane on a freezing microtome and processed for histological examination to verify the location of the recording electrodes. Tissue was Nissl stained to visualize the electrode tracks.

Spikeing network model. Our model consists of two large recurrent networks, both comprising$$N_{e}$$ excitatory (E) and$$N_{i}$$ inhibitory (I) neurons. For simplicity, we take$$N_{e} = N_{i} = N$$. These two networks represent a premotor and a motor network. The premotor network projects in a FF manner to the motor network, and the latter activates a small number of effectors, consistent with the songbird anatomy10,12,25,29.

Single neuron and synaptic dynamics. All the neurons in the circuit are modelled as leaky-integrate-and-fire (LIF) units. The subthreshold dynamics of the membrane potential,$$V_{m}(t)$$, of neuron$$i$$ in population$$z (i = 1, \ldots, N_{z} = E,I)$$ obey:

$$\frac{dV_{m}(t)}{dt} = (V_{m}(t) - V_{s}) + I_{rec}(t) + I_{geo}(t)$$

where$$V_{s}$$ is the neuron membrane time constant,$$I_{rec}(t)$$ is the recurrent input into neuron$$i$$, whose$$I_{geo}(t)$$ is the total FF input into that neuron. The$$V_{m}$$ is the reversal potential of the leak current (taken to be$$V_{m} = -60 \text{ mV}$$. This subthreshold dynamics are supplemented by a reset condition: at$$t = t_{r}$$, the membrane potential of neuron$$i$$ crosses the threshold$$V_{th}(c_{th}) = 10 \text{ mV}$$, the neuron fires an action potential and the voltage reset to$$V_{m}(t_{r} + \tau_{r}) = -60 \text{ mV}$$. We model all synaptic inputs as pure currents. The total current into neuron$$i$$ due to its recurrent interactions yields:

$$I_{rec}(t) = \sum_{j} I_{ij}^{alpha}(t)$$

where$$I_{ij}^{alpha}(t)$$ is the current from presynaptic neuron$$j$$ with$$i$$.
neuron \((i, a)\) and \(S_p^{ij}(t)\) are the synaptic variables, which follow the dynamics:

\[
\tau_d E \frac{dS_p^{ij}(t)}{dt} = -S_p^{ij}(t) + \sum_{b} \delta(t - t_b).
\]

Here \(\tau_d\) is the synaptic time constant (assumed to depend solely on the nature—excitatory or inhibitory—of the presynaptic and postsynaptic neuron) and the sum is over all spikes emitted at times \(t_b < t\).

**Recurrence architecture.** The recurrent connectivity of the E and I populations in the premotor network is random (Erdös–Renyi graph). In each network, the connectivity matrix, \(C_{\beta\beta}\), between presynaptic population \(\beta\) and postsynaptic population \(\gamma\) is a random \(N \times N\) matrix such that \(C_{\beta\beta} = 1\), with probability \(K/N\) and zero otherwise, where \(K\) is the average number of inputs a neuron receives from population \(\beta\). We assume that the strength of the synapses depends solely on these populations yielding: \(I_p^{\beta\gamma} = I_{p0}C_{\beta\gamma}{\gamma}\), where \(I_{p0} > 0\) (excitation) and \(I_{p0} < 0\) (inhibition). When comparing the dynamics of networks with different connectivity, we follow the prescription\(^{15,16}\):

\[
I_{p0} = I_p/\sqrt{K}
\]

where the parameters \(I_{p0}\) are of order unity and can be different for the premotor and motor networks.

**Distance-dependent recurrent architecture.** In the motor network, the connectivity is random with probability, which depends on the distance between the neurons. The probability of connections between two neurons is \(p^a_{\beta\gamma} = f((x_a - x_g)\), where \(x_a = i\) is the location of neuron \(i = 1, \ldots, N\) in population \(\beta\), and

\[
f(x_a - x_g) = \frac{1}{\sqrt{2\pi}\sigma_{rec}} \sum_{-\infty}^{\infty} \exp\left(-\frac{1}{2}\left(\frac{x_a - x_g}{\sigma_{rec}}\right)^2\right)
\]

where \(\sigma_{rec}\) is the footprint of the recurrent interactions. Here we have assumed for simplicity that the motor network is one dimensional, of size \(L\) and periodic boundary conditions. For large values of \(\sigma_{rec}\), distant neurons are as likely to be connected as close ones, while for small values of \(\sigma_{rec}\), only neurons which are close have a significant probability to be connected (Fig. 2h). In most of the results depicted in the paper, we assume that the recurrent interactions in the motor network have a wide footprint \((\sigma_{rec} = 2L)\), except for Fig. 2h,i, where we investigate how the results depend on the value of \(\sigma_{rec}\).

**Feed-forward architecture.** The premotor network receives external FF inputs, which in the context of the songbird system represent the thalamic (the medial part of the dorsolateral nucleus of the anterior thalamus, DLM) inputs that may tonically activate LMAN during song. The total number of FF inputs to a premotor neuron is modelled as a constant drive \(I_0F\sqrt{K}/\sqrt{F}\). Similarly, the motor network receives a FF input from outside the circuit that we model as a constant drive. Importantly, the motor network also receives FF inputs from outside the circuit that we model as a constant drive. For large values of \(\sigma_{rec}\), distant neurons are as likely to be connected as close ones, while for small values of \(\sigma_{rec}\), only neurons which are close have a significant probability to be connected (Fig. 2h). In most of the results depicted in the paper, we assume that the recurrent interactions in the motor network have a wide footprint \((\sigma_{rec} = 2L)\), except for Fig. 2h,i, where we investigate how the results depend on the value of \(\sigma_{rec}\).

**The vocal organ.** We modelled the vocal tract as in Amador et al.\(^{24}\) In particular, we did not include the trachea or the Helmholtz filter, as these filters are species specific and in general will not affect gesture and inter-gesture durations. Two variables activate the vocal organ: tension and pressure. We modelled the pressure variable as \(P = \frac{1}{\sigma_{rec}} \cdot \max \{E, E_0\}\), where \(\sigma_{rec}\) is a rectified linear function. The tension is modelled as a linear combination of nine effectors: \(T = \frac{1}{\sigma_{rec}} \sum W_j E_j\), where, \(W_j\) (\(j = 2, \ldots, D\)), are random weights, \(W_j = N(0,1)\). Tension and pressure are then scaled to fit the dynamic range of the oscillating phase (see ref. 34).

\[
T = \mu_t + \nu_T z
\]

and

\[
Pr = \max\left(\frac{E - E_0}{P_{\max} - E_0}, 0\right)
\]

where \(z\) is the z-score of \(\sigma_{rec}\), \(\sigma_{rec}\), \(\mu_t\) and \(\nu_T\) are constant parameters that define the dynamic range and \(\sigma_{rec}\) is a bias that ensures that when there is no pressure the system is at a fixed point. We take: \(\mu_t = 0.6\); \(\sigma_{rec} = 0.2\); \(P_{\max} = 0.21\); \(b = 0.01\). The tension and pressure were then smoothed by a rectangular window of 20 ms and interpolated to a sampling frequency of 44,100 Hz. We then used the tension and pressure parameters to simulate the model by Amador et al.\(^{24}\) Finally, to reduce transient effects at the boundaries of the gestures (as a result of crossing the bifurcation) generated by the vocal tract model, the sound signal was taken as the product of the output model and the Pr signal.

**Model parameters.** Unless specified otherwise, the parameters used in the simulations were: \(N = 10,000; K = 4000; D = 10; \tau_m = 10\); \(\tau_d = 10\) ms. In the simulations depicted in Fig. 1, synaptic strengths and external FF inputs were: \(I_{f0} = 0.3\); \(I_{f0} = 3\); \(I_{f0} = -1.5\); \(\tau_f = -2\); \(\tau_f = 0.2\); \(\tau_f = 0.1\) for the premotor network as well as for the motor network. All synaptic time constants were 3 ms and \(\tau_d = 5\). In Fig. 3, the parameters in the premotor network were: \(I_{f0} = 0.3\); \(I_{f0} = 6\); \(I_{f0} = -1.8\); \(\tau_f = -2.2\); \(\tau_f = 0.8\); \(\tau_f = 0.2\) and for the motor network: \(I_{f0} = 5\); \(I_{f0} = 0.5\); \(\tau_f = 0.75\); \(\tau_f = -0.75\); \(\tau_f = -1\); \(\tau_f = 0.05\); \(\tau_f = 0.025\); \(\tau_f = 5\) ms. All synaptic time constants were 3 ms except for the pre-motor-to-motor pathway to the excitatory neurons in the motor network, which represents the slow NMDA synapses in the LMAN–RA pathways. The parameters used in the simulations depicted in Figs 4 and 6 were chosen such that the mean firing rates of the neurons in the premotor and motor networks were in agreement with previous experimental data as well as our own data in LMAN and RA. Given these parameters, the average firing rates of excitatory and inhibitory neurons in the premotor network were 14.7 and 46.8 Hz, consistent with our data and with previous reports for adult and juvenile finches.\(^{10}\) The mean firing rates in the motor network were 40 Hz for the E cells and 100 Hz for the I cells, as reported for RA neurons. The time constant of AMPA- and GABA_A-mediated synapses are all taken to be 3 ms. NMDA-mediated synapses in the premotor-to-motor pathway are modelled in a minimal manner, neglecting their voltage dependence, with very fast (instantaneous) rise and slow exponential decay with time constants of \(\sim 100\) ms, in line with experiments\(^{31}\).
Numerical integration. The dynamics of the model circuit were numerically integrated using the Euler method supplemented with an interpolation estimate of the spike times\(^4\). In all simulations the integration time step was 0.1 ms. We verified the validity of the results by performing complementary simulations with smaller time steps.

Autocovariance and crosscovariance of spike activities. Neuronal spike trains were filtered with an exponential kernel (time constant = 5 ms). ACs and CCs of neuronal activities were estimated from the resulting smoothed signals. Population-averaged ACs and CCs were computed over all neurons in the corresponding population. The Pearson CC was defined as the crosscovariance normalized by the autocovariance at zero lag.

Measure of synchrony and variability of the effectors. We quantified the degree of synchrony in the activities of the premotor or motor network using the synchrony measure, \(\chi(M)\), defined by\(^5\):

\[
\chi^2 = \left( \frac{\text{Var}(m(M,t))}{\sum_i \text{Var}(v_i)} \right)
\]

where the sum is over a population of \(M\) neurons in the network, \(v(t)\) is the instantaneous firing rate of neuron \(i\) and \(m(M,t) = \frac{1}{M} \sum_i v_i(t)\) is the instantaneous firing rate averaged over the population of \(M\) neurons. Here, \(\text{Var}(v(t))\) denotes the variance of the temporal fluctuations of \(v(t)\) (and \(\bar{x}\) denotes the average over a large number of realizations of the population \(S\)). For \(1 < M < N\),

\[
\chi^2(M) \approx \alpha(N) + \frac{1}{M}
\]

where \(a\) and \(b\) are numbers which depend on the network parameters. By definition, the network is in an asynchronous state if \(\alpha\) vanishes for sufficiently large \(N\). In that case, pair-wise correlations are small, of the order of \(1/N\) and the population average firing rate is constant in time. In contrast, if \(\alpha\) converges to a non-zero value for large \(N\), the network is in a synchronous state. To quantify the variability of the inputs to the effectors (receiving inputs from \(M\) neurons in the motor network), \(E(M, 0) = \{1, \ldots, D\}\), we computed the coefficient of variation, \(CV_{\text{eff}}\) such that:

\[
CV_{\text{eff}}^2 = \frac{\left( \frac{1}{D} \sum_i \frac{\text{SD}(E_i)}{\text{Mean}(E_i)} \right)^2}{A + \frac{B}{M}}
\]

If the motor network is in an asynchronous state, \(A \sim \frac{1}{D}\), since \(E(t)\) is linearly related to the population averaged activity in a functional group \(I\).

Data availability. All relevant data and computer codes are available from the authors.

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\[
\chi^2 = \left( \frac{\text{Var}(m(M,t))}{\sum_i \text{Var}(v_i)} \right)
\]

where the sum is over a population of \(M\) neurons in the network, \(v(t)\) is the instantaneous firing rate of neuron \(i\) and \(m(M,t) = \frac{1}{M} \sum_i v_i(t)\) is the instantaneous firing rate averaged over the population of \(M\) neurons. Here, \(\text{Var}(v(t))\) denotes the variance of the temporal fluctuations of \(v(t)\) (and \(\bar{x}\) denotes the average over a large number of realizations of the population \(S\)). For \(1 < M < N\),

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\[
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\]

If the motor network is in an asynchronous state, \(A \sim \frac{1}{D}\), since \(E(t)\) is linearly related to the population averaged activity in a functional group \(I\).
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Author contributions

R.D. and A.L. performed the behavioural data analysis. S.P. provided the sparrows data. W.E.W. and A.L. performed the electrophysiological experiments. R.D., W.E.W. and A.L. performed the electrophysiological data analysis. R.D. and D.H. designed the theory and performed the simulations. R.D., A.L. and D.H. designed the study and wrote the manuscript.

Additional information

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