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Synchrony and idiosyncrasy in the gut microbiome of wild primates

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Abstract: Human gut microbial dynamics are highly individualized, making it challenging to link microbiota to health and to design universal microbiome therapies. This individuality is typically attributed to variation in diets, environments, and medications, but it could also emerge from fundamental ecological forces that shape primate microbiota more generally. Here we leverage extensive gut microbiome time series from wild baboons—hosts who experience little interindividual dietary and environmental heterogeneity—to test whether gut microbial dynamics are synchronized across hosts or largely idiosyncratic. Despite their shared lifestyles, we find strong evidence for idiosyncrasy. Over time, samples from the same baboon were much more similar than samples from different baboons, and host-specific factors collectively explained 30% of the deviance in microbiome dynamics, compared to just 3% for factors shared across hosts. Hence, individualization may be common to mammalian gut microbiota, and designing universal microbiome interventions may face challenges beyond heterogeneity in human lifestyles.

Introduction

Mammalian gut microbiomes are highly complex, dynamic ecosystems. From these dynamics emerge a set of life-sustaining services for hosts, which help them digest food, process toxins, and resist invading pathogens. Despite their importance, our understanding of gut microbial dynamics, especially the collective dynamics of microbial communities from hosts living in the same population, is remarkably poor (*1*). This gap exists in part because we lack time series data that track gut microbiota longitudinally across many hosts in the same population. As a result, we cannot answer key questions. For example, when host populations encounter shifting environments and resources, does each host's microbiota respond similarly—i.e., in synchrony—or idiosyncratically to these changes? Further, are microbial dynamics especially similar when hosts live in the same social unit or have shared traits, such as age, sex or social status?

Answering these questions is important because synchronized host microbiomes could help explain shared microbiome-associated traits in host populations, such as patterns of disease susceptibility (*2, 3*). A high degree of microbiome synchrony could also be good news for researchers working to predict microbiome dynamics because it would suggest that similar

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ecological principles govern microbiome dynamics across hosts (*4*). There is also theoretical justification to expect some degree of synchrony, as host populations and their microbiomes can be considered a 'microbiome metacommunity' (see e.g., *5, 6-8*). Metacommunity theory predicts that synchrony will arise across microbiomes if hosts experience similar environmental conditions and/or high rates of microbial dispersal between each host's microbiome (*9, 10*).

However, even in the presence of synchronizing forces like shared environments and high rates of microbial dispersal, there are many reasons why hosts in a microbiome metacommunity could exhibit idiosyncratic (i.e., individualized) microbiome compositions and dynamics. Idiosyncratic dynamics are expected when the same microbes in different hosts respond in different ways to environmental fluctuations, chance events, and/or interactions with other microbes (*11-14*). These forces are likely to be important in the gut microbiome where priority effects, functional redundancy, and horizontal gene flow can cause the same microbial taxon to perform different functions, play different ecological roles, and exhibit different environmental responses in different hosts (*15, 16*). Furthermore, in humans, gut microbiome dynamics are often described as "personalized" (*17, 18*). However, personalized dynamics in humans are nearly always attributed to large interpersonal differences in diet, medications, and lifestyles (*19-22*), and not to fundamentally different microbiome responses to the environment itself (*19*). If personalized dynamics persist in a different primate species, even in the presence of shared environments, this pattern would suggest that: (i) host-specific dynamics are a common feature of primate gut microbial communities (i.e., are not unique to humans and are not solely attributable to large interpersonal differences in human lifestyles); (ii) predicting gut microbial dynamics in individual hosts may prove difficult; and (iii) microbiome interventions to improve human health may face challenges beyond heterogeneity in human lifestyles, and instead may be related to the fundamental ecological principles that govern the gut microbiome.

Data and methods

Here we test the degree to which gut microbiome compositions and dynamics in a host population are synchronized versus idiosyncratic using extensive time series data from a population of wild baboons in the Amboseli ecosystem in Kenya (*23*). Baboons are terrestrial primates that live in stable social groups, typically with 20 to 130 members. The 600 baboons in

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our data set lived in 12 social groups over a 14-year span (April 2000 to September 2013; 5 original groups and 7 groups that were fission/fusion products from these original groups; **Fig. 1A**). The baboons were members of the well-studied Amboseli baboon population (*23*), which has been studied by the Amboseli Baboon Research Project since 1971. This project collected detailed longitudinal data on the weather the animals experienced; their social group memberships, ranging patterns and diets; and host traits such as age, sex, social relationships, and dominance ranks (see Supplementary Materials).

Importantly, like many natural host populations, the Amboseli baboons experience shared diets, environments, and opportunities for between-host microbial dispersal that could drive microbiome synchrony across hosts. Because baboons are not territorial, all 12 baboon social groups used an overlapping ~60 km^2 range (**Fig. 1B**; **video S1**; (*24*)). Hence all animals were exposed to similar microbes from the environment and shared seasonal changes in rainfall and temperature (*24-26*). The Amboseli ecosystem is a semi-arid savanna where very little rain falls from June to October, with highly variable rainfall between November and May (**Fig. 1C**; mean annual rainfall between 2000 and 2013 was 319 mm; range = 140 mm to 559 mm). These seasonal shifts in climate drive a rotating set of foods consumed by the baboons: during the dry season the baboons rely largely on grass corms, shifting to growing grass blades and grass seed heads in the wet season (**Fig. 1D**). Within baboon social groups, diets and environments are especially congruent because group members travel together in a coordinated fashion across the landscape, encountering and consuming resources and feeding on the same seasonally available foods at the same time (*24, 27-31*). Group members also groom each other, combing through each other's fur and placing some items in their mouths, which may contribute to host-to-host microbial transmission (*32*). Finally, at the level of individual hosts, host genetic variation has a consistent, albeit modest, effect on gut microbiome composition in this population (*24*). Other host-specific traits, like age, sex, and social status, also lead some individuals to share aspects of their behavior, immune profiles, and physiology, which could also lead to more congruent microbiome dynamics.

A key advance in our study is longitudinal sampling of gut microbial composition via 16S rRNA gene sequencing from fecal samples collected from hundreds of known baboons throughout their lives (**Fig. 1A**). Such dense, long-term, longitudinal microbiome sampling is difficult to achieve in many animals, including humans. The 17,265 fecal samples in our study

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were collected from baboons who ranged in age from 7.4 months to 27.7 years, spanning these animals' natural lifespans (**fig. S1A**). Each baboon was sampled a median of 19 times, and 124 baboons were sampled at least 50 times (**fig. S1B**). On average, these samples spanned 4.3 years of a baboon's life (range = 4 days to 13.2 years; **fig. S1C**), with a median of 35 days between consecutive samples (**fig. S1D**).

A large majority of the microbiome samples we use here were published in Grieneisen et al. (*24*), but we include 1,031 additional samples that were generated at the same time using the same methods (they were not included in Grieneisen et al. (*24*) because we lack pedigree information for these hosts). Briefly, we generated 896,911,162 sequencing reads (mean = 51,913.6 reads per sample; range = 1021 - 477,241, **fig. S1E**). We retained microbial amplicon sequence variants (ASVs) with a minimum of 3 reads per sample that were seen in at least 20% of the samples, resulting in 341 microbial taxa at the ASV level (mean = 162 ASVs per sample; range = 19 - 311 ASVs; **fig S1F**). DNA concentration and ASV diversity were not predicted by time since sample collection (**fig. S1G, S1H**). Read counts were centered log-ratio transformed prior to all subsequent analyses (*33*, *34*).

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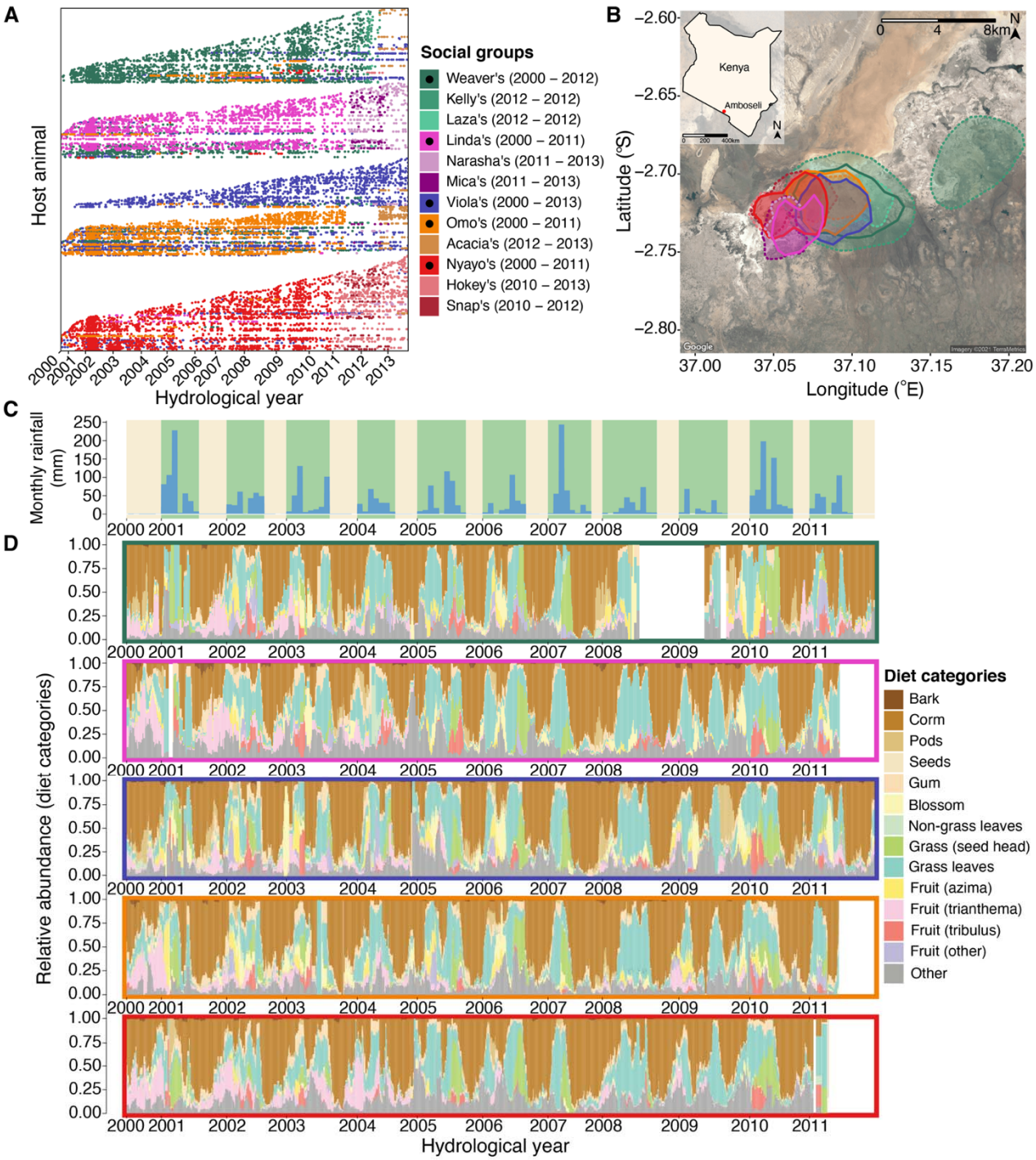

Fig. 1. Baboons in Amboseli experience shared environments at multiple scales. (A) The microbiome time series consisted of 17,265 16S rRNA gut microbiome profiles. Each point represents a microbiome sample, plotted by the date it was collected (x-axis). Each row (y-axis) corresponds to a unique individual host. Samples were collected from 600 wild baboons living in 5 original social groups (indicated by dark colors marked with black dots in the legend) and 7

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groups that fissioned/fused from these original groups (no black dots). **(B)** All baboon groups ranged over a shared ~60 km^2 area, and the social groups had largely overlapping home ranges. Ranges are shown as 90% kernel densities over the sampling period specific to each group; 5 original social groups are shown with solid borders, fission and fusion products with dashed borders. **(C)** Monthly rainfall amounts (blue bars, in mm) with yellow and green stripes representing dry and wet seasons, respectively, with the width reflecting the number of months within the focal year that had at least 1 mm rainfall. **(D)** Temporal shifts in diet from the years 2000 – 2013, shown as the relative abundance of diet components in the 5 original social groups over 30-day sliding windows. Colors correspond to the 13 most common food types, while the grey bars correspond to other or unknown food types. Colored boxes around each panel reflect each of the 5 original, most extensively sampled social groups (colors as in plots A and B). The white bars indicate time periods where no diet data were collected.

To test whether shared environmental conditions and host traits lead to similar gut microbial compositions and synchronized dynamics across the microbiome metacommunity, we used three main approaches (see Supplementary Materials for details of all analyses). First, we characterized patterns of temporal autocorrelation to identify hallmarks of compositional similarity and synchrony over time. Our expectation was that, if different baboons exhibit similar gut microbiome compositions and synchronized microbiome dynamics, then samples collected close in time across the metacommunity should be compositionally similar, and samples collected from the same host should not be substantially more similar than samples from different baboons. Alternatively, if hosts or social groups exhibit idiosyncratic compositions and dynamics, then samples collected close in time from the same baboon, or the same group, should be much more similar than they are to samples collected from different baboons living in different groups. These analyses were run in R (v 4.0.2; (*35*)) using custom-written functions (code and analyzed data are available on GitHub/OSF; see Data Statement).

Second, to test whether dispersal limitation could explain microbiome idiosyncrasy, we estimated metacommunity-wide microbial migration probabilities in each season and year using the Sloan Neutral Community Model for Prokaryotes (*36, 37*). This model assumes that each local community, defined as the microbial composition of a single host in a given season-year

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combination, is the outcome of stochastic population dynamics and microbial immigration from other hosts in the microbiome metacommunity (i.e., other local communities). Briefly, local communities have a constant size *N*, and individual microbes within each local community die at a constant rate. These deaths create vacancies that can be occupied, either by individuals immigrating from the microbiome metacommunity (with probability *m*), or by the offspring from any taxon within the local community (i.e., from reproduction within the same host, with probability 1-*m*). Species that are common in the metacommunity have a higher chance of occupying vacancies than rare species. Without immigration from the microbiome metacommunity, ecological drift leads each host's microbial diversity to reduce to a single taxon. Thus, the migration probability, *m*, represents the metacommunity-wide probability that any taxon, randomly lost from a given host/local community, will be replaced by dispersal from the microbiome metacommunity, as opposed to reproduction within hosts (*36*, *37*). Following Burns et al. (*38*), *m* can be interpreted as a measure of dispersal limitation, such that low migration probabilities signify high dispersal limitation. We estimated season and hydrological year-specific values for *m* by defining the microbiome metacommunity as either the hosts' social group or the whole host population. We fit neutral models using nonlinear least-squares regression as implemented in the R package tyRa (*39*).

Third, to quantify the relative magnitude of idiosyncratic versus synchronized gut microbiome dynamics for different microbiome features, we used generalized additive models (GAMs) to capture non-linear, longitudinal changes in 52 gut microbiome features, including three principal components of microbial community variation, three indices of alpha diversity (species richness, the exponent of Shannon's H, and the inverse Simpson index, as computed by the function reyni from the R package vegan (*40*)), and the relative abundances of all 12 phyla and 34 families present in our data set, post filtering. GAMs allowed us to calculate the percent deviance in each feature's dynamics attributable to factors that could contribute to synchronized dynamics at different scales; percent deviance is a measure of goodness-of-fit for nonlinear models and is analogous to the unadjusted R^2 for linear models. We considered three scales: factors experienced by the whole host population (e.g., rainfall and temperature), those differentiated by social groups (e.g., group identity, group home range location, and diet), and those differentiated at the level of individual hosts (e.g., host identity, sex, age, and social dominance rank; see below for complete model structures). If shared environments and traits

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synchronize gut microbiome dynamics across hosts, these factors should explain substantial deviance in microbiome dynamics. Alternatively, if microbiome dynamics are idiosyncratic, population- and group-level factors will not explain considerable deviance and, instead, a large fraction of the deviance will be attributable to host identity, controlling for shared environments, behaviors, and traits. To ensure sufficiently dense sampling for identifying host- and group-level dynamics, all three models were run on a subset of the full data set, consisting of 4,277 16S rRNA gene sequencing profiles from the 56 best-sampled baboons living in the 5 social groups sampled the longest (between 2002 and 2010; min=48; median = 72.5; max = 164 samples; **fig. S2**). GAMs were fit using the R package mgcv (*41-43*).

Notably, the GAM approach allows us to identify the percent deviance attributable to host identity, but does not identify the specific characteristics that account for host identity effects. Genetic effects are a likely candidate, as previous analyses demonstrate that taxon abundance and summaries of gut microbiome position are lowly to moderately heritable in this population (*24*). To evaluate this possibility, we tested the relationship between the deviance explained in our GAMs for each microbiome taxon and the heritability of that taxon's relative abundance (*24*). If host effects on microbiome dynamics are in part explained by host genotype, we predicted that taxon heritability should be positively correlated with deviance explained at the host level (i.e., model P+G+H), but not at the group or population level (i.e., model P and model P+G).

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Results and Discussion

Baboon gut microbiota exhibit cyclical shifts in community composition across seasons and years

We began by visualizing annual and inter-annual fluctuations across the gut microbiome metacommunity over the 14-year span of the data. Consistent with prior research on primates (*44-46*), we found population-wide, cyclical shifts in microbiome composition across seasons and years (**Fig. 2**). This wet-dry seasonal cyclicity was primarily observable in the first principal component (PC1) of a principal component analysis (PCA) of clr-transformed read counts for all 17,265 samples (**Fig. 2A, 2B**; **fig. S3-S5**; PC1 explains 16.5% of the variance in microbiome community composition). PC1 tended to exhibit low values during the dry season, and high values during the wet season, mirroring monthly rainfall (**Fig. 2B; fig. S5**). PC1 was also linked to annual rainfall across years, exhibiting especially low values throughout 2008 and 2009, which corresponded to the worst continuous drought in the Amboseli ecosystem in 50 years (**Fig. 2A, 2B**). We also observed small, but statistically significant seasonal differences in PC2 and PC3 (8.4% and 3.7% of variation in community composition; **Fig. 2C**; **fig. S3-S5**) and in measures of alpha diversity (**Fig. 2C**; **fig. S5, S6**), as has been reported in other ecosystems (*47*).

In terms of individual microbiome taxa, 17% of phyla (2 of 12) and 38% of families (13 of 34) exhibited significant changes in relative abundance between the wet and dry seasons (**Fig. 2C**; **table S1**; linear models with a false discovery rate (FDR) threshold = 0.05 for n = 393 models). These changes were significant for the phyla Firmicutes and Tenericutes (**Fig. 2C, 2D**; **fig. S7**), and were especially pronounced for the families Helicobacteraceae, Coriobacteriaceae, Burkholderaceae, Bacteroidales RF16 group, vadinBE97, Spirochaetaceae, and Campylobacteraceae (**Fig. 2C**; **fig. S8**). 28% of ASVs also exhibited significant changes in abundance across seasons (97 of 341 ASVs; linear models with FDR threshold = 0.05 for n = 393 models; **fig. S9**; **table S2**). The majority of gut microbial taxa at the ASV, family and phylum level did not exhibit significant changes in abundance across seasons, suggesting that these taxa play consistent roles in the gut ecosystem throughout the year, including Kiritimatiellaeota, Elusomicrobia, Ruminococcacaceae, Clostridiaceae 1, and Rikenellaceae (**Fig. 2C**; **fig. S7, S8**; **table S1**).

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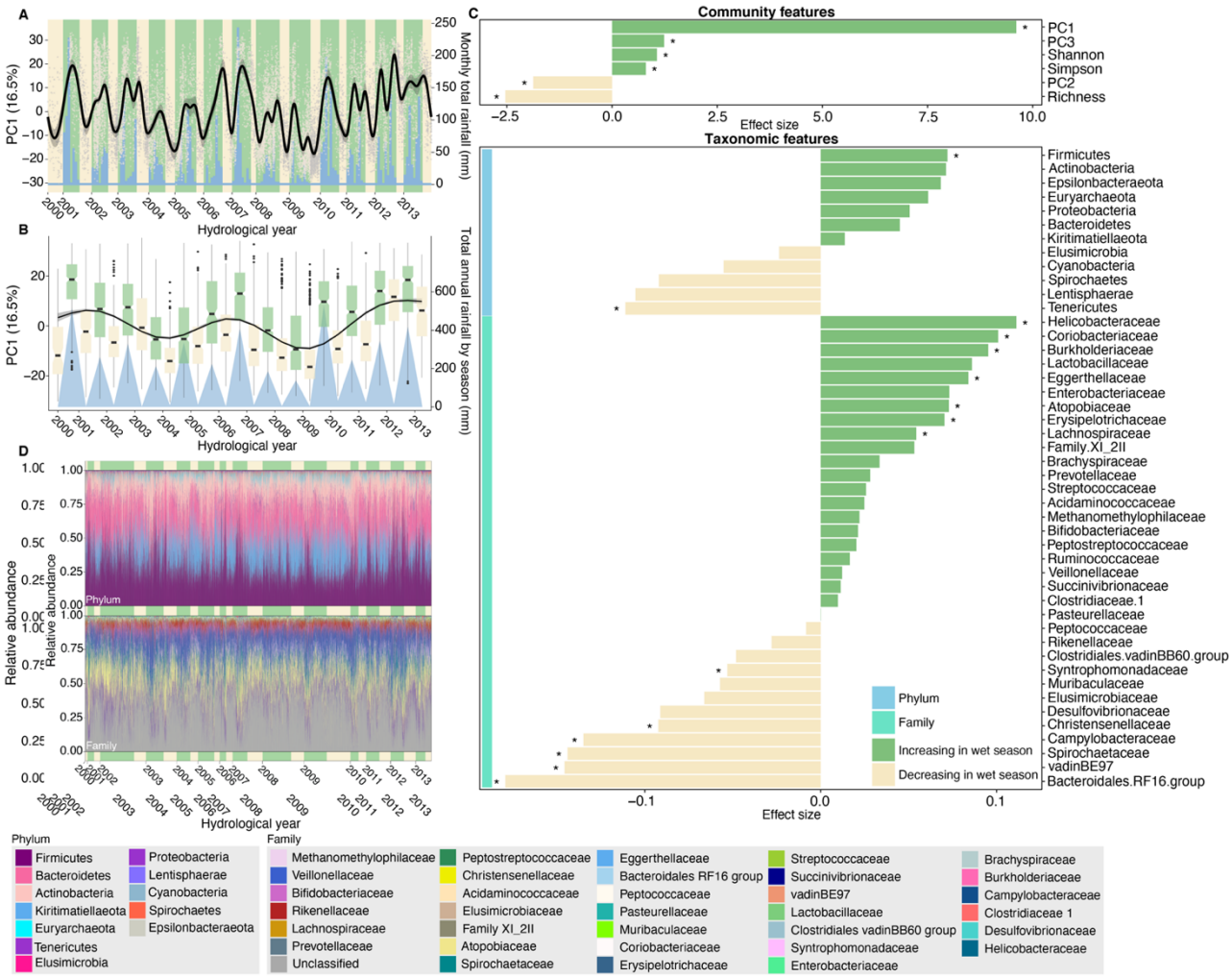

Fig. 2. Baboons show population-wide, cyclical shifts in microbiome composition across seasons and years. (A) Changes in microbiome PC1 mirror monthly rainfall across the 14 years of the data set. The grey points show each sample's value for PC1 (y-axis) on the date it was collected. The black line shows the predicted daily trend for PC1 across samples, treating time (x-axis) as a continuous variable from April 21, 2000 to September 19, 2013. The corresponding gray ribbon shows the 95% simultaneous confidence interval. Blue bars show monthly rainfall (right-hand y-axis). Yellow and green bars in the background represent dry and wet seasons, respectively, with the width reflecting the number of months within the focal year with at least 1 mm rainfall. **(B)** Changes in microbiome PC1 on an annual scale across the 14 years of the data set. The box plots show the average distribution of microbiome PC1 in wet (green) and dry (yellow) seasons. The black line shows the estimated annual trend for PC1 across all hydrological years, and the blue triangles show total annual rainfall (right-hand y-axis). **(C)** The

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effect of season varies across 52 features of the microbiome, including six community features (top panel) and 46 taxonomic features (bottom panel; 12 phyla: light blue vertical bar; 34 families: turquoise vertical bar; for 341 ASVs, see Fig. S12). Each horizontal bar shows the effect of season from linear mixed models, with each feature as the dependent variable. Asterisks indicate features that changed significantly between the wet and dry seasons (FDR threshold = 0.05 for n = 393 models). See **figs. S7, S8** for feature-specific smooths and **fig. S9** and **table S2** for results for ASVs. Samples from the same host collected on the same date were averaged prior to running the linear models. **(D)** Bar plots showing the relative abundance of all 12 microbial phyla (above) and 34 families (below) across all samples. Green and yellow bars in the background represent wet and dry seasons, with the width corresponding to the number of samples in the focal hydrological year and season.

Baboons exhibit largely idiosyncratic gut microbiome compositions and dynamics

While the microbiome metacommunity exhibited cyclical, seasonal shifts in composition, microbiome dynamics across different baboons were not strongly synchronized. Instead, patterns of temporal autocorrelation indicated that each baboon exhibited largely individualized gut microbiome compositions and dynamics (**Fig. 3**). In support, samples collected from the same baboon within a few days were much more similar to each other than they were to samples collected from different baboons over the same time span, regardless of whether those animals lived in the same or a different social group (**Fig 3A, 3B**; Kruskal-Wallis: $p < 2.2x10^{-16}$ for all comparisons). Likewise, a PERMANOVA of Aitchison distances between all samples revealed that host identity explained 8.6% ($p < 0.001$) of the variation in community composition, much larger than sampling day or month (r^2 = 2.5% and 1.4%), group membership (2.2%), or the first three principal components of diet (0.04% to 2.4%; **table S3**; **fig. S10**).

Compositional similarity among samples from the same baboon fell steeply for samples collected a few days to a few months apart (**Fig. 3A, 3C**). However, similarity rose again slightly at 12-month intervals, reflecting the seasonal dynamics in **Fig. 2**. These 12-month peaks in similarity were visible, even for samples collected more than 5 years apart, indicating that individual hosts and the population at large return to somewhat similar microbiome community states on 12-month cycles across years (**Fig. 3C**). Individualized host compositional signatures

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persisted for several years (**Fig. 3A, 3C**; **fig. S11**). Indeed, the 95% confidence intervals on Aitchison similarity between samples collected from the same vs different hosts rarely overlapped for samples collected less than two years apart (**fig. S11B, S11C**).

Individualized gut microbiome dynamics can also be seen by visualizing microbiome compositional similarity between hosts living in the population at the same time (**Fig. 3D**; **fig. S12**). For instance, especially dense sampling during the 2008-2009 hydrological year meant that we were able to collect at least one sample, for at least 10 months of the year, from 17 of our study subjects. When we aligned these time series, we observed no shared pattern of change in the top three principal components of microbiome composition across time beyond some overall seasonal patterns in PC1, nor did we see convergence to similar values within any given month (**Fig. 3D**). Consequently, the microbiome of each baboon took a different path over the ordination space over the same 1-year span (**fig. S12**). We found similar results for another dense sampling period in the 2007-2008 hydrological year (**fig. S13**).

Microbiome taxa varied in their contributions to individualized gut microbiome compositions (**Fig. 3E**; **fig. S14**). For example, for the 56 best-sampled hosts (**fig. S2**), several phyla and families exhibited substantial variation in host mean (clr-transformed) relative abundance (i.e., across repeated samples for that host) compared to their mean (clr-transformed) relative abundance across all hosts. These taxa included members of the phyla Cyanobacteria, Spirochaetes, Lentisphaerae, and Elusimicrobia, and the families Spirochaetaceae, vadinBE97, Elusimicrobaceae, and Muribaculaceae (**Fig. 3E**; **fig. S14**). These highly variable taxa tended to exhibit, on average, below-average abundance compared to less variable taxa that tended to exhibit, on average, above-average abundance, indicating that idiosyncratic dynamics may be more often linked to uncommon than common taxa (**fig. S15**).

To test whether individualized gut microbiome compositions and dynamics could be explained by microbial dispersal limitation between hosts, we used the Sloan Neutral Community Model for Prokaryotes to estimate metacommunity-wide migration probabilities, m, for each season and hydrological year (*36*, *37*). As described above, m provides a measure of dispersal limitation because it represents the probability that "vacancies" in a local community (i.e. a host's microbiome) will be replaced by the process of dispersal from the microbiome metacommunity (i.e. other hosts), as opposed to reproduction within a focal host's microbial

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community (*36, 37*). We found little evidence that dispersal limitation contributed to idiosyncratic compositions and dynamics; the estimated probability that a given ASV lost from a host's microbiota would be replaced by an ASV from another host in the population was nearly 40% (the average host population-wide across season and hydrological years m = 0.373; range = 0.332 to 0.416; black points on **fig. S16**). These migration probabilities are generally lower than those Sieber et al. (*8*) found for marine sponges sampled from the same coastal location (range of m across sponge species: min=0.36; median=0.78; max=0.86) but much higher than for mice and nematodes, both in natural and laboratory populations (mice: m_{wild} = 0.11 and m_{lab} = 0.18; nematode: m_{wild} = 0.03 and m_{lab} = 0.01), indicating that dispersal limitation is relatively low for baboon microbiota in Amboseli.

Interestingly, when we re-defined the microbiome metacommunity to be the host's social group, instead of the whole host population, migration probabilities were similar (average m across groups = 0.355; range = 0.347 to 0.365; colored points on **fig. S16**). Hence, social group membership likely does not represent a large barrier to microbial colonization between baboons, as ASVs are widely shared across all members of the host population.

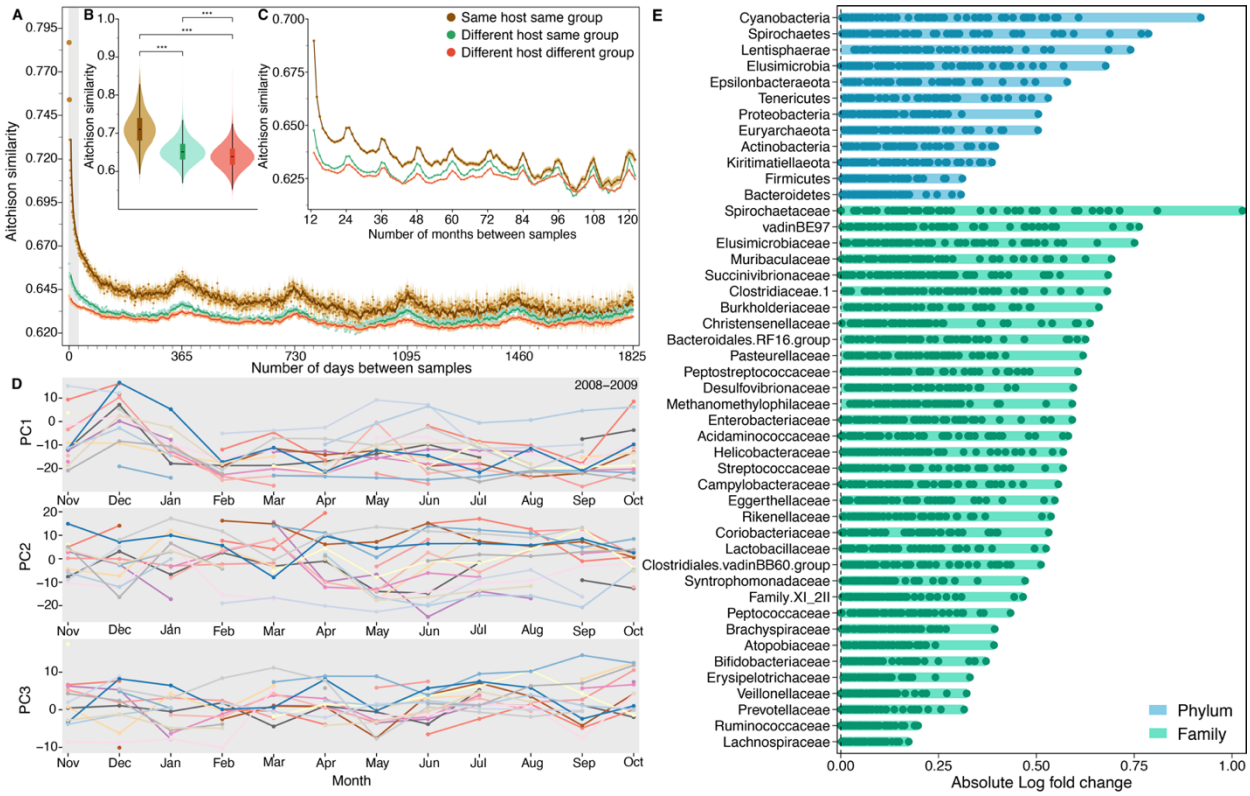

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Fig. 3. Baboons exhibit idiosyncratic gut microbiome compositions and dynamics. (A) Temporal autocorrelation in microbiome Aitchison similarity (y-axis) as a function of the time between samples, plotted on a daily scale (x-axis), ranging from samples collected on the same day to samples collected 5 years apart. Small tick marks correspond to months. Brown points show average Aitchison similarity between samples collected from the same baboon; green points show similarity between samples from different baboons living in the same social group; orange points show similarity between samples from different baboons living in different social groups. The lines represent moving averages (window size = 7 days). **(B)** Average Aitchison similarity among samples collected within 10 days of each other. Samples from the same baboon are significantly more similar than samples collected from different baboons in the same or different social groups (Kruskal-Wallis; $p = 2.22 \times 10^{-16}$). **(C)** Temporal autocorrelation in microbiome Aitchison similarity on monthly scales for samples collected up to 10 years apart. **(D)** Microbiome dynamics for 17 baboons for which we had at least one sample from 10 of the 12 months of the 2008-2009 hydrological year (Nov 2008 to Oct 2009). Panels show each individual's values for microbiome PC1, PC2, and PC3; each colored line represents a distinct host. See **fig. S13** for similar results during another densely sampled time period. Gaps indicate that the focal host did not have a sample in a given month. **(E)** Some taxa have more idiosyncratic abundances than others. Each horizontal bar shows a given taxon's minimum and maximum absolute log fold change in abundance across the 56 best-sampled hosts (hosts are represented as points within the bars; see **fig. S2** for information on the best-sampled hosts). Absolute fold changes were calculated, for a given taxon in a given host, as the taxon's average clr-transformed abundance across all samples from that host, relative to the taxon's grand mean in all hosts in the population. Hosts with large absolute fold changes for a given taxon therefore have abundances of that taxon that are either well above or below-average compared to its abundance in the host population at large (hosts with points close to zero exhibited taxonomic abundances typical of the population at large). For many taxa, hosts varied in their absolute log ratio values, indicating that they also deviated substantially from each other in the abundance of those taxa. Taxa (y-axis) are ordered (from top to bottom) by their highest absolute log ratio value across the 56 best-sampled hosts. Blue bars represent microbial phyla; green bars represent families. See **fig. S14** for a longitudinal version of this analysis for the most and least idiosyncratic phyla and families.

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Shared environmental conditions are linked to modest synchrony across hosts

To quantify the relative magnitude of idiosyncratic versus synchronized gut microbiome dynamics across the host population, social groups, and individual hosts, and to test whether synchrony varies for different microbiome features, we used generalized additive models (GAMs) to capture the nonlinear, longitudinal changes in 52 microbiome features (3 PCs of community variation, 3 metrics of alpha diversity, and clr-transformed relative abundances of 12 phyla and 34 families). For each feature, we ran three GAMs to measure the deviance explained in gut microbiome dynamics by successive sets of parameters, reflecting the nested nature of our variables (**Fig. 4A**; x-axis of **Fig. 4C**; **table S4**). The population-level model (i.e., model P) captured factors experienced by the whole host population, including average rainfall and maximum daily temperature in the 30 days before sample collection and random effect splines to capture monthly and annual cyclicity in microbiome features (e.g., **Fig. 2A and B**). The group-level model (i.e., model P+G) included all the predictor variables in model P, and added a random effect spline for each social group, as well as variables to capture temporal changes in each group's diet, home range use, and group size (**Fig. 4A, 4C**). The host-level model (i.e., model P+G+H) included all of the predictor variables in model P+G, and added a random effect spline for each host, and variables for host traits, including sex, age, and social dominance rank (**Fig. 4A, 4C**).

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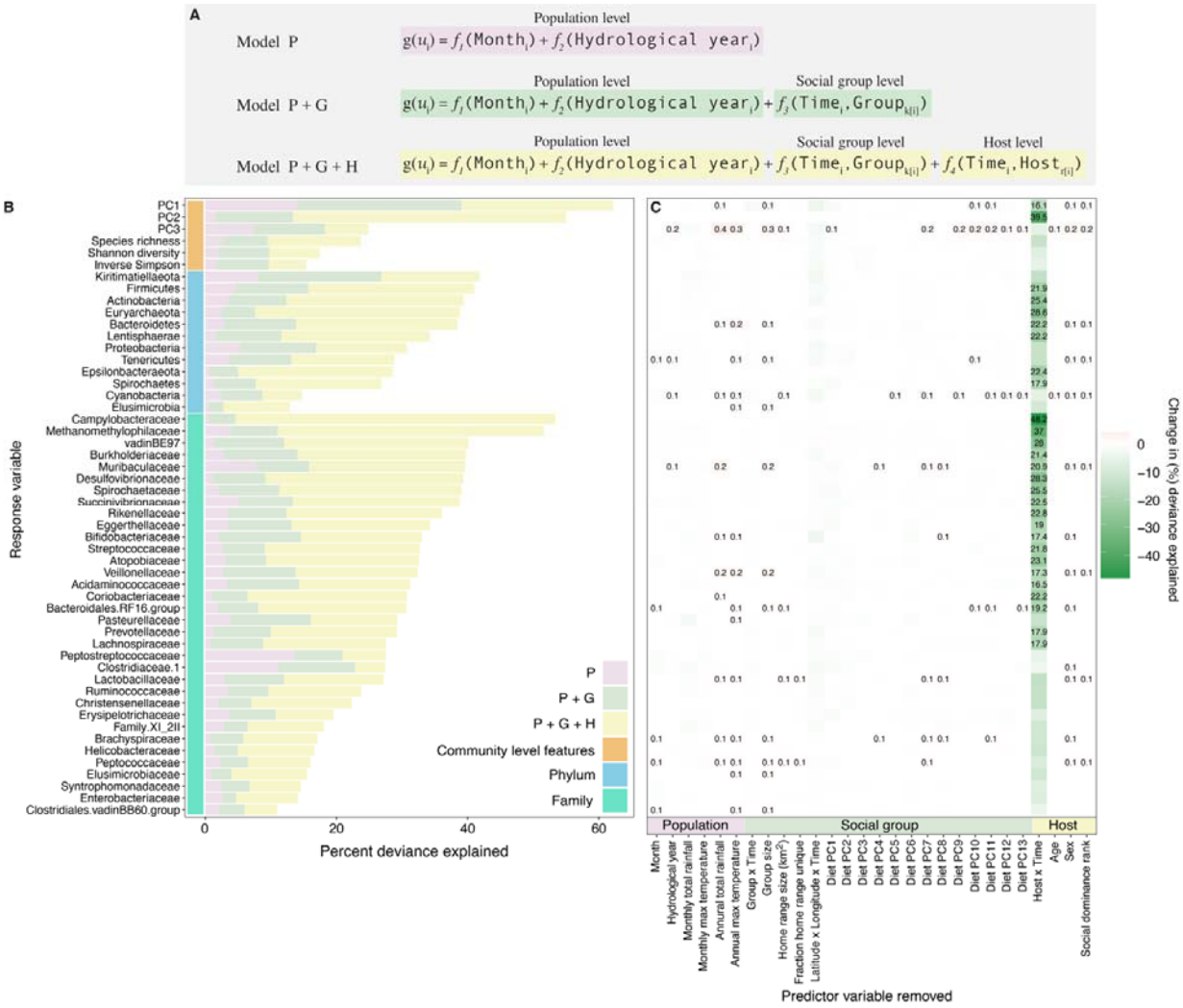

Fig. 4. Multilevel modeling identifies idiosyncratic dynamics. (**A**) We fit three hierarchical GAMs to 52 microbiome features measured in 4,277 samples from the 56 best-sampled baboons living in the 5 social groups sampled the longest (between 2002 and 2010; min=48; median = 72.5; max = 164 samples; **fig. S2**). Each model contained successive sets of predictor variables reflecting population-level factors (pink), group-level factors (green) and host-level factors (yellow). The factors at each level are listed at the bottom of panel C and defined in **table S4**). Panel (**B**) shows for each microbiome feature (i.e., response variable), the deviance explained by model P and the successive sets of predictor variables added in model P+G and model P+G+H, respectively (**table S5**). Panel (**C**) shows the loss in deviance explained for model P+G+H as we successively removed each predictor variable in turn from model P+G+H, keeping the model otherwise intact (**table S6**). Losses in deviance are shown in green, and we only provide numeric

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values for losses in deviance > 15%. Gains in deviance are shown in red; we only show numeric values for gains > 0.1%.

Consistent with our autocorrelation analyses (**Fig. 3**), comparing the deviance explained for each microbiome feature across the three models revealed primarily idiosyncratic dynamics for most microbiome features (**Fig. 4B, 4C**). Specifically, model P only explained on average 3.3% (range = 0.46% to 14.0%) of the deviance across all 52 microbiome features (pink bars in **Fig 4B**; **table S5**), compared to 8.1% on average for adding group-level factors to the population-level model (increase from model P to model P+G; range = 2% to 25%; green bars in **Fig. 4B**; **table S5**), and 30.1% of the deviance for including host-level dynamics (model P+G+H; range = 11.0% to 62.2%) in the same set of features (yellow bars in **Fig. 4B**; **table S5**). Importantly, the added deviance for model P+G+H compared to model P or model P+G was not simply caused by including more parameters. Specifically, randomizing host identity and traits across samples, while keeping each sample's annual, seasonal, and group identity intact, led to a substantial drop in deviance explained relative to the real data (**fig. S17**). For instance, for PC2, which captured the strongest host-level effects of all three PCs, the deviance explained by model P+G+H dropped from 55% to 16.6% when host identity and traits were randomized (**fig. S17;** see supplement and **fig. S18** for an additional analysis investigating the effect of model complexity on deviance explained). That said, for PC3, the addition of randomized host-level dynamics still resulted in more than negligible deviance explained relative to the real data (3% vs 6.6%) suggesting that deviance explained may be inflated for some microbiome features.

44 of the 52 microbiome features exhibited greater gains in deviance explained by adding host-level factors to model P+G, compared to adding group-level factors to model P, with 22 features gaining more than 20% deviance explained between model P+G and model P+G+H (**Fig. 4B**; **table S5**). Three of the most common phyla, Actinobacteria, Bacteroidetes, and Firmicutes all gained >20% deviance explained between model P+G and model P+G+H (Actinobacteria = 27.1%; Bacteroidetes = 24.6%, and Firmicutes = 25.2%; **Fig. 4B**; **table S5**). The most idiosyncratic features (i.e., those that gained >30% deviance explained by adding host-level factors), were microbiome PC2, the phylum Euryarchaeota, and the families Campylobacteraceae, Methanomethylophilaceae and Desulfovibrionaceae (**Fig. 4B**; **table S5**).

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Notably, even the most synchronous feature, microbiome PC1 (14% deviance explained by the P model), gained 23.2% deviance explained when adding host-level factors to the P+G model.

Removing covariates from model P+G+H one at a time, while keeping all other covariates intact, revealed that host identity explained nearly all of the deviance in our models (**Fig. 4C**; **table S5**; average loss in deviance explained by removing host identity = 17.3% compared to 0.2% deviance for all other factors). Beyond host identity, the next most important factor was the geographic area where the group had been travelling in the 30 days prior to sample collection, which on average, explained 1% of the deviance across all 52 features, with the strongest effects on microbiome PC1, Bifidobacteraceae, and Kiritimatiellaeota (**fig. S19**; **table S5**). The removal of all other individual predictor variables had only minor effects on deviance explained (**fig. S19**; **table S5**).

To investigate whether some of the idiosyncrasy we observed, especially at the host level, was due to genetic effects, we tested for a relationship between the deviance explained by each GAM and the narrow-sense heritability (h^2) of microbiome taxon abundance as estimated by Grieneisen et al. (*24*). We found that higher levels of deviance explained by model P+G+H were predicted by higher taxon heritability (Pearson correlation: R=0.37, p=0.016; **Fig. 5A**). Reassuringly, we found no such effect at the population or group level, as expected since genotype is a property of individual hosts, not groups or populations (model P+G: R=0.047, p=0.76; model P: R=0.0085, p=0.96; **Fig. 5B**). In particular, we explained substantially more deviance by adding the host level to model P+G for microbiome taxa with moderate to high h^2 values (i.e., those > 0.05) than we did for taxa with low h^2 values (model P+G+H: min=16.0, median=32.6, max=53.4 vs model P+G: min=4.6, median=11.1, max=26.8; **Fig. 5B**). These results suggest that some idiosyncrasy in gut microbiome dynamics is a consequence of host differences in genotype. We note, however, that because h^2 estimates from the animal model cannot be mapped directly onto estimates of deviance explained in GAMs, direct estimates of genetic versus environmental effects on host dynamics remain an important topic for future work.

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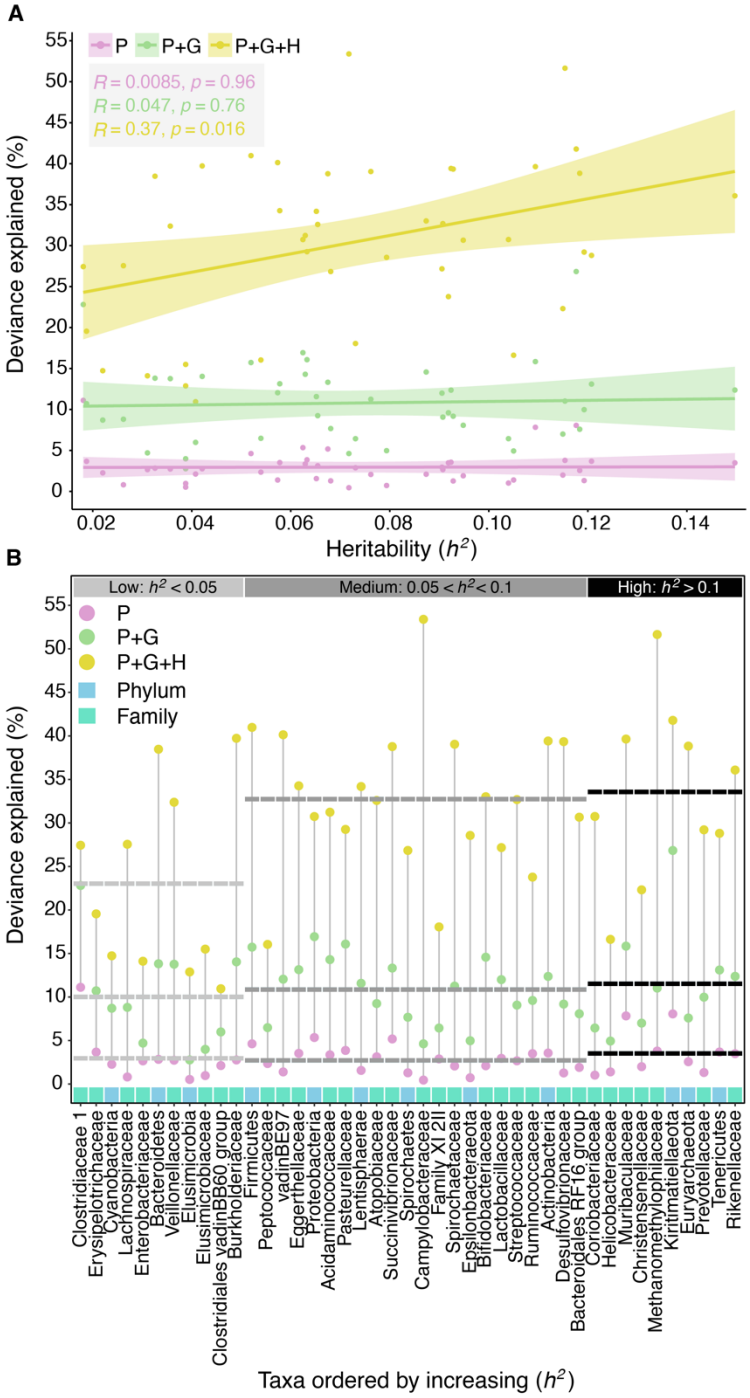

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462 **Fig. 5. Microbiome taxon heritability is associated with idiosyncratic dynamics.** (**A**)
463 Deviance explained (y-axis) by the phylum and family level GAMs (from **Fig. 4**) plotted against
464 the focal taxon's heritability estimate (h^2; x-axis). Pink, green and yellow denote model P, model
465 P+G and model P+G+H, respectively. (**B**) Deviance explained (y-axis) across the model
466 hierarchy (pink: model P; green: model P+G; yellow: model P+G+H) for each taxonomic feature

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(i.e., at the phylum and family level; x-axis). The x-axis is ordered by increasing heritability with light blue and turquoise squares representing phyla and families, respectively. Horizontal dashed lines show the average deviance explained per model for taxa with low heritability estimates ($h^2 < 0.05$; light gray); medium heritability estimates ($0.05 < h^2 < 0.1$; dark gray); and high heritability estimates ($h^2 \geq 0.1$; black).

Gut microbiome dynamics among social group members are more synchronized than for the host population at large

Previous research in humans and other social mammals, including the Amboseli baboons, finds that hosts in the same social group often have more similar gut microbiome compositions than hosts in different social groups (e.g. *32*, *48-50*). Likewise, in our current data set, several taxa exhibited abundances that were, on average, higher or lower within a given social group compared to their average abundance in the host population at large (**fig. S20, S21**). Hence, we tested whether shared social group membership is linked to greater microbiome synchrony than hosts in different groups. In support, the patterns of temporal autocorrelation in **Fig. 3A** showed that hosts in the same group have detectably more similar microbiomes than those in different groups, especially for samples collected within 10 days of each other (**Fig. 3B**; Kruskal-Wallis: $p < 2.2\text{x}10^{-16}$). Likewise, samples from the same group tended to occupy similar ordination space over time (**video S2**). While small, these group-level similarities were detectable, even for samples collected more than 2 years apart (**Fig. 3C**; **fig. S11A**). The addition of group-level splines to our GAMs led to gains in deviance that explained more than 10% for 15 of 52 microbiome features, including all three microbiome PCs, five phyla, and seven families (**Fig. 4B, 4C**; **table S5**). Several of these taxa were abundant in hosts, such as Firmicutes, Bacteroidetes, and Bifidobacteriaceae (**Fig. 4B, 4C**; **table S5**).

Because each social group has a somewhat distinctive gut microbiota, the effects of climate and diet on microbiome dynamics may differ across groups. To test this idea, we added interaction effects between group identity and climate variables (rain and temperature), or between group identity and the first three PCs of diet to model P+G+H. However, these interactions did not lead to substantial gains in deviance explained in our models (**fig. S22**; **table S7**). For instance, adding the climate interactions explained on average an additional 0.95%

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deviance across all 52 features (range = -1.9% to 5.4%; **table S7**), and diet interactions explained, on average, an additional 1.2% deviance across all 52 features (range = -0.7% to 5.6%; **table S7**).

Gut microbial congruence among group members could also be linked to shared behaviors and environments: baboons in the same group eat the same foods at the same time, travel as a unit across the landscape, and may be grooming partners that are frequently in physical contact (**Fig. 1B, 1C**; **video S1**; (*24, 27-31*)). Indeed, after host identity, the next most important predictor variable in model P+G+H was the group's home range in the 30 days before sample collection (**fig. S19**; **table S6**). Despite previous evidence for increased similarity in microbiome profiles among grooming partners in the Amboseli baboons (*32*), we did not find evidence for this pattern in our current data set (**fig. S23**). Indeed, samples collected within 30 days of each other from individuals with strong grooming bonds were not substantially more similar than samples from animals with weak or no observed grooming relationship (mean Aitchison similarity between pairs with strong bonds = 0.645; mean Aitchison similarity between pairs weak or no bond = 0.646; **fig. S24**). Because of differences in methodology, the lack of a grooming effect in this data set should be interpreted with caution. Our prior research on this topic (*32*) characterized microbial communities using shotgun metagenomic sequencing from >90% of social network members, all within 30 days of each other. In contrast, this current data set relies on 16S rRNA gene sequencing data from sparsely-sampled networks. Shotgun metagenomic data provide much higher taxonomic resolution than 16S rRNA identities, and may therefore more accurately capture the direct transmission between hosts.

Conclusions

We tested, for the first time, whether gut microbiome dynamics are synchronized among hosts experiencing strong synchronizing forces, including shared environments, similar diets, and high rates of between-host microbial dispersal. Despite these forces, baboons in Amboseli exhibit largely idiosyncratic gut microbiome dynamics: samples from the same baboon collected within a few days of each other were much more similar to each other than samples from different baboons, and host-specific factors, especially host identity, collectively explained 30% of the deviance in microbiome dynamics, compared to just 3% for factors shared across the host

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population. These idiosyncratic dynamics suggest that microbiome personalization is a widespread phenomenon that is likely not unique to humans, and instead may be shared with other social mammals. This microbiome personalization likely emerges from ecological and evolutionary phenomena that are a normal part of complex microbial communities, such as priority effects, functional redundancy, and horizontal gene flow. Together, these forces are expected to lead microbes with the same taxonomic identity in different hosts to perform somewhat different functions, experience different competitive landscapes and selective regimes, and play different ecological roles (*15, 16*). As a result, the same microbial taxa may often respond in different ways to environmental fluctuations, chance events, and/or interactions with other microbes in different hosts, producing personalized, rather than synchronized dynamics.

This personalization means that microbiome research aimed at improving human and animal health could face challenges to developing broadly applicable therapies, beyond those caused by heterogeneity in host diets, behaviors, and environments. Microbiome researchers aim to predict microbiome changes, link microbiome taxa and dynamics to health outcomes, and design microbiome interventions that work well for large segments of the human population. Personalization in humans is already presenting problems in attaining these goals. For instance, predictive models of gut microbiome dynamics from one person have been shown to fail when they are applied to other people (*19*). Our results suggest that microbiome predictions and interventions focused on microbiome taxa will require approaches that are either personalized or focus on microbial functions, as opposed to taxonomic identities. Even then, “universal” microbiome therapies that work the same way for all hosts may be unattainable. Instead, microbiome interventions will likely work best when they are designed for specific host groups or populations that have shared compositions and dynamics. Further, we expect that the types of prediction and intervention efforts that will suffer least from gut microbiome personalization are those that focus on microbiome functional traits (e.g., metabolites; functional pathways), rather than taxonomic composition. Together, our results provide novel insights about the extent and ecological causes of microbiome personalization, and point towards ways to overcome these barriers.

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Competing interests: The authors declare no competing interests.

Data and materials availability: 16S rRNA gene sequences are deposited on EBI-ENA (project ERP119849) and Qiita [study 12949, (*51*)]. Analyzed data and code is available on the first author's Open Science Framework / GitHub repository; for peer-review purposes, this is an anonymized link: https://osf.io/erdxa/?view_only=3323f05a5a9b479bac1124a5b07a62a9 .

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