

Supplementary Materials for “Social environment influences the relationship between genotype and gene expression in wild baboons”

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Supplementary Methods: SNP ascertainment and genotyping

We used the Genome Analysis Toolkit (GATK) for variant discovery and genotyping of the target flanking regions for the 96 individuals. We first used PicardTools (MarkDuplicates) to remove potential PCR duplicate reads from the overall read set for each individual. Then, we performed five rounds using the following GATK tools to identify variable sites in the set of 35 target regions: 1) RealignerTargetCreator, 2) IndelRealigner, 3) CountCovariates, 4) TableRecalibration, 5) AnalyzeCovariates, 6) UnifiedGenotyper, 7) VariantFiltration. For all but 3 samples, Steps 1-5 were run on each sample individually; for the three samples with the fewest total reads, we merged the samples prior to running this pipeline for computational efficiency. Steps 6-7 were run on all samples at the same time. We repeated these procedures five times because GATK relies on a set of known SNPs for accurately recalibrating quality scores at each sequenced site. Relatively few SNPs have been identified in baboons, particularly in our target regions, which would lead to assignment of recalibrated quality scores that were too low relative to the actual quality of the sequencing (all SNPs would be assumed to be sequencing errors in the recalibration pipeline). Thus, for the first round, we ran the pipeline without base quality recalibration (steps 3-5). For each subsequent round, we treated the set of high quality SNPs inferred in the previous round as “known” SNPs. To define high quality SNPs, we used the following GATK filters: $QD < 2.0$, $MQ < 40.0$, $FS > 60.0$, $HaplotypeScore > 13.0$, $MQRankSum < -12.5$, $ReadPosRankSum < -8.0$. After five repetitions, the analysis converged to a consensus number of variable sites (i.e., we no longer identified additional SNPs beyond those treated as known variants).

This analysis resulted in a high quality set of SNPs containing 3527 high confidence segregating sites (among 250,333 total base pairs for which at least 10 individuals were sequenced at a coverage of at least 30x). Among these sites, which were the sites in which SNPs were most likely to be called with high confidence, we therefore identified a total of one variant per ~70 base pairs (including both rare and common variants). This large number of sites is due to our sample size (95 individuals, after dropping one individual for whom we did not obtain many sequencing reads, or 190 chromosomes): extensive resequencing studies in humans show that the number of newly discovered SNPs increases approximately linearly, for both common and rare SNPs, within this range of samples (the number of newly discovered rare SNPs continues to increase linearly with sample size even into sample sizes >2000 individuals: Tennessen et al 2012, *Science* 337: 64 - 69). Two additional diagnostics also indicate that the SNP data set is high quality. First, SNPs in the data set exhibit a transition-transversion ratio (Ti/Tv) of 2.26, close to the genome-wide Ti/Tv ratio for highly filtered Illumina data on humans, with SNPs called at a false discovery rate of 1% (Ti/Tv = 2.15: DePristo et al 2011, *Nature Genetics* 43: 491 - 498). Poorer quality SNP data sets (i.e., pre-filtering) exhibit substantially lower Ti/Tv ratios. Second, the overall data set yields an estimate of π , a measure of population genetic diversity, within the range of π values for gene regulatory regions in this population based on Sanger sequencing data. Here, we estimate $\pi = 0.003$; from Sanger sequencing of 22 regulatory regions, $\pi = 0.002 \pm 0.002$ s.d. (Tung et al 2009, *Nature* 460: 388-391).

For missing genotypes in the data set, we used the Bayesian imputation program BEAGLE to infer missing data, which occurred at individual-site combinations with too low coverage to call genotypes with high confidence. Local linkage disequilibrium sometimes allows these sites to be imputed with high confidence, if other linked sites in the same individual have been successfully genotyped. To perform imputation, we used the genotype likelihoods produced by GATK as input into BEAGLE, and then filtered the post-imputation results to include only genotypes with posterior probability >0.98 . Target regions for each gene were analyzed separately, as genes are not expected to be in high LD with one another (and are therefore mutually uninformative with respect to imputing missing sites). Even after imputation, very few genotypes were called with high confidence for one individual (FACE) for whom we had the fewest number of reads.

Supplementary Methods: Potential confounds in the GEI analysis

GEIs are indicated by cases in which the magnitude of ASGE is correlated with environmental variation, which suggest that environmental effects influence the expression levels of the two alleles of a gene differently (i.e., the environment interacts with genetic variation). We hypothesized that, in these cases, individuals heterozygous for a functional *cis*-regulatory variant (indexed by the SNP that best partitioned overall variance in the ASGE data) would exhibit different levels of ASGE depending on environmental conditions, provided that the variant was also linked to the ASGE assay SNP. This hypothesis assumes that the social environmental variables of interest are not correlated with genetic background. If this is not the case, then apparent GEIs could actually represent cases of gene-gene interactions instead of gene-environment interactions. To exclude the likelihood that the GEIs we detected are in fact due to gene-gene interactions, we considered two major sources of genetic background in this population: *i*) admixture-related genetic background, as some baboons in Amboseli are hybrids between yellow baboons and anubis baboons (Tung et al 2008, *Molecular Ecology* 17: 1998 – 2011); and *ii*) kinship.

Admixture. Three lines of evidence suggest that our results are not confounded by admixture-related genetic background, assessed using the microsatellite marker-based approach in Tung et al (2008). First, there is no correlation between genetic estimates of admixture proportions and any of the environmental variables significantly involved in GEIs (p-values for these variables range from 0.06 – 0.85, and the maximum R^2 for all of these correlations, for female social connectedness, is small: $R^2 = 0.042$, $p = 0.15$). Second, including these estimates as a covariate in our GEI models does not qualitatively change the results. Finally, admixture estimates do not correlate with ASGE for any gene in our analysis ($p > 0.38$).

Kinship. Within social groups, females tend to be similar in rank to their mothers, potentially producing a close relationship between kinship and female dominance rank. However, we sampled individuals from seven different social groups, and females with similar ranks in different social groups are not expected to be closely related. Thus, although females with more similar ranks in the overall data set do tend to be slightly more closely related (correlation between pairwise relatedness derived from known pedigrees and pairwise rank similarity for females: $p = 0.0004$), rank similarity explains only 1% of variation in relatedness, and is therefore unlikely to confound our results. Similarly, relatedness explains less than 1% of variation in maternal dominance rank ($p = 0.0003$; $R^2 = 0.0064$) and female social connectedness ($p = 0.083$; $R^2 = 0.002$), and there is no relationship between pairwise relatedness and pairwise similarity in male rank ($p = 0.582$) or male social connectedness ($p = 0.573$).

Social groups themselves could also contribute to population structure in our sample, which could affect social environmental measures that involve group-level characteristics. In our analysis, this condition could apply to group size, if groups that are genetically more similar also tend to be of similar size. However, our sample does not exhibit substantial population structure explained by group identity (Figure S1). Additionally, there is no strong relationship between the major axes of population structure and group size (Figure S2): among the top 10 principal components of the SNP data, none are significantly correlated with group size after multiple hypothesis correction. PC1 is correlated with group size at a nominal $p = 0.02$, but group size explains only 6% of the variation in PC1, which itself explains only 7.3% of the variation in the overall data set (i.e., group size explains <1% of genetic variation in the sample). This lack of strong social group-mediated population structure in the sample likely arises from the high rate of migrant exchange between social groups due to (repeated) male dispersal.

Table S1. Number of individuals assayed and SNPs tested for each gene

Gene	# Males ¹	# Females ¹	Total SNPs ²	Best SNP ³	Model R ² ⁴
<i>AIM2</i>	7	9	5	AssaySNP	0.8831643
<i>APOBEC3G</i>	24	20	78	AssaySNP	0.853127
<i>CCR1</i>	16	17	7	AssaySNP	0.4296628
<i>CD5</i>	9	12	2	S_12611661	0.318564
<i>CD58</i>	7	3	0	AssaySNP	0.6864284
<i>CD8A</i>	22	22	18	S_85851418	0.5543711
<i>CD9</i>	9	13	11	S_6281260	0.8139922
<i>CLC</i>	9	9	5	AssaySNP	0.7199155
<i>CXCR4</i>	21	24	28	AssaySNP	0.3709335
<i>CYP17A1</i>	8	10	0	NONE	0.0792132
<i>DAP</i>	17	23	27	S_10531885	0.6178548
<i>GBP1</i>	18	21	15	AssaySNP	0.7546709
<i>GIMAP2</i>	23	19	6	AssaySNP	0.3707347
<i>GZMA</i>	12	25	13	S_51139696	0.5538139
<i>HLA-F</i>	24	21	8	S_29346355	0.3599964
<i>IFNAR1</i>	16	23	1	AssaySNP	0.8647966
<i>IL10</i>	14	12	3	AssaySNP	0.5763804
<i>IL4R</i>	19	23	12	AssaySNP	0.3019124
<i>IL6</i>	6	4	3	AssaySNP	0.7829775
<i>IRF2</i>	12	21	22	AssaySNP	0.3198682
<i>KLF6</i>	10	17	8	AssaySNP	0.6517723
<i>LGALS3</i>	17	17	17	S_111537912	0.3394394
<i>MNDA</i>	14	17	8	S_132726692	0.795599
<i>MSR1</i>	18	16	11	S_14106376	0.6950351
<i>MSRA</i>	13	19	19	S_8851908	0.5362489
<i>NMI</i>	13	21	5	AssaySNP	0.2747337
<i>OAS2</i>	18	21	10	S_112177655	0.5412198
<i>OASL</i>	19	15	3	AssaySNP	0.2213378
<i>PHF11</i>	12	14	4	AssaySNP	0.7464097
<i>PRF1</i>	7	11	3	S_59137422	0.5318094
<i>PRNP</i>	13	20	6	AssaySNP	0.5421846
<i>RNASE2</i>	18	15	7	S_77517141	0.786341
<i>S100A9</i>	7	13	9	AssaySNP	0.4413103
<i>SLA</i>	11	20	12	AssaySNP	0.5917604
<i>SLAMF7</i>	16	12	0	AssaySNP	0.4248022

¹In some cases, the sample sizes for each gene were reduced in GEI analyses due to missing environmental data.

²Number of SNPs in the resequenced region with complete genotypes for all individuals and at least 5 heterozygous and 5 homozygous genotypes. A model incorporating genotype at the pyrosequencing assay SNP (AssaySNP) was also tested for all genes.

³SNP that best partitioned ASGE data by heterozygous/homozygous genotype. AssaySNP is the SNP used in the ASGE assay and was required to be heterozygous. Other SNPs were in the resequenced regions and are named according to their chromosomal location in *Panu2.0*, except for *CYP17A1*, for which we identified no SNPs that partitioned the ASGE data well.

⁴Fraction of variance in ASGE levels explained by the model that partitioned the data on heterozygosity versus homozygosity at the best SNP. All models assume that homozygotes have mean ASGE = 0 and heterozygotes have mean ASGE ≠ 0. R² is calculated as the fraction change in variance relative to a model with no partition and global mean = 0.

Table S2. SureSelect sequencing targets, by gene

Gene	Chromosome ¹	Start ¹	End ¹	Length	Median depth ²	Median % coverage ³	Median coverage length ⁴
<i>AIM2</i>	chr1	132955593	132964498	8905	26428	0.71089275	6330.499939
<i>APOBEC3G</i>	chr10	79180057	79300153	120096	44114	0.2169223	26051.50054
<i>CCR1</i>	chr2	89160123	89169348	9225	18998.5	0.4441734	4097.499615
<i>CD5</i>	chr14	12610915	12612817	1902	3597.5	0.8267613	1572.499993
<i>CD58</i>	chr1	117335732	117370301	34569	74809.5	0.4198849	14515.00111
<i>CD8A</i>	chr13	85849748	85858937	9189	35535.5	0.94912395	8721.499977
<i>CD9</i>	chr11	6276833	6284714	7881	17203	0.69337645	5464.499802
<i>CLC</i>	chr19	33978585	34069409	90824	18985.5	0.0921893	8373.000983
<i>CXCR4</i>	chr13	106907441	106916614	9173	43677	0.9824485	9012.000091
<i>CYP17A1</i>	chr9	94642524	94644509	1985	7188.5	0.82493705	1637.500044
<i>DAP</i>	chr6	10527992	10537175	9183	29481	0.9560601	8779.499898
<i>GBP1</i>	chr1	89753327	89815458	62131	56381	0.25832515	16049.99989
<i>GIMAP2</i>	chr3	171952919	171962210	9291	16912.5	0.4336993	4029.500196
<i>GZMA</i>	chr6	51130538	51139709	9171	26794	0.77679645	7124.000243
<i>HLA-F</i>	chr4	29337813	29347221	9408	14933	0.5085034	4783.999987
<i>IFNARI</i>	chr3	13197137	13206645	9508	11005.5	0.42306475	4022.499643
<i>IL10</i>	chr1	157147958	157157149	9191	39402	0.9652921	8871.999691
<i>IL4R</i>	chr20	24996265	25005057	8792	10666	0.5444154	4786.500197
<i>IL6</i>	chr3	88209583	88218789	9206	52410.5	0.9994569	9201.000221
<i>IRF2</i>	chr5	173549606	173556760	7154	21244.5	0.87517475	6261.000162
<i>KLF6</i>	chr9	3931870	3941043	9173	21298.5	0.6321269	5798.500054
<i>LGALS3</i>	chr7	111534356	111542889	8533	33693	0.9697644	8274.999625
<i>MNDA</i>	chr1	132719928	132729183	9255	22194.5	0.53922205	4990.500073
<i>MSR1</i>	chr8	14099697	14109244	9547	18311.5	0.5683461	5426.000217
<i>MSRA</i>	chr8	8851858	8861084	9226	26998	0.7217646	6659.0002
<i>NMI</i>	chr12	13558857	13567522	8665	8067	0.3369302	2919.500183
<i>OAS2</i>	chr11	112176742	112185686	8944	14174.5	0.5380702	4812.499869
<i>OASL</i>	chr11	120304339	120313512	9173	13571	0.40711875	3734.500294
<i>PHF11</i>	chr17	27796800	27806083	9283	26405	0.84175375	7814.000061
<i>PRF1</i>	chr9	59130679	59139400	8721	13692	0.495356	4319.999676
<i>PRNP</i>	chr10	29256070	29264681	8611	24881.5	0.8659273	7456.49998
<i>RNASE2</i>	chr7	77516298	77570255	53957	135740	0.48084585	25944.99953
<i>SI00A9</i>	chr1	127176726	127185598	8872	27742.5	0.8321686	7382.999819
<i>SLA</i>	chr8	128026290	128035625	9335	32572.5	0.8619175	8045.999863
<i>SLAMF7</i>	chr1	134666586	134674540	7954	14467.5	0.56675885	4507.999893

¹Chromosome, start, and end coordinates are relative to the current baboon genome assembly: Panu2.0.

²Median depth refers to the median number of reads mapped within each genomic region across samples.

³Coverage percentage is the percentage of bases in each region covered by at least 1 read in a single sample. Median % coverage is the median of this value across samples.

⁴Coverage length is the number of bases in each target region covered by at least 1 read in a single sample. Median coverage length is the median of this value across samples.

Table S3. Summary of sequencing reads generated for each individual.

Individual	Total	Total mapped	MAPQ >= 20	Uniquely mapped	MAPQ >= 20 in target regions	Percent MAPQ >= 20	Percent MAPQ >= 20 in target region
ABBY	1495197	1284435	1263648	1284435	905249	0.845	0.716
ADRIANO	1798096	1477988	1445153	1477988	1071758	0.804	0.742
ALEX	2001812	1707333	1678424	1707333	1212607	0.838	0.722
AMOK	2329111	1892345	1833991	1892345	1181222	0.787	0.644
APOLLO	2121523	1559974	1464098	1559974	686488	0.690	0.469
ARAB	3409925	2598523	2452965	2598523	1175920	0.719	0.479
ARSENAL	3779403	3124554	3046763	3124554	2115252	0.806	0.694
BAGINE	2490863	1702171	1553332	1702171	484762	0.624	0.312
CABANA	937243	770628	749770	770628	534322	0.800	0.713
CADET	1529456	1281390	1252588	1281390	915867	0.819	0.731
DAGGER	1258604	835379	750164	835379	183762	0.596	0.245
DASTON	1583926	1333211	1305471	1333211	980207	0.824	0.751
DRONGO	1904545	1596975	1564779	1596975	1124503	0.822	0.719
DUBU	1939102	1640573	1603934	1640573	1104390	0.827	0.689
DUNLIN	1891468	1580926	1546046	1580926	1168150	0.817	0.756
EAGLE	1701029	1434898	1405878	1434898	1031456	0.826	0.734
EVA	1221561	1020398	998907	1020398	757724	0.818	0.759
FACE	4711	3929	3825	3929	2729	0.812	0.713
GABRIEL	2738398	2302724	2257486	2302724	1651088	0.824	0.731
GANJA	1812979	1535843	1506154	1535843	1139160	0.831	0.756
HAPPY	2208938	1880251	1843191	1880251	1297384	0.834	0.704
HIBISCUS	1883451	1580385	1547114	1580385	1235748	0.821	0.799
HOLLAND	1892393	1594988	1562216	1594988	1093883	0.826	0.700
HONEY	1750707	1384873	1322520	1384873	550086	0.755	0.416
HYMN	1742364	1424267	1367784	1424267	384530	0.785	0.281
JAGGER	2133118	1725098	1674126	1725098	1170823	0.785	0.699
KATHRYN	1734508	1456590	1429459	1456590	1027413	0.824	0.719
KELLY	1598776	1249200	1193960	1249200	696801	0.747	0.584
KERNEL	1695791	1429004	1399530	1429004	1025653	0.825	0.733
KOLA	1911984	1642751	1609226	1642751	1082691	0.842	0.673
LANCASTER	1793612	1512642	1483932	1512642	1129860	0.827	0.761
LAOS	1585401	1348401	1324626	1348401	1035298	0.836	0.782
LAX	1651447	1381654	1351408	1381654	1015551	0.818	0.751
LAZA	1544048	1296925	1263368	1296925	826936	0.818	0.655
LEBANON	1641208	1396178	1368273	1396178	1049222	0.834	0.767
LIBERTY	2310008	1925813	1886916	1925813	1461683	0.817	0.775
LIMAU	1347576	1168852	1150994	1168852	831181	0.854	0.722
LIWAZA	2113135	1819200	1790803	1819200	1359548	0.847	0.759
LIZZY	1729591	1479438	1454425	1479438	1028346	0.841	0.707
LOBO	1848783	1515929	1476402	1515929	1105407	0.799	0.749
LOCUST	1889743	1612426	1581762	1612426	1233640	0.837	0.780
LOFTY	1954771	1654565	1624983	1654565	1259010	0.831	0.775
LOGAN	1552940	1278863	1251634	1278863	903802	0.806	0.722
LOLLIPOP	1540787	1288010	1260253	1288010	913853	0.818	0.725
LOZENGE	2136115	1710896	1654864	1710896	1077508	0.775	0.651
LUIGI	2024469	1671928	1637009	1671928	1202670	0.809	0.735
LUNA	1325864	1110374	1090682	1110374	762428	0.823	0.699
LURCH	2336220	1862254	1799145	1862254	1197373	0.770	0.666
LUTHER	1857327	1521234	1475632	1521234	1020158	0.794	0.691
LYENA	2515672	2087527	2041782	2087527	1505985	0.812	0.738
LYME	3037665	2509487	2455186	2509487	1816432	0.808	0.740
MBEGU	2490597	2076865	2026238	2076865	1501781	0.814	0.741
MONGOOSE	1597737	1359409	1336468	1359409	1004937	0.836	0.752
MORRIS	130063	109486	107222	109486	80988	0.824	0.755
NARASHA	1760026	1452741	1418760	1452741	1012058	0.806	0.713
NAWA	1652478	1391371	1363432	1391371	927442	0.825	0.680
NIKE	2984474	2435007	2360916	2435007	1582831	0.791	0.670
NJUGU	1951141	1644186	1612064	1644186	1159629	0.826	0.719
NOBEL	1981065	1606228	1554598	1606228	997006	0.785	0.641
NOODLE	2514187	2103848	2059431	2103848	1494440	0.819	0.726
NOZZLE	2140135	1813680	1771657	1813680	1219592	0.828	0.688
NUTTY	2512710	2019505	1951954	2019505	1271117	0.777	0.651
NYUKI	1998866	1650531	1605480	1650531	1025602	0.803	0.639
OCTAGON	2070496	1746428	1705866	1746428	1194235	0.824	0.700
ODESSA	1707076	1418214	1382300	1418214	973885	0.810	0.705

OJUANG	2328275	1922423	1870298	1922423	1236535	0.803	0.661
OPHELIA	2176368	1792728	1748752	1792728	1222978	0.804	0.699
OXYGEN	2607572	2204115	2158514	2204115	1546290	0.828	0.716
PAISLEY	1524350	1260078	1229222	1260078	848766	0.806	0.690
PITTSBURGH	2749209	2196233	2108937	2196233	1196407	0.767	0.567
PLATO	2481620	2079009	2030144	2079009	1389591	0.818	0.684
QUASI	2826516	2298516	2228635	2298516	1386191	0.788	0.622
RAJA	1959767	1608845	1571956	1608845	1109879	0.802	0.706
RHODA	1566052	1304426	1273013	1304426	885832	0.813	0.696
RWANDA	1575757	1319767	1293848	1319767	998054	0.821	0.771
SCENIC	2455244	2038224	1986074	2038224	1399561	0.809	0.705
SORGHUM	1426866	1214169	1193727	1214169	895523	0.837	0.750
TALBOT	922880	768311	753614	768311	580537	0.817	0.770
VAIN	1828717	1498088	1461161	1498088	1048856	0.799	0.718
VAPOUR	2141347	1832473	1799016	1832473	1385872	0.840	0.770
VEIL	2309459	1954522	1915038	1954522	1346214	0.829	0.703
VET	1660238	1120578	1017076	1120578	391977	0.613	0.385
VIBRANT	778148	663835	651911	663835	511036	0.838	0.784
VOTE	1713897	1477187	1452205	1477187	1123113	0.847	0.773
VOYAGE	1184658	1003416	981830	1003416	746491	0.829	0.760
WABASH	1964590	1666075	1636388	1666075	1169307	0.833	0.715
WASP	1214645	1025543	1004707	1025543	774576	0.827	0.771
WENDY	3632203	2934107	2836423	2934107	1841581	0.781	0.649
WIPER	951874	815014	799850	815014	626898	0.840	0.784
WIRE	1929046	1622437	1591236	1622437	1197723	0.825	0.753
WIVU	1790159	1551534	1524955	1551534	1109327	0.852	0.727
WIZARD	1536865	1285554	1254572	1285554	878552	0.816	0.700
WRINKLE	2582734	2163015	2115896	2163015	1490519	0.819	0.704
WYNNE	1455187	1239105	1214818	1239105	936456	0.835	0.771
YAI	1522068	1303177	1278714	1303177	954547	0.840	0.746
YOGI	1742380	1503305	1477320	1503305	1097120	0.848	0.743
median	1853055	1543688.5	1495043	1543688.5	1080099.5	0.819	0.717
sd	602151.4965	487903.969	474198.3193	487903.969	349100.5869	0.044	0.103
min	4711	3929	3825	3929	2729	0.596	0.245
max	3779403	3124554	3046763	3124554	2115252	0.854	0.799

Table S4. GEI model results.¹

Sex	Environment	Df_N ²	DF_R ³	Likelihood ratio	p-value	BH q-value ⁴
Males	age at darting	30	329	28.54372633	0.541647258	0.541647258
Males	maternal rank	24	205	40.32814378	0.019714451	0.043809891
Males	group size at birth	24	205	24.51213285	0.432645591	0.480717323
Males	group size at darting	30	300	55.79755812	0.002880249	0.014401247
Males	male dominance rank	30	300	33.90377159	0.284696327	0.355870409
Males	male social connectedness	28	278	56.44851553	0.001133818	0.01133818
Males	maternal social connectedness	24	193	28.57051048	0.23671417	0.315618894
Females	age at darting	31	381	58.04568673	0.002277703	0.014401247
Females	maternal rank	31	355	31.04701475	0.46384505	0.488257947
Females	group size at birth	31	381	63.78119868	0.000472557	0.00945114
Females	group size at darting	31	381	52.53741093	0.009199003	0.030663344
Females	female dominance rank	31	381	39.80571732	0.13348055	0.1906865
Females	female social connectedness	31	381	50.7289888	0.014147561	0.035368904
Females	maternal social connectedness	30	336	39.14795398	0.122448536	0.188382363
Both	sex	34	787	57.8813826	0.00650066	0.02600264
Both	age at darting	34	753	45.04296071	0.097551753	0.177366823
Both	maternal rank	34	603	37.68510893	0.3043452	0.358053177
Both	group size at birth	34	629	52.14243613	0.024070279	0.048140558
Both	group size at darting	34	722	55.21693698	0.012152455	0.034721299
Both	maternal dominance rank	34	569	44.43609912	0.108542801	0.180904668

¹Each row of the table provides details for a model that tested the hypothesis that GEIs influenced gene expression for one or both sexes (specified in column 1), involving the environment specified in column 2.

²Model degrees of freedom

³Residual degrees of freedom

⁴Benjamini-Hochberg corrected p-values.

Table S5. Evidence for GEIs involving tested interaction effects, for genes (n = 13) in which ASGE was best explained by a putative regulatory SNP in the resequencing data¹

Interaction effect	p-value (females) ²	p-value (males)	p-value (both sexes)
<i>social environments</i>			
dominance rank			
early (maternal)	0.313 (12)	0.225 (5)	0.187 (13)
adult	0.080 (12)	0.047 (9)	NA ³
social connectedness			
early (maternal)	0.126 (11)	0.643 (5)	0.098(13)
adult	0.030 (12)	1.20 x 10 ⁻³ (7)	NA
group size			
early	3.63 x 10 ⁻⁴ (12)	0.377 (5)	0.027 (13)
adult	0.003 (12)	0.074 (9)	0.635 (13)
<i>non-social effects</i>			
age	0.049 (12)	0.331 (9)	0.181 (13)
sex	NA	NA	0.06 (13)

¹This table recapitulates Table 1 in the main manuscript, but focuses on the smaller subset of genes for which the SNP that best explained ASGE variation was a SNP in the resequenced putative regulatory region (i.e., not the assay SNP)

²P-values from likelihood ratio tests comparing a model with GEIs to a model without GEIs are provided in each cell. Values in parentheses provide the number of genes included in each test.

³Social connectedness and dominance ranks in adulthood could not be evaluated for both sexes combined, as SCI values were calculated separately for males and females.

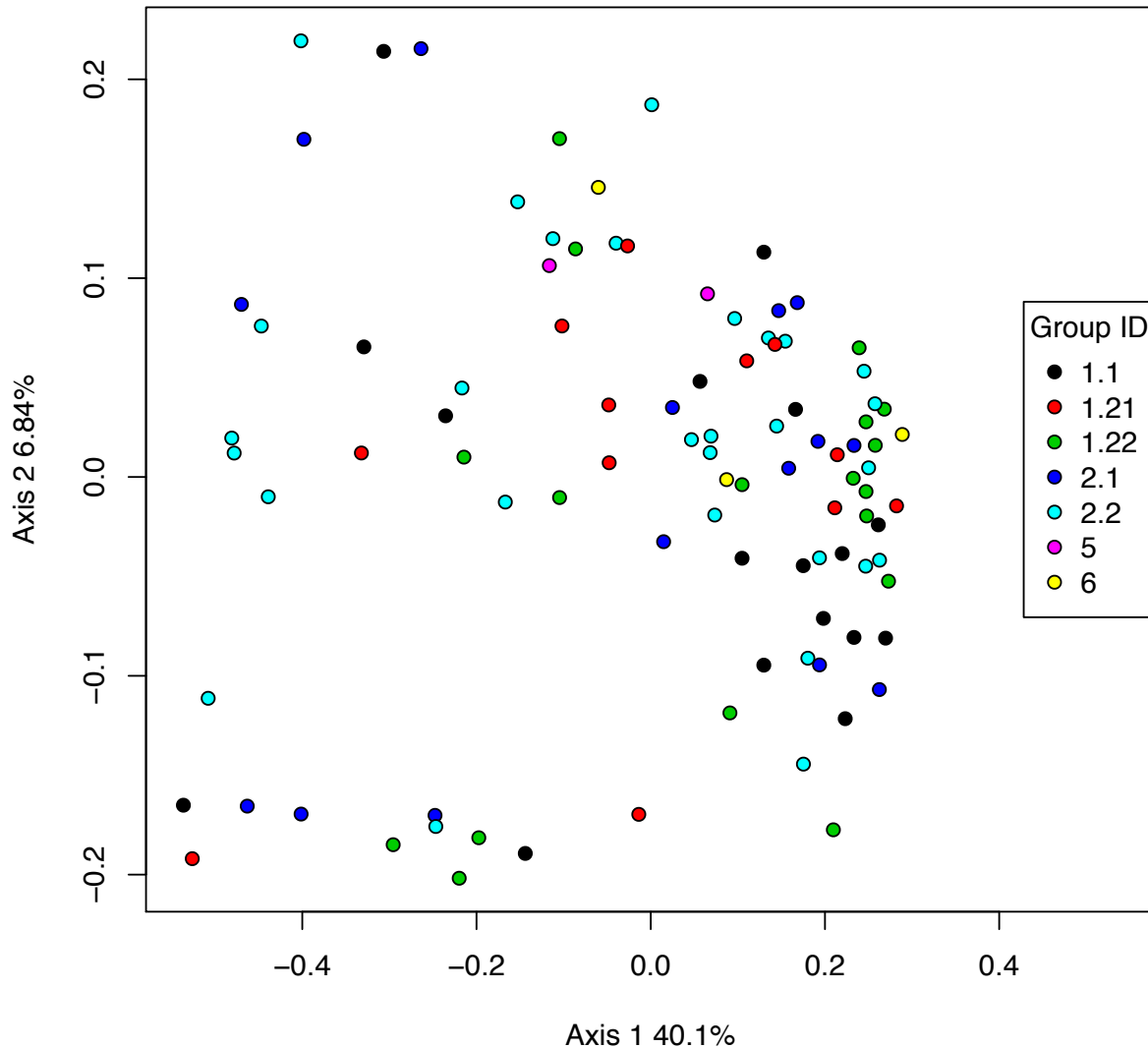


Figure S1. Population structure in the Amboseli study sample largely does not reflect social group. Classical (metric) multidimensional scaling was used to project a genetic distance matrix among the 96 individuals in the study sample onto two axes. The distance matrix was calculated as $d_{ij} = 1 - R^2_{ij}$ where R^2_{ij} is the squared correlation coefficient of genotypes (coded as 0,1,2) for individuals i and j . Only SNPs that passed all GATK quality filters were considered. The percent of variation in d (i.e., the percentage of overall genetic variation in the data set) accounted for by each axis is reported. Dots represent individual study subjects, and are coded according to the group that they belonged to at the time of darting. Note that subjects do not cluster by social group, reflecting the weak contribution of social group to overall population genetic structure in this population.

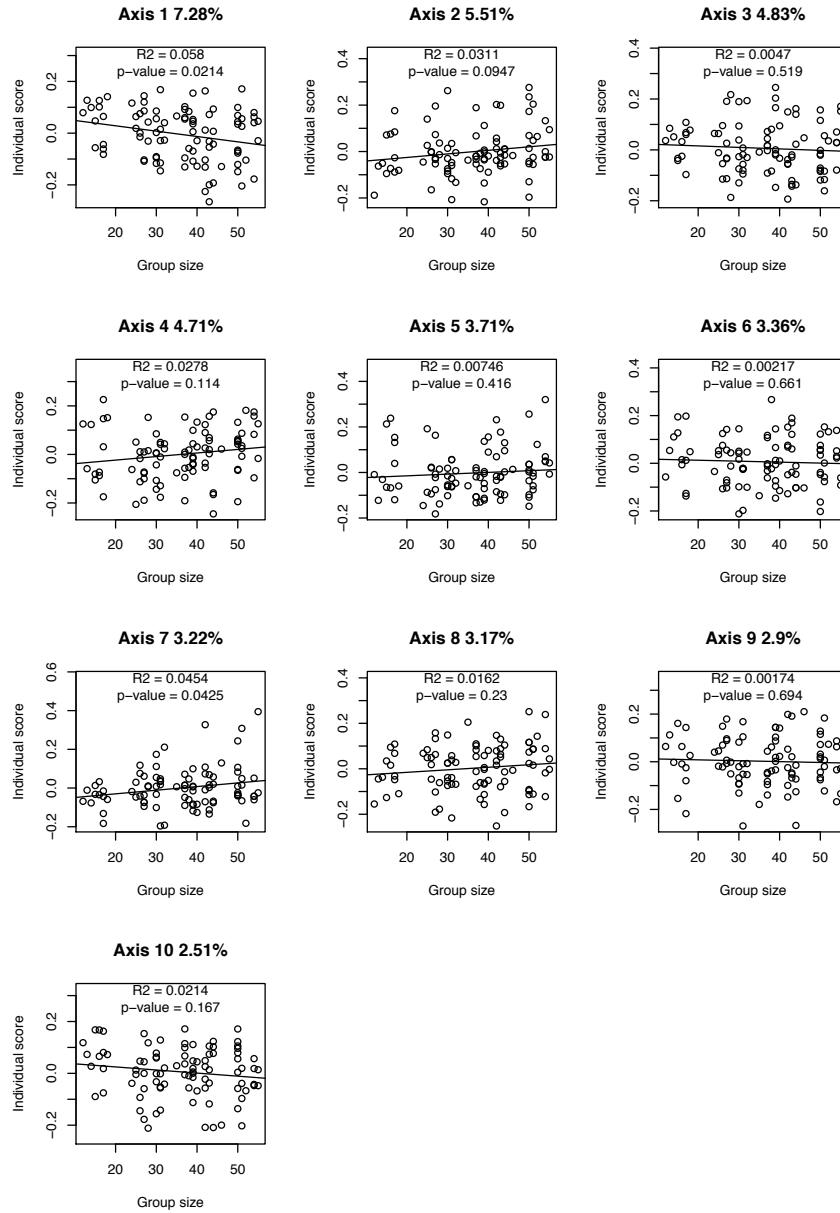


Figure S2. Group size does not explain substantial variation in the major axes of population structure. Principal components decomposition was performed on SNPs identified in the data set (using the R package *SNPRelate*) to identify the major axes of genetic structure in the sample. Each panel shows the correlation between one of these axes and group size at the time of darting of each individual. Panel titles provide the percentage of overall genetic variation accounted for by each axis, and inset text shows the R^2 and p-values for a linear regression of PCA axis score on group size. Note that even where these p-values are nominally significant, group size explains very little overall genetic structure in the Amboseli population.

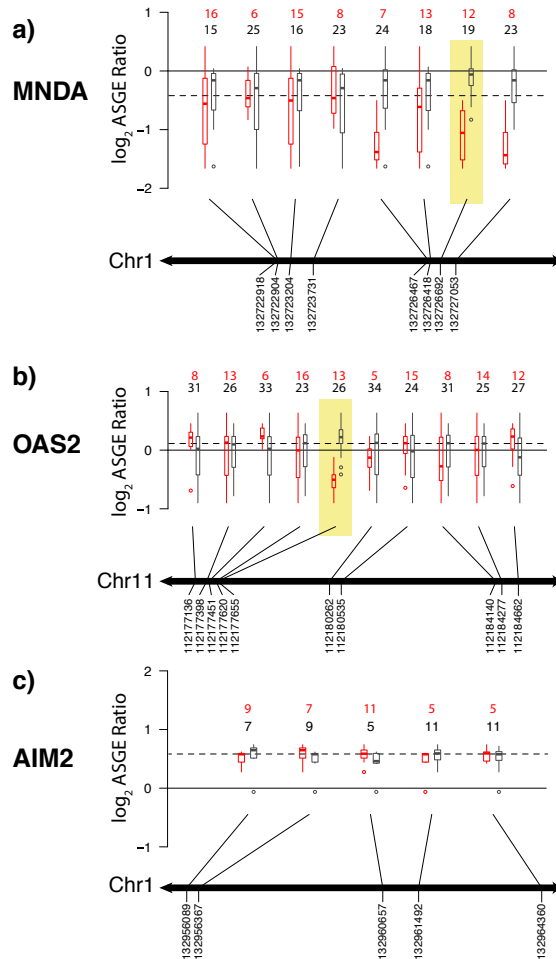


Figure S3. Identification of SNPs for downstream GEI analysis. Common ASGE implies the presence of functional *cis*-regulatory variation. Individuals who exhibit high (low) levels of ASGE are expected to be heterozygous (homozygous) for the functional *cis*-regulatory site or sites. Thus, genotype at the functional site, or sites in high linkage disequilibrium (LD) with the functional site, should explain substantial variance in ASGE levels. For each gene that exhibited common ASGE in the Amboseli samples, we identified the SNP for which genotype explained the most variance in ASGE levels, constraining homozygotes to have ASGE levels near zero, and heterozygotes to have non-zero ASGE levels. In each case, we considered SNPs in the putative upstream *cis*-regulatory region as well as the SNP used to assay ASGE itself. We calculated the variance in ASGE (R^2) explained by each SNP, and identified the model that explained the most variance in the ASGE data as focusing on the most suitable SNP for downstream GEI analysis. a) and b) show cases in which the best SNP was located in the putative *cis*-regulatory region resequenced for the two respective genes. c) shows a case in which all individuals in the sample exhibit consistent non-zero ASGE and variance in ASGE was best explained by the assay SNP (not shown); if the assay SNP is in high LD with a functional site, all individuals in the sample are expected to exhibit non-zero ASGE. In each panel, SNPs are shown in their relative positions on the chromosome (not to scale), with gene ASGE ratios partitioned by heterozygous (red) versus homozygous (black) genotype. Number of heterozygotes and homozygotes for each SNP are provided about the boxplots. Horizontal dashed lines in each plot show the median ASGE ratio of all samples.