References

Aktuell

KARLSSON 2016

Sten Karlsson, Ola H. Diserud, Peder Fiske & Kjetil Hindar, Widespread genetic introgression of escaped farmed Atlantic salmon in wild salmon populations. ICES Journal of Marine Science (2016), preprint, 1–11. DOI:10.1093/icesjms/fsw121.

Farmed Atlantic salmon (Salmo salar) escape from net pens and enter rivers to spawn, potentially resulting in genetic introgression and reduced fitness of wild salmon. Here, we quantify genetic introgression of farmed to wild salmon, using molecular genetic markers, in populations from 147 salmon rivers, representing three-quarters of the total wild salmon spawning population in Norway. For 109 rivers with adult modern samples and sample sizes of 20 or more, the average level of farmed genetic introgression was 6.4% (median=2.3%), with a range between 0.0% and 42.2%. Fifty-one of these rivers showed significant farmed genetic introgression when compared with historical reference samples. We observed a highly significant correlation between estimated farmed introgression and average proportion of escaped farmed salmon. We quantify levels of introgression as unweighted averages or weighted by population sizes, to compare geographical regions and to compare levels of introgression in rivers and fjords designated as locations deserving a high level of protection. We found a generally lower level of introgression in National Salmon Rivers and National Salmon Fjords subjected to formal protection by parliament. We conclude that farmed to wild genetic introgression is high in a large proportion of Norwegian salmon rivers, with the highest levels found in the most intensive areas of salmon farming. The extensive genetic introgression documented here poses a serious challenge to the management of farmed and wild Atlantic salmon in Norway and, in all likelihood, in other regions where farmedsalmon escape events occur with regularity

Keywords: atlantic salmon, aquaculture, farmed salmon, genetic introgression, genetics, SNPs.

KIM 2016

Myunghoo Kim, Yaqing Qie, Jeongho Park & Chang H. Kim, Gut Microbial Metabolites Fuel Host Antibody Responses. Cell Host & Microbe (2016), preprint, 1–13. DOI:10.1016/j.chom.2016.07.001.

Antibody production is a metabolically demanding process that is regulated by gut microbiota, but the microbial products supporting B cell responses remain incompletely identified. We report that shortchain fatty acids (SCFAs), produced by gut microbiota as fermentation products of dietary fiber, support host antibody responses. In B cells, SCFAs increase acetyl-CoA and regulate metabolic sensors to increase oxidative phosphorylation, glycolysis, and fatty acid synthesis, which produce energy and building blocks supporting antibody production. In parallel, SCFAs control gene expression to express molecules necessary for plasma B cell differentiation. Mice with low SCFA production due to reduced dietary fiber consumption or microbial insufficiency are defective in homeostatic and pathogen-specific antibody responses, resulting in greater pathogen susceptibility. However, SCFA or dietary fiber intake restores this immune deficiency. This B cell-helping

function of SCFAs is detected from the intestines to systemic tissues and conserved among mouse and human B cells, highlighting its importance.

Highlights

- Short-chain fatty acids(SCFAs) produced by gut microbiota promote antibody responses
- SCFAs activate B cell metabolism for production of energy and building blocks
- SCFAs control gene expression for plasma B cell differentiation
- SCFAs boost antibody responses during infection, decreasing susceptibility to pathogens

In Brief: Kim et al. demonstrate that short-chain fatty acids (SCFAs), produced by the gut microbiota as fermentation products of dietary fiber, support host antibody responses by regulating gene expression and enhancing cellular metabolism and plasma B cell differentiation. SCFAs boost mucosal and systemic antibody responses during steady state and infection.

PITMAN 2016

Robert L. Pitman et al., Humpback whales interfering when mammal-eating killer whales attack other species, Mobbing behavior and interspecific altruism? Marine Mammal Science (2016), preprint, 1–52. DOI:10.1111/mms.12343.

Robert L. Pitman, Volker B. Deecke, Christine M. Gabriele, Mridula Srinivasan, Nancy Black, Judith Denkinger, John W. Durban, Elizabeth A. Mathews, Dena R. Matkin, Janet L. Neilson, Alisa Schulman-Janiger, Debra Shearwater, Peggy Stap, Richard Ternullo Humpback whales (Megaptera novaeangliae) are known to interfere with attacking killer whales (Orcinus orca). To investigate why, we reviewed accounts of 115 interactions between them. Humpbacks initiated the majority of interactions (57 % vs. 43 %; n = 72), although the killer whales were almost exclusively mammal-eating forms (MEKWs, 95%) vs. fish-eaters (5%; n = 108). When MEKWs approached humpbacks (n = 27), they attacked 85% of the time and targeted only calves. When humpbacks approached killer whales (n =41), 93% were MEKWs, and >87% of them were attacking or feeding on prey at the time. When humpbacks interacted with attacking MEKWs, 11% of the prey were humpbacks and 89 % comprised 10 other species, including three cetaceans, six pinnipeds, and one teleost fish. Approaching humpbacks often harassed attacking MEKWs (\geq 55% of 56 interactions), regardless of the prey species, which we argue was mobbing behavior. Humpback mobbing sometimes allowed MEKW prey, including nonhumpbacks, to escape. We suggest that humpbacks initially responded to vocalizations of attacking MEKWs without knowing the prey species targeted. Although reciprocity or kin selection might explain communal defense of conspecific calves, there was no apparent benefit to humpbacks continuing to interfere when other species were being attacked. Interspecific altruism, even if unintentional, could not be ruled out.

 $\label{lem:keywords: humpback whale | interspecific altruism | killer whale | Megaptera novaeangliae | mobbing behavior | Orcinus orca | predation.$

Anthropologie

HAYWARD 2016

Adam D. Hayward, Francesca L. Rigby & Virpi Lummaa, Early-life disease exposure and associations with adult survival, cause of death, and reproductive success in preindustrial humans. PNAS 113 (2016), 8951–8956.

A leading hypothesis proposes that increased human life span since 1850 has resulted from decreased exposure to childhood infections, which has reduced chronic inflammation and later-life mortality rates, particularly from cardiovascular disease, stroke, and cancer. Early-life cohort mortality rate often predicts later-life survival in humans, but such associations could arise from factors other than disease exposure. Additionally, the impact of early-life disease exposure on reproduction remains unknown, and thus previous work ignores a major component of fitness through which selection acts upon life-history strategy. We collected data from seven 18th- and 19th-century Finnish populations experiencing naturally varying mortality and fertility levels. We quantified early-life disease exposure as the detrended child mortality rate from infectious diseases during an individual's first 5 y, controlling for important social factors. We found no support for an association between early-life disease exposure and all-cause mortality risk after age 15 or 50. We also found no link between early-life disease exposure and probability of death specifically from cardiovascular disease, stroke, or cancer. Independent of survival, there was no evidence to support associations between early-life disease exposure and any of several aspects of reproductive performance, including lifetime reproductive success and age at first birth, in either males or females. Our results do not support the prevailing assertion that exposure to infectious diseases in early life has long-lasting associations with later-life all-cause mortality risk or mortality putatively linked to chronic inflammation. Variation in adulthood conditions could therefore be the most likely source of recent increases in adult life span.

Keywords: inflammation | stress | infection | life history | fitness

Significance: Why has human life expectancy increased since 1850? A leading hypothesis proposes that limited exposure to childhood infections has reduced lifelong inflammation and enhanced survival, but tests of this hypothesis typically use all-cause mortality rates to estimate disease exposure. Meanwhile, links between early-life disease and reproduction have been neglected. We used data from preindustrial Finnish populations to show that early-life disease exposure was not associated with all-cause mortality, mortality from cardiovascular disease, stroke, and cancer, or reproductive success. Our study therefore does not support the prevailing contention that reduced exposure to early-life infections has increased life expectancy in modern populations.

SALA 2016

Nohemi Sala, Ana Pantoja-Pérez, Juan Luis Arsuaga, Adrián Pablos & Ignacio Martínez, The Sima de los Huesos Crania, Analysis of the cranial breakage patterns. Journal of Archaeological Science **72** (2016), 25–43.

JAS072-0025-Supplement.docx

The Sima de los Huesos (SH) site has provided the largest collection of hominin crania in the fossil record, offering an unprecedented opportunity to perform a complete Forensic-Taphonomic study on a population from the Middle Pleistocene. The fractures found in seventeen crania from SH display a postmortem fracturation pattern, which occurred in the dry bone stage and is compatible with collective burial assemblages. Nevertheless, in addition to the postmortem fractures, eight crania also display some typical perimortem traumas. By using CT images we analyzed these fractures in detail. Interpersonal violence as a cause for the perimortem fractures can be confirmed for one of the skulls, Cranium 17 and also probable for Cranium 5 and Cranium 11. For the rest of the crania, although other causes cannot be absolutely ruled out, the violence-related traumas are the most plausible scenario for the perimortem fractures. If this hypothesis is con-

firmed, we could interpret that interpersonal violence was a recurrent behavior in this population from the Middle Pleistocene.

Keywords: Taphonomy | Bone breakage | Atapuerca | Interpersonal violence

Biologie

CHISHOLM 2016

Rebecca H. Chisholm, James M. Trauer, Darren Curnoe & Mark M. Tanaka, Controlled fire use in early humans might have triggered the evolutionary emergence of tuberculosis. PNAS 113 (2016), 9051–9056.

Tuberculosis (TB) is caused by the Mycobacterium tuberculosis complex (MTBC), a wildly successful group of organisms and the leading cause of death resulting from a single bacterial pathogen worldwide. It is generally accepted that MTBC established itself in human populations in Africa and that animal-infecting strains diverged from human strains. However, the precise causal factors of TB emergence remain unknown. Here, we propose that the advent of controlled fire use in early humans created the ideal conditions for the emergence of TB as a transmissible disease. This hypothesis is supported by mathematical modeling together with a synthesis of evidence from epidemiology, evolutionary genetics, and paleoanthropology.

Keywords: tuberculosis | pathogen evolution | cultural evolution | epidemiology | mathematical modeling

Significance: Tuberculosis is an ancient human disease that continues to affect millions of people worldwide. A crucial component of the origins of the tuberculosis bacterium remains a mystery: What were the conditions that precipitated its emergence as an obligate transmissible human pathogen? Here, we identify a connection between the emergence of tuberculosis and another major event in human prehistory, namely the discovery of controlled fire use. Our results have serious and cautionary implications for the emergence of new infectious diseases—feedback between cultural innovation and alteration of living conditions can catalyze unexpected changes with potentially devastating consequences lasting thousands of years.

Energie

DE OLIVEIRA E SILVA 2016

Guilherme de Oliveira e Silva & Patrick Hendrick, Lead-acid batteries coupled with photovoltaics for increased electricity self-sufficiency in households. Applied Energy 178 (2016), 856–867.

With distributed generation of electricity growing in importance (especially with photovoltaics) and buildings being one of the main consumers of energy in modern societies, distributed storage of energy in buildings is expected to become increasingly present. This paper analyses the use of residential lead—acid energy storage coupled with photovoltaics and its possible interaction with the grid for different limits of feed-in power without any support policies. In the literature, these subjects are often treated independently and for very specific, non-optimised cases, thus motivating further research. Results show that reaching self-sufficiency values up to $40\,\%$ is possible, close to grid parity values, and only with photovoltaics. Beyond $40\,\%$, energy storage must be used, strongly raising the cost of the electricity consumed and therefore the need for support policies for widespread adoption. Also, peak power consumption from the grid remains constant and load variability

rises, suggesting that an increase in self-sufficiency would be accompanied by lower utilisation factors of power plants and, consequently, higher wholesale electricity prices during no sunshine hours. Limiting feed-in power attenuates the increased load variability and only slightly affects the economic viability of such installations. These results present a novel optimisation tool for developers and should be considered in future studies of distributed photovoltaics and energy storage as well as in energy policy.

Highlights:

- Grid parity is reached for PV installations up to nearly 40 % self-sufficiency.
- Reaching beyond 40% self-sufficiency requires storage and support policies.
- Peak consumption remains constant but load variability rises with self-sufficiency.
- Changes in power plants portfolio and wholesale electricity prices are expected.
- Limiting feed-in power is a promising solution for reducing load variability. Keywords: Home energy storage | Distributed energy storage | Lead-acid battery energy storage | Self-sufficiency | Photovoltaics (PV) | Levelised Cost of Energy (LCOE)

Mathematik

Constable 2016

George W. A. Constable, Tim Rogers, Alan J. McKane & Corina E. Tarnita, Demographic noise can reverse the direction of deterministic selection. PNAS 113 (2016), E4745–E4754.

pnas113-E4745-Supplement1.mp4, pnas113-E4745-Supplement2.mp4

Deterministic evolutionary theory robustly predicts that populations displaying altruistic behaviors will be driven to extinction by mutant cheats that absorb common benefits but do not themselves contribute. Here we show that when demographic stochasticity is accounted for, selection can in fact act in the reverse direction to that predicted deterministically, instead favoring cooperative behaviors that appreciably increase the carrying capacity of the population. Populations that exist in larger numbers experience a selective advantage by being more stochastically robust to invasions than smaller populations, and this advantage can persist even in the presence of reproductive costs. We investigate this general effect in the specific context of public goods production and find conditions for stochastic selection reversal leading to the success of public good producers. This insight, developed here analytically, is missed by the deterministic analysis as well as by standard game theoretic models that enforce a fixed population size. The effect is found to be amplified by space; in this scenario we find that selection reversal occurs within biologically reasonable parameter regimes for microbial populations. Beyond the public good problem, we formulate a general mathematical framework for models that may exhibit stochastic selection reversal. In this context, we describe a stochastic analog to r-K theory, by which small populations can evolve to higher densities in the absence of disturbance.

 $\begin{tabular}{ll} Keywords: stochastic dynamics | nonfixed population size | cooperation | public goods | timescale separation \\ \end{tabular}$

Significance: Demographic stochasticity—the population-level randomness that emerges when the timing of birth, death, and interaction events is unpredictable—can profoundly alter the dynamics of a system. We find that phenotypes that pay a cost to their birth rate tomodify the environment by increasing the global carrying capacity can be stochastically selected for, where they would otherwise be deterministically disfavored. Our results hold for a general class of mathematical models but we use a model of public good production for illustration. In this

case, demographic stochasticity is exploited by populations of cooperators to turn selection in their favor; it therefore operates as a mechanism that supports the evolution of public good production.