an inch deep. And I would say only in the last few years — weathering some stiff criticism across different fields — that we are gaining traction covering this much space. So, the question is, how do we value that a novice, trying to get into an uncomfortable space, might bring in viewpoints from different fields is a strength and not a weakness? While that sounds great in a magazine, in practice we still have a disconnect here that we need to work on.

With your field of study, we cannot help but talk about the COVID-19 pandemic. How do you think your field has been impacted? There has been a massive impact — a sea change in not just the scientific perception but the perception of the general public. Two things: viruses matter, and we can see evolution unfold in real time. The Delta variant in particular — seeing it sweep across the world and outcompete other variants of SARS-CoV-2 — is a natural lesson in the impact of viruses on our lives and evolution in real time.

During the pandemic, we've seen a huge gulf, or canyon, open in the sense that science has delivered incredibly well, but the public response was abysmal, with the virus even being weaponized for various political purposes. How do you see the role of a scientist in this complicated, fractured landscape? I think the pandemic has really illustrated the mismatched tempos of how science and politics can unfold. So, on the one hand, we can sample and measure virus diversity and how it's changing almost by the day — millions of sequences, available publicly worldwide — that's the very quick tempo. Also, the vaccines advanced quickly because of decades-long-running investment in basic science. On the other hand, scientific understanding, why is one variant outcompeting others, as well as public understanding — is the vaccine safe? — and political action — should the vaccine be mandated? — is a slower process, where we require a deeper understanding to advance knowledge and set good policy. And that mismatch has caused so much tension. Science can be confusing. This is that uncomfortable space we've been describing. And for someone who doesn't practise science, that discomfort might draw you away from what you need to do to contribute to help us move past the pandemic. It's a complex moment that demands a new level of communication.

I know that you are also a long-time podcaster, before it became trendy. Correct. The podcast I co-host is called 'This week in evolution' (TWiEVO), co-hosted with Vincent Racaniello of Columbia University, and we're now getting started on our sixth year. The last two years have largely been dedicated to the evolution of SARS-CoV-2 and in trying to deal with these mismatches in tempo of scientific and public information. I think this is another area where we scientists really need to perk up and hone our skills as communicators, not just with our colleagues, but with our communities, like the students who might be majoring in the humanities but are really curious and need to understand complex situations involving biology, like pandemics. As we get more specialized, how do we step back and share and facilitate a bigger conversation?

What's next? That's a perfect question as we've run out of beers, so that should be the next endeavor. During the pandemic, among all the challenges that have shaken up all of our lives, I've also had this almost dream-like, incredible last year. Joining the Howard Hughes Medical Institute, being named a MacArthur Fellow and having these resources to hopefully do something new and interesting in the area of science communication. I have had all of this incredibly generous recognition and am entrusted with resources but when you ask an evolutionary biologist about the future, they'll always say: "I'll figure out how to move forward by looking backwards". I hope that, despite these accolades, I'll still find myself feeling like a novice, still uncomfortable and still approach my science and work as a communicator as an amateur.

If you had an extra two hours a day, what would you do with them? Sleep.

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Quick guide

Dietary wariness

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What is dietary wariness? The term 'dietary wariness' describes animals' reluctance to sample novel foods. It encompasses two related, yet distinct processes: neophobia, which is a short-term refusal to touch unfamiliar foods, and dietary conservatism, a longer-term hesitancy to fully incorporate a new food into the diet, even after some level of familiarity has been gained. These are considered to be discrete processes because dietary conservatism is a complex multi-stage process that is resistant to deactivation, and is often present in only a subset of individuals in a population. This is not thought to be true of food neophobia. However, neophobia may be more complex than is currently appreciated (Figure 1), and there is little direct evidence that it is easier to overcome than dietary conservatism (see below). Food neophobia is near universal and has been documented in insects, fish, birds and mammals, but is absent in a small number of species. It has a genetic component in humans and birds, but the genes that influence it are poorly understood. Dietary conservatism is a more recently described phenomenon that has been observed in several species of birds and fish, but this list is likely to expand as research continues.

Why would animals reject perfectly good food? Animals face a dilemma when encountering novel food: in the absence of knowledge about the food's palatability or toxicity, should they invest time exploring the novel food and take the risk that it could be toxic, or disregard it in favour of familiar foods of known value? Models of this exploration–exploitation trade-off suggest that wariness of novel food is the optimal strategy under a range of ecological conditions. They predict that animals should be wary when novel food is rare and the cost of sampling toxic food is high. The benefits of sampling novel food outweigh the costs only when novel food is found at high densities,

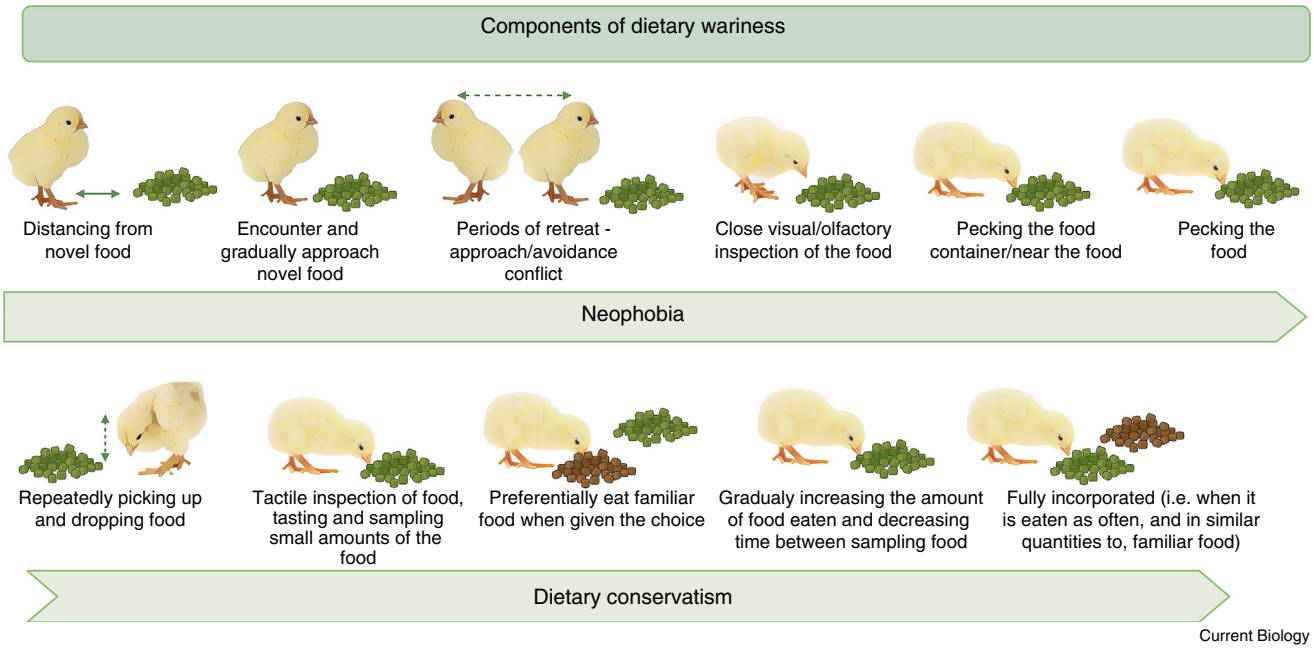

Figure 1. Components of dietary wariness in domestic chicken chicks.
Top: the stages involved in overcoming neophobia. When faced with novel food, chicks initially distance themselves from it (left). As neophobia wanes, chicks move progressively closer to the food until they eventually touch it (right). At this point neophobia has been overcome. Bottom: the stages involved in overcoming dietary conservatism. Chicks begin by handling food carefully (left), slowly incorporate it into their diet, and eventually eat it at similar levels to familiar food (right). At this point dietary conservatism has been overcome. (Figure created using BioRender.com.)

and under these circumstances animals should expand their diets to include novel food. These predictions are supported by findings that dietary wariness can be overcome by repeated exposure to novel food, which serves to enhance the food's perceived density. However, there is no empirical evidence that dietary wariness enhances an individual's fitness, and mathematical models are yet to directly explore why levels of wariness vary so much among individuals.

What factors affect the expression of dietary wariness? Food density is not the only factor that can influence the expression of dietary wariness. Any factor that decreases the cost or increases the benefit of sampling novel food should reduce dietary wariness, whilst factors that do the reverse should enhance it. For example, wariness is reduced when competition for food is high or when familiar individuals are observed eating the novel food. It is enhanced when a novel food is perceived to be high risk: for example, when the food has a conspicuous colour or pattern, or causes conspecifics to perform distaste responses. An individual's physiological and energetic state — hunger levels, fat stores, toxin burden, current diet quality, and previous experience with food — are also likely to alter the costs and benefits of sampling novel food, and could explain why the expression of dietary wariness varies considerably among individuals. It has been suggested that neophobia is deactivated more easily than dietary conservatism, and that factors that affect one of these processes may not necessarily affect the other. There is some support for this latter hypothesis: in blue tits, the presence of a conspecific reduces dietary conservatism, but not neophobia. However, this is not the case in domestic chicks, and the results of other experiments testing these ideas are difficult to interpret as they measure wariness rather than dietary conservatism.

Why does any of this matter? Determining how animals respond to novel food allows us to understand their diet breadths, and why they may choose imbalanced diets associated with adverse health and development outcomes. It helps us to predict how animals might adapt to both natural and anthropogenic environmental changes in food availability, and has important implications for signal evolution. Vertebrate predators that express dietary wariness avoid novel prey, which can allow mutations that cause warning signals to spread through populations of defended prey species. Understanding what factors influence the expression of dietary wariness will also allow us to manipulate its expression for welfare, commercial, and conservation purposes. In agriculture, decreasing dietary wariness in hens could eliminate the reduced growth rates and outbreaks of injurious feather pecking associated with the diet changes common in commercial farming. It could also allow translocated animals of conservation concern to better integrate into novel environments that do not contain foods that pose a significant health risk. When releasing animals into riskier environments, increasing dietary conservatism, if teamed with training on 'safe' food, could be used to reinstate natural levels of wariness that may have been lost in captivity. As we increasingly need to find sustainable food sources, the ability to overcome western societal wariness to alternative protein sources like insects

will be crucial to ensuring future food security.

Where do we go from here? Many questions remain about dietary wariness. Are neophobia and dietary conservatism distinct processes, and how should they be measured? How widespread are these traits and how are they affected by genetics, ontogeny, and experience? What are the fitness benefits to being adventurous versus wary, and how do these differ among individuals and species? Is dietary wariness part of a general anxiety trait, and can it be manipulated without changing other behaviours? Are there parallels between picky/fussy eating in humans and dietary conservatism in non-human animals, and to what degree are the techniques that have been developed to manipulate the expression of these traits transferable? Dietary wariness promises to be a fruitful area of research that will have important practical applications.

Where can I find out more?

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Primer

Cell-size control

Nicholas Rhind

A fundamental and still mysterious question in cell biology is "How do cells know how big they are?". The fact that they do is evident from the strict maintenance of size homeostasis within populations of cells and has been verified by a variety of creative experiments over the past 100 years. An increasingly sophisticated understanding of cell-cycle-control mechanisms and innovations in cell imaging and analysis tools have allowed recent progress in proposing and testing models of cell-size control. Nonetheless, a biochemical understanding of how proposed cell-size mechanisms might work is only beginning to be developed. This primer introduces the field of cell-size control and discusses some of the questions that are yet to be answered.

The question of cell-size control

Size is a biological variable that affects life on every scale, from organelles to ecosystems. One key scale is the size of cells. Cell size is remarkable both for its interspecies variability — with volumes ranging over 14 orders of magnitude, from 0.1 femtoliter ultramicrobacteria to 10 milliliter amoebae — and its intraspecies homogeneity, with populations of cells regularly exhibiting as little as 10% variance in size at division. Moreover, many factors can cause variation in cell size — from external nutritional conditions to internal cell-cycle delays — requiring some sort of control system to counteract these drivers of cell-size heterogeneity. Therefore, how cells maintain size homeostasis over such a wide range of potential sizes has long fascinated biologists.

In framing the question of cell size, a few issues need to be addressed. The first is the definition of size itself. Generally, total cell mass is used as the primary definition of cell size and we will use that definition here. However, cells can vary independently in volume, length, surface area and protein content. Correlations between these parameters and other relevant parameters, such as density, growth rate and genome content, complicate the definition of cell size. Furthermore, it is unclear which of these parameters, if any, are actually measured by cells to facilitate cell-size regulation.

A second issue is the cellular life-history context in which cell size is considered. Size homeostasis in a vertebrate neuron that will not divide for 100 years is a much different problem than size homeostasis in a yeast cell that will divide every 100 minutes. We will leave to others the question of size homeostasis in terminally differentiated cells and other quiescent cells, and focus here on populations of growing and dividing cells. In such populations, cell size is controlled by a balance between growth and division, and the size of a newborn cell is determined by the size at which its parental cell divided.

Finally, it is important to consider the difference between the questions of how cells determine what size they should be and how they maintain homeostasis at that target size. The former question — how cells decide how big they should be — is influenced by nutritional status in single-celled organisms and by cellular differentiation in multicellular organisms and, in eukaryotes, involves the TOR signaling pathway. However, disruption of the mechanisms that determine the target size do not disrupt the ability of cells to maintain size homeostasis. Cells that have their target size perturbed may divide at the 'wrong' size, but they maintain that wrong size with a robustness comparable to normal cells. The latter question — how cells maintain size homeostasis — is the focus of this primer.

An important distinction between the cell-size control systems that determine a cell's target size and the cell-size control systems that maintain that target size is that target-size determination is not generally essential for cell viability, but target-size homeostasis generally is. Many mutations have been identified that cause cells to be bigger or smaller than their wild-type target size, without obvious fitness effects. The systems for maintaining size homeostasis, on the other hand, are expected to be essential. A cell that has no size homeostasis mechanism will either never divide or divide repeatedly without sufficient growth, both of which