with explanations that are a vast tangle of multidimensional jargon and you never really understand what is going on in your work. At least that is my experience. Besides, teaching can be great fun in vision science where any good class should be punctuated by screams of surprise and delight for the in-class demonstrations.

What do you think is the big question to be answered in your field? Our big question is consciousness. This is on a par with the nature of matter, space, and time, and the origin of the universe. It was once a dark, unfundable subject but thanks to the efforts of a few like Christof Koch, Francis Crick, Bernie Baars, Dan Dennett, Stan Dehaene and others, it has taken its deserved central place in science. Studying consciousness is one thing but, in my opinion, understanding its mechanisms will require a whole new physics. There is no known physical property that can produce the unity of experience from the interconnected activity of billions of neurons. So off the top of my head, let me suggest, as others have, that information itself is consciousness: the current informational state, of the brain, of your smartphone, or of a rock, comes with a unified experience of that state. That experience just stands on its own — it is what an information state feels like, in and of itself, not needing any particular organism or homunculus to experience the experience. Now, confession, I just made that all up to answer this question, and that is the attraction of research in consciousness and in neuroscience, its theoretical landscape is wide open, as yet no more constrained than current new theories of the nature of space, time and matter. The difference is, we are trying to explain the existence of our inner world and all it can represent whereas physicists have to be content with explaining just the existence of the external world. My personal opinion is that the understanding of consciousness is the greater prize.

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Quick guide

Apicoplast

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What is it? An apicoplast (apicomplexan plastid) is a vestigial plastid found in parasites belonging to the phylum Apicomplexa. Plastids are better known as the green, subcellular compartment of plants and algae in which photosynthesis occurs. Apicoplasts are non-photosynthetic, pigment-free versions of plastids. Phylum Apicomplexa comprises some 6,000 species of parasites, the most notorious of which is the genus *Plasmodium* that causes malaria in humans, other primates, rodents, bats, birds and reptiles. Less deadly, but more common, is *Toxoplasma gondii*, an apicomplexan that infects most mammals (Figure 1). Apicomplexa also cause coccidiosis of fouls, red water fever of cattle, and babesiosis (tick fever) of cattle and dogs. The common human diarrhoeal apicomplexan *Cryptosporidium* is the only parasite in the group known to lack the apicoplast, though it might also be absent from gregarines, a large but poorly studied group of Apicomplexa that infects mostly invertebrates and protists.

Where did it come from? Plastids arose by endosymbiosis of a cyanobacterium approximately one billion years ago, and apicoplasts ultimately trace their ancestry back to this same event. After the initial (primary) endosymbiosis, secondary endosymbioses, in which one eukaryote engulfed and retained a plastid-containing eukaryote, created several new lines of photosynthetic organisms. Apicomplexa are the descendants of such a secondary endosymbiosis. The discovery in Australia of the coral symbiont *Chromera* solved the protracted debate about what kind of secondary endosymbiont apicomplexans acquired. Apicomplexa clearly harbour a red algal symbiont acquired by the common ancestor of *Chromera*, dinoflagellates and Apicomplexa ~400 million years ago. This ancestor was probably a symbiont of invertebrates. Its descendants developed into the

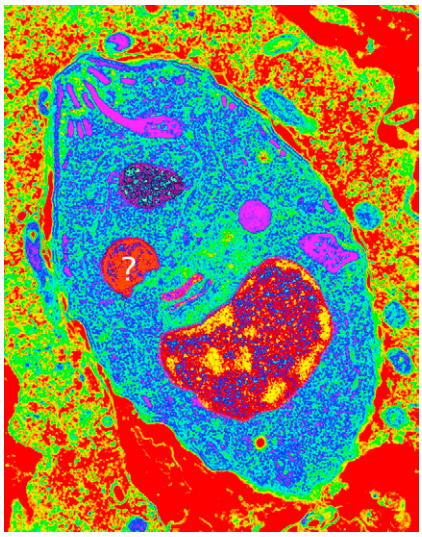

Figure 1. *Toxoplasma gondii*.
Electron micrograph of *Toxoplasma gondii* parasite inside a human fibroblast (pseudo-coloured) showing the apicoplast denoted with a question mark.

dinoflagellate zooxanthellae that live in corals, anemones, jellyfish and molluscs, while a separate lineage converted to parasitism and lost photosynthesis to create Apicomplexa. These parasites have likely co-evolved with their animal hosts for almost as long as animals have existed, evading immune attack and adapting complex life cycles to multiple hosts.

What does it do? When first identified in 1996, it was not at all obvious what the apicoplast did. Every apicoplast has a small circular genome (DNA) that encodes about 50–60 genes, but the sequences of the genes gave no clue to the organelle's vital role in parasite survival. The apicoplast seemed little more than a device for making copies of itself. Clever genetic and pharmacological experiments showed that apicoplasts are indispensable: without it parasites die. The full nuclear genome of the malaria parasite yielded the first clues to the apicoplast *raison d'être*, showing that apicoplasts make essential cellular building blocks such as fatty acids, isoprenoid precursors, haem and iron/sulphur clusters. Because apicoplasts have the same ancestry, the way they make these components is identical to the way plant plastids do. Although the genes provided a window into the apicoplast's potential for synthesis, they didn't tell us about the *when* or the *why*. Apicomplexan

parasites have complicated life cycles, often spread across vertebrate and invertebrate hosts and inhabiting multiple tissues therein. A roadmap of when particular apicoplast capacities are essential in each host for each parasite is gradually being pieced together. For instance, apicoplast fatty acid biosynthesis is essential to *Toxoplasma* parasites living in mice. However, in malaria parasites of mice apicoplast fatty acid biosynthesis is dispensable in the blood phase but essential in the liver phase. In human malaria parasites the isoprenoid precursor pathway is the only essential apicoplast function when the parasites inhabit our red blood cells.

Can we kill it? The apicoplast is essentially a reduced cyanobacterium living inside the parasite. Cyanobacteria are Gram-negative bacteria and sensitive to many antibiotics that target prokaryotic metabolism, and malaria parasites succumb to common antibacterials like ciprofloxacin, clindamycin and doxycycline, the latter being widely used as a malaria prophylactic. Initial suggestions that herbicides inhibit plant-like fatty acid biosynthesis pathways in malaria apicoplasts have proved unfounded. However, herbicidal antibacterials inhibiting apicoplast isoprenoid precursor synthesis are being pursued as novel antimalarials.

Where can I find out more?

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Sniffing patterns uncover implicit memory for undetected odors

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Consciously undetected events are represented at the sensory-motor level and in the neurons of sensory-motor control, for example, consciously undetected visual targets drive eye movements [1] and neural activity [2]. Olfaction offers an opportunity to investigate processing of undetected stimuli through measurements of the sniff-response: odorant-specific modulations of nasal airflow [3–6]. Here, we report evidence that consciously undetected odorants modulate sniffing in a predicted manner. Moreover, in our study we observed that sniff-modulations recurred at least 10 seconds after the onset of an undetected odor, implying that information which was not consciously perceived was nevertheless maintained in memory, available for future decision making.

To test the hypothesis that odors modulate sniff duration in the absence of conscious detection, we measured sniffs in 27 subjects (see the on-line Supplemental Information) during a maximum-likelihood, adaptive staircase olfactory detection task involving a forced-choice between two alternatives. Each trial entailed consecutive presentation of two jars (~10 s between jars), one containing an odorant diluted in mineral oil (*odor*), and the other containing mineral oil only (*blank*), counterbalanced for order. Participants sniffed each jar once and determined which contained an odor. Estimates of conscious perception were based on detection, and estimates of sensory-motor performance on concurrent precise sniff measurement.

Given expected canceling effects between trials where blank preceded odor and odor preceded blank (see the Supplemental Information), we analyzed these trials separately. For trials where blank preceded odor, an analysis of variance (ANOVA) with conditions of *odor* (odor/blank jar) and *accuracy* (correct/incorrect verbal report) revealed a main effect of *odor* ($F(1,26) = 30.37$, $p < 0.00005$), reflecting longer sniffs for blank than odor, no effect of *accuracy* ($F(1,26) = 0.01$, $p > 0.91$), and a significant interaction between *odor* and *accuracy* ($F(1,26) = 4.90$, $p < 0.05$), reflecting a larger difference between odor and blank in correct vs. incorrect trials. Follow-up tests revealed that sniff duration was longer for blank vs. odor for correct (blank = 2491.3 ± 705.2 ms, odor = 2312.4 ± 594.2 ms, $t(26) = 6.2$, $p < 0.0001$) and critically, also for incorrect trials (blank = 2450.0.4 ± 648.9 ms, odor = 2346.3 ± 648.7 ms, $t(26) = 3.4$, $p < 0.005$; Figure S1A), implying olfactory sensory-motor adjustments for events that were not consciously perceived (incorrect trials).

Given that in the above analysis blank was always first, it does not discriminate an effect of odor from an effect of order (Figure S1). To address this, we first analyzed the responses to the first sniff alone, thus avoiding order. An ANOVA on *odor* (odor/blank) and *accuracy* (correct/incorrect) revealed no effect of *odor* ($F(1,26) = 0.11$, $p > 0.74$), no effect of *accuracy* ($F(1,26) = 1.71$, $p > 0.20$) but a significant interaction ($F(1,26) = 11.52$, $p < 0.005$), reflecting longer sniff duration for blank vs. odor in correct trials (odor = 2417.2 ± 589.2 ms, blank = 2491.3 ± 635.7 ms, $t(26) = 2.7$, $p < 0.05$) and shorter in incorrect trials (odor = 2540.6 ± 674.1 ms, blank = 2450.0 ± 648.9 ms, $t(26) = 2.2$, $p < 0.05$; Figure 1A).

We next conducted two follow-up experiments in 54 subjects where trials of two consecutive blanks were embedded without participants' knowledge. In contrast to the expectation following an order effect, we found no difference in sniff duration between the two consecutive blanks (8.3 ± 146.9 ms difference, $t(53) = 0.42$, $p > 0.68$), and importantly, sniff duration difference between two consecutive blanks was significantly smaller than the sniff duration difference between blank and odor, in correct (175.9 ± 146.4 ms difference, $t(79) = 4.8$, $p < 0.00001$) and incorrect (108.6 ± 167.6 ms difference, $t(79) = 2.76$, $p < 0.01$; Figure 1B) trials.

Moreover, in an analysis of trials *around threshold* only (see the