to be more immediate and stereotyped than those that are studied within their natural ecological contexts. These artificially constrained, laboratory-evoked responses are then mapped on to neural circuits without opportunity to observe the graded, contingent and goal-directed nature of natural survival-related behaviors.

Third: the influence of scientists' own inferences is under-appreciated, leading them to confuse what is measured with what is inferred.

Fourth: the distinction between affect and emotion is underappreciated. Affective properties, like valence and arousal, are important features of emotional states, but they are not specific to those states. Affect derives from interoception, and so any brain state that involves interoception will necessarily have affective properties (at least in vertebrates).

RA: I agree with the first two. I would also add the lack of clarity in what people mean by 'emotion'. They use the word typically without explanation, and they often use it in very different senses. The problems are most apparent when trying to relate emotion research in animals (for example, studies of anxiety in rodents) with psychological studies in humans (for example, asking people how anxious they feel with a questionnaire). The dependent measures are vastly different, and so are the concepts of 'emotion' that the researchers are using. We need a common vocabulary.

LM: Any last words?

RA: I thank both of you for this discussion: Len for his help in bringing our viewpoints together and extensive editing, and Lisa for repeated and patient debates with me. I have the greatest respect for Lisa's view and scholarship, and do think she has identified a problem with my functionalist view that I don't know how to answer — how to pick the 'correct' function(s).

LFB: Len, I enthusiastically second Ralph's thanks for guiding us in this discussion. Ralph, my friend, it's always a pleasure. You are a generous colleague, a trusted critic, and I deeply admire your open-mindedness and willingness to consider a range of scientific views. I look forward to our next discussion!

LM: Thanks to you both. I think your insightful dialog illuminated many important issues in the field.

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Quick guide

Striga

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***What is* Striga?** *Striga* (witchweed) is a genus of parasitic plants belonging to the family Orobanchaceae. Five of the most economically devastating *Striga* species are *S. hermonthica* (Figure 1), *S. asiatica*, *S. forbesii*, and *S. aspera*, which infect sorghum (*Sorghum bicolor*), finger millet (*Eleusine coracana*), maize (*Zea mays*), and sugarcane (*Saccharum officinarum*); and *S. gesnerioides*, which infects cowpea (*Vigna unguiculata*). The resulting annual losses in cereal productivity alone, especially in sub-Saharan Africa, are over 1 billion USD.

How does* Striga *find a host? *Striga* plants produce several hundred thousand tiny seeds that can survive in soil for decades, germinating only in response to host root-derived germination stimulants such as strigolactones. Strigolactones are exuded by many non-parasitic plants to attract symbiotic mycorrhizal fungi. *Striga* spp. take advantage of this signal to detect nearby hosts. In fact, genes encoding strigolactone receptors are highly expanded in *Striga* genomes, allowing the detection of various strigolactone derivatives to ensure efficient host detection. *Striga* roots grow for a few millimeters toward the host root, but the exact mechanisms controlling this chemotaxis are unclear. Upon reaching the host root, the *Striga* root produces a multicellular structure called a haustorium, which invades the host. Haustorium initiation is stimulated by host-derived compounds called haustorium-inducing factors (HIFs). These include 2,6-dimethoxy-*p*-benzo-quinone (DMBQ), an oxidized lignin-derived molecule.

How does* Striga *infect the host? Haustorium initiation is followed by the development of haustorial hairs, which secrete adhesive substances that anchor the haustorium to the attachment site on the host. Epidermal cells at the haustorium apex enlarge to form intrusive cells, which produce host cell wall loosening and degrading enzymes. These allow the intrusive cells to invade

the host cortical parenchyma without apparent damage. Within a few days, the parasite establishes a connection with the host vasculature. *Striga* infection severely affects the growth and development of the host plant, with visible symptoms including desiccation, necrosis, and severe stunting.

What does* Striga *steal from the host? *Striga* spp. can photosynthesize, but they are obligate pathogens and require host plants to survive. Extraction of water from the hosts is crucial, especially in extreme dry conditions such as those in parts of sub-Saharan Africa. *Striga* spp. are often insensitive to abscisic acid, a plant hormone that controls the closing of stomata. This insensitivity allows *Striga* stomata to remain open and generate hydrological pressure for obtaining water from the host xylem. In addition to water, nutrients and organic solutes are also transferred from the host. However, since the phloem cells are not connected, the molecular mechanisms underlying this phenomenon remain unknown. Intriguingly, *Striga* transcriptome and genome studies indicate that host genes have also been transferred from hosts to *Striga* plants, indicating that nucleotide information can also be stolen by these parasites.

***How do hosts defend against* Striga?** The cowpea–*S. gesnerioides* interaction follows a gene-for-gene resistance mechanism. The host resistance gene encodes a nucleotide leucine rich repeat-type receptor that recognizes unidentified effector(s) from the pathogen. Rice cultivars that are resistant to *S. hermonthica* activate a lignin-related defense pathway meditated by a WRKY-type transcription factor. The locus that activates this pathway has been mapped, but the resistance gene at this locus has not yet been identified.

***How can we control* Striga?** One effective method for the control of *Striga* spp. is chemical treatment of the infested soil. Since *Striga* spp. cannot live without a host, enforced germination in the absence of host plants can be used to kill the parasites. The germination-inducing hormone ethylene was used to fumigate *Striga*-infested soils in the U.S. in the 1950s. Various strigolactone-mimicking chemicals have also been developed to specifically induce germination. In addition, the use of non-host plants that exude high levels of strigolactones has also been effective. Resistant cultivars and wild relatives of several crop species including sorghum, rice, and cowpea have been identified and will be used to create new resistant cultivars. For example, some sorghum lines are resistant to *S. hermonthica* due to their low germination stimulation activity.

Figure 1. Flowering *Striga hermonthica* in Kenya.
S. hermonthica mainly infects sorghum and maize in Kenya, and the farmers often abandon the infested field.

What is the future outlook for* Striga *study? Complete genome sequencing of more *Striga* spp., especially *S. hermonthica* and *S. gesnerioides*, which are the most problematic for agriculture, will deepen our understanding of the mechanisms and evolution of plant parasitism. Furthermore, the establishment of a transformation methodology for *Striga* spp. will provide a powerful tool for the functional analysis of their genes. Additionally, the identification of more host resistance genes will help to elucidate how plants resist infection, and in the development of new breeding strategies for controlling witchweed.

Where can I find out more?

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