Discussion draft

Quantifying the impact of parasitism on wild host populations is critical in both disease modeling and wildlife management. Ideally the relationship between infection intensity and host survival would be measured experimentally, but for logistical and ethical reasons, this is often impossible. Looking for evidence of mortality in parasite distribution data requires the least amount of information, but is notoriously difficult to implement. The methodological flaws in the adjei method limit its utility, so here we propose an alternative, likelihood based, method to estimated host survival and LD50 from observed parasite intensity data. At a theoretical level his method is a significant improvement because it, estimates fewer parameters, provides a statistically meaningful output and does not require extensive data manipulation.

Under simulated conditions, the likelihood method always out performs the earlier technique. For simply detecting the presence of PIHM, the likelihood method is both more powerful and has fewer false detection events (type 1 errors). When both methods were applied to published datasets previously used in PIHM analyses, the AM tended to detect PIHM where it had not previously been reported, consistent with the high type 1 error rate observed in our simulations. The likelihood method is also universally more precise and less biased in calculations of both the parasite ld50 and host survival curve. However, while only the likelihood method produces precise and unbiased LD50 estimates, neither method can provide unbiased estimates of the host survival function at realistic sample sizes. These simulations studies demonstrate that the novel likelihood method is more powerful and precise than AM, but even this preferred method may be limited by real world data sets.

Although superior to the Am, the LM may still not always be applicable to real data. The LM requires relatively large sample sizes (n>50-100), that although reasonable to obtain for invertebrates or small fish may be completely unfeasible for most vertebrates, particularly the species of conservation concern where addressing the impact of parasitism would be most important. An even larger sample size is required to capture the full parasite distribution when parasites are highly aggregated (k<.1), mean infection intensity is high, or parasite prevalence is low, all of which are common in many parasitic helminths. Low parasite induced host mortality, as might be predicted in many definitive hosts, may also be very difficult to detect and require astronomically large sample sizes. And, even when sample size is sufficient, these methods can only detect PIHM is the host survival curve is non-linear (L+B). Most host-parasite models assume a linear relationship between survival and infection intensity (eg A+M 1978), however nonlinear survival functions are not uncommon in empirical host-parasite systems (Benesh 2011). And, while linear functions make PIHM undetectable, at the other extreme, steep, non-linear survival curves produce severely biased estimates of the survival function. Even the LM is likely limited to detecting PIHM and estimated LD50 only in systems where large samples can be collected and parasites are common and only moderately aggregated, and substantial host mortality occurs at relatively low parasite intensity.

While we have improved on the existing methods for quantifying PIHM from parasite intensity data, all methods to do this require several fundamental, and potentially problematic assumptions. All methods to date derive from Crofton (1971) and assume that, prior to any parasite induced host mortality, parasites are distributed in the host population following a negative binomial distribution. But, it is fundamentally impossible to know what the pre-mortality parasite distribution was in a wild host population and it is widely recognized that different processes can lead to a variety of parasite distributions in hosts. However, the negative binomial is extremely flexible and there is substantial empirical and theoretical evidence to support the assumption that prior to any PIHM, parasite distribution can be modeled as a neg binom. [Other crofton assumptions worth mentioning?]. Unfortunately, this flexibility in the distribution may also reduce our ability to detect PIHM. If a negative binomial can be fit to the observed post-mortality parasite distribution then, regardless of how lethal the parasite was, it will be impossible to detect PIHM because there is no need for a more complex model. Most observed parasite distributions are well fit by the neg binom, suggesting that systems where these methods are applicable may be more the exception than the rule. And, even when truncation is detected, it may be caused by other processes such as density dependence, age dependent variation in host resistance and heterogeneous infection rate. Meaning, that in the rare event that PIHM is detected, it may actually not be the result of PIHM.

Conclude with existing last paragraph