NON-REPRODUCTIVE EFFECTS OF INSECT PARASITOIDS ON THEIR HOSTS

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ABSTRACT

The main modes of action of insect parasitoids are considered to be killing their hosts with egg laying followed by offspring development (reproductive mortality), and feeding on them directly (host feeding). However, parasitoids can also negatively affect their hosts in ways that do not contribute to current or future parasitoid reproduction (non-reproductive effects). Outcomes of non-reproductive effects for hosts can include death, altered behavior, altered reproduction, and altered development. Based on these outcomes and the variety of associated mechanisms we categorize non-reproductive effects into: (i) non-consumptive effects; (ii) mutilation; (iii) pseudoparasitism; (iv) immune defense costs; and (v) aborted parasitism. These effects are widespread and can cause greater impacts on host populations than successful parasitism or host feeding. Non-reproductive effects constitute a hidden dimension of host-parasitoid trophic networks, with theoretical implications for community ecology as well as applied importance for the evaluation of ecosystem services provided by parasitoid biological control agents.

Keywords: parasitism, population dynamics, trophic networks, pseudoparasitism, non-consumptive effects, mutilation

1. INTRODUCTION

Insect parasitoids are important components of natural communities and are used to control insect pests in biological control programs worldwide, in addition to being fruitful models in theoretical ecology (Godfray 1994; Jervis 2007; Wajnberg et al. 2008; Heimpel and Mills 2017). As biological control agents, their main mode of action is considered to be killing their hosts as a result of egg laying and offspring development, what we will term "reproductive parasitoid-induced mortality" (Fig. 1A). In many parasitoid species, adult females also feed directly on hosts (Jervis & Kidd 1986). Host feeding is another well understood mode of action of parasitoids, and contributes to future parasitoid reproduction (*via* nutrient intake necessary for egg maturation) while often causing host death (Fig. 1B) (Kidd and Jervis 1989; Heimpel and Rosenheim 1995; Giron et al. 2004). Parasitoids can, however, negatively affect their hosts without their offspring successfully developing or directly feeding on them. These modes of action – which we will term "non-reproductive effects"– can have a variety of negative consequences for hosts (including mortality), but do not carry clear benefits for parasitoids in terms of current or future reproduction (Fig. 1C).

Non-reproductive effects of parasitoids on their hosts and their associated mechanisms and outcomes are scattered throughout the scientific literature under highly variable terminology, and have never been properly defined and categorized. For example, at least nine terms have previously been used to describe host mortality without parasitoid reproduction: unsuccessful parasitism, non-reproductive killing, hypersensitivity, dudding, host destruction, surplus killing, abortion, residual mortality, and parasitoid-induced other mortality. These effects remain largely underappreciated and it is likely that they have often been overlooked. As previously recognized by several authors (e.g., Legner 1979; Van Driesche 1983; Barnay et al. 1999), failing to explicitly consider and measure non-reproductive effects underestimates the impact of parasitoids by neglecting a major component of their direct and indirect ecological effects within insect communities. Ignoring non-reproductive effects also reduces the realism of host-parasitoid trophic networks and population dynamics models, most of which currently assume that each parasitoid oviposition results in host death and parasitoid offspring production (*sensu* Brodeur and Rosenheim 2000; Condon et al. 2014; but see Heimpel et al. 2003; Abram et al. 2016; Kaser and Heimpel 2015; Kaser et al. 2018). The aim of this paper is to review and categorize the effects of parasitoids that do not involve host feeding or the succesful production of offspring, but have consequences for host and parasitoid fitness – and, therefore, have important implications for the evolution of host-parasitoid interactions, population dynamics, and biological control.

2. MECHANISMS AND CONSEQUENCES OF NON-REPRODUCTIVE EFFECTS

Parasitism involves a sequence of processes that enables parasitoids to locate, select, parasitize, and alter host physiology to allow the development of their offspring in the selected hosts (Vinson 1976; Vet and Dicke,1992; Godfray 1994). During this sequence, several mechanisms can result in a variety of outcomes that affect host and parasitoid fitness, even when the encounter does not end with the successful development of the parasitoid's offspring (Fig. 2). Based on these mechanism-outcome associations, we propose a classification system, which will help to unify terminology and describe parasitoids' non-reproductive effects in the future. We classified mechanisms following the chronological order of the parasitism sequence: (i) host searching and acceptance behaviors that occur before ovipositor probing; (ii) ovipositor probing that includes physical damage and injection of chemical (e.g., venom) or biological (e.g., symbionts, viruses) factors; and (iii) egg laying followed by unsuccessful development of immature parasitoids (as eggs, larvae, or pupae) (Fig. 2). The resulting effects of each of these mechanisms can have a variety of outcomes for hosts. The outcomes, in order of increasing general impact on host fitness, are: (i) altered behavior; (ii) altered development; (iii) altered reproduction; and (iv) death (Fig. 2). Based on the processes resulting from each mechanism, we developed unified terms for five types of non-reproductive effects (Fig. 2), which are reviewed below. Terms were chosen based on their descriptive value as well as their current level of usage in the literature we reviewed.

2.1 Non-consumptive effects

Non-consumptive (or "trait-mediated") interactions, constituting effects of natural enemies that do not result in prey or host consumption but cause them to adopt costly defensive behaviors, are increasingly being recognized as critically important components of ecological processes (Priesser and Bolnick 2008; Hermann and Landis 2017). For parasitoids, non-consumptive interactions refer to non-reproductive effects in which parasitoids reduce host fitness, which can occur before they insert their ovipositor into the host but could also result from unsuccessful attacks by the parasitoid that hosts survive. One of the best-known examples is the dispersion of aphids responding to the alarm pheromone of nearby conspecifics being attacked by parasitoids. Many of these dispersing aphids drop from the plant to the ground (Tamaki et al. 1970; Dill et al. 1990; Gowling and van Emden 1994) and some of them that cannot return to the plant die from starvation, desiccation (especially at high temperatures), or predation by ground-dwelling arthropods (Gowling and van Emden 1994; Roitberg and Myers 1979). Similar effects may also result from aphids being directly contacted by parasitoids (e.g., through antennation) (Ingerslew and Finke 2016). Tamaki et al. (1970) were the first to measure the population-level consequences of non-consumptive effects in an aphid population using the pea aphid *Acrythosiphon pisum* (Harris) (Hemiptera: Aphididae). They showed that altered females (with ablated ovipositors that prevent parasitism) of the parasitoid *Aphidius smithi* Sharma & Subba Rao (Hymenoptera: Braconidae) caused about a 30% reduction in population growth of aphids relative to undisturbed controls. The presence of parasitoids can also alter host development (Fig. 2). For example, the proportion of winged dispersing offspring of the pea aphid *A. pisum* increases between 10-30% when female parasitoids were present (Sloggett and Weisser, 2002).

Other non-consumptive effect of parasitoids that result in costly host behavioral modification has been described in the seed beetle *Mimosestes amicus* (Horn) (Coleoptera: Bruchidae). Females sometimes cover their viable eggs with additional unviable eggs to reduce mortality of the protected eggs from parasitism by *Uscana semifumipennis* Girault (Hymenoptera: Trichogrammatidae) when adults or immature stages (i.e., parasitized eggs) of the parasitoid are present (Deas and Hunter 2011, 2013). However, this defensive strategy also incurs a cost for the host; the reproductive output of parasitoid-exposed beetles (which show high rates of defensive egg stacking) is up to ~50% lower than that of control beetles (Deas and Hunter 2011).

2.2 Mutilation and pseudoparasitism

When female parasitoids find a host, they may insert their ovipositor to assess the suitability of the host or to destroy competing offspring (ovicide or larvicide) before laying eggs. During this process, which may involve the injection of viruses, venom, teratocytes, or other biological/chemical factors, parasitoids may ultimately reject the hosts, or can be disrupted by predators, host defensive behaviors, competitors, or abiotic factors before laying an egg. For example, the parasitoid complex of the moth *Epinotia tedella* (Clerck) (Lepidoptera: Tortricidae) rejects about 75% of the larvae they probe (Münster-Swendsen 2002). Oviposition by parasitoids in the genera *Aphytis* (Hymenoptera: Aphelinidae) and *Metaphycus* (Hymenoptera: Encyrtidae) is often disrupted by ants (Barzman and Daane 2001;

Martinez-Ferrer et al. 2002). Two different, but often coinciding, non-reproductive effects can result from ovipositor insertion without egg laying: mutilation and pseudoparasitism (Fig. 2). While mutilation can result from the mechanical damage (i.e., puncturing) to hosts resulting from ovipositor insertion during probing (Flanders 1953; Quendau et al. 1970), pseudoparasitism occurs when parasitoids inject chemical substances without laying eggs that alter host physiology (Jones et al. 1981; Münster-Swendsen 1994).

The most commonly reported outcome of mutilation and pseudoparasitism on hosts is death. Interestingly, when parasitism, host feeding and mutilation/pseudoparasitism have been measured in a given host-parasitoid association, mutilation/pseudoparasitism can sometimes be the principal cause of host mortality. For example, Campbell (1963) estimated that *ltoplectis conquisitor* (Say) (Hymenoptera: Braconidae), a parasitoid of the gypsy moth *Lymantria dispar* (L.) (Lepidoptera: Lymantriidae), kills three hosts by lethal probing for every one host killed by host feeding, and 200 hosts by lethal probing for every one parasitoid offspring produced. Mutilation and pseudoparasitism can also have sub-lethal effects for surviving hosts, resulting in lower reproductive output (Cebolla et al. 2018; Ingerslew and Finke 2016). Finally, it is possible that mutilation may favor secondary infection of hosts by entomopathogens. Despite their potential detrimental effects on host populations, mutilation and pseudoparasitism have often been underestimated. Indeed, these effects are not always discernible from other causes (Legner 1979; Barrett and Brunner 1990; Mandeville and Mulleins 1992; Urbaneja et al. 2000; Lysyk et al. 2004; Grabenweger et al. 2009; Keinan et al. 2012), or are sometimes erroneously attributed to host feeding (e.g., DeBach 1943).

In some cases, effects of mutilation can be separated from unequivocal pseudoparasitism, which refers to the physiological changes in living hosts resulting from the injection of biological/chemical factors by parasitoid females or larvae (Brown and Kainoh 1992; Jones et al. 1986b; Munster-Swendsen 1994; Tillinger et al., 2004; Vereijssen et al. 2011). Host regulation by parasitoids (*sensu* Vinson and Iwantsch 1980) begins with the injection of various factors when adult females probe hosts, even in the absence of oviposition or immature parasitoid development (Jones 1986). The chemical composition of injected substances and their effects on host physiology were reviewed in detail by Asgari and Rivers (2011) and Strand (2014). Pseudoparasitism can result in temporary or permanent paralysis, suppression of immune responses, altered host development, reduced adult longevity and fecundity, host castration, and death (Tillinger et al., 2004; Asgari and Rivers 2011; Ingerslew and Finke 2016). These effects arise from the need of parasitoids to regulate host endocrinology in order to successfully complete immature development (Strand et al., 1983; Jones 1986; Asgari and Rivers 2011), but in the case of pseudoparasitism they represent pathological symptoms with no function for the parasitoid. Unfortunately, studies focusing on host-parasitoid physiological interactions are rarely accompanied by investigations of the frequency of pseudoparasitism in nature and its implications for host-parasitoid evolutionary relationships, population dynamics, and biological control. One notable exception consists of several studies of the forest pest *Epinotia tedella* (Cl.) (Lepidoptera: Tortricidae) in Denmark (Münster-Swendsen 1994, 2002). In this system, host larvae often escape parasitism by *Apanteles* (Hymenoptera: Braconidae) and *Pimplopterus* (Hymenoptera: Ichneumonidae) following probing but before the parasitoid lays an egg, possibly due to environmental factors and/or host defensive behavior. However, parasitoid probing (which likely includes the injection of chemical substances) results in less fertile or completely sterile hosts. Population dynamic models fit to long-term host-parasitoid population data showed that rates of successful parasitism were too low to account for the high correspondence between relative parasitism rates and variation in host densities from year to year. Rather, reduced host fertility (which was highly correlated with parasitism) was the key factor explaining variation in host densities. Including pseudoparasitism as the proximate cause of reduced host fertility in the model resolved this issue, with the assumption that 75% of all parasitoid attacks resulted in pseudoparasitism giving the best model fit (explaining 70-80% of the variation in host densities).

2.3 Immune defense costs and aborted parasitism

Parasitoids frequently lay their eggs in hosts that do not support the development of their offspring (Abram et al., 2016; Blumberg 1997; Kraaijeveld and Godfray, 1997; Heimpel et al. 2003; Desneux et al. 2009; Condon et al. 2014). Mortality of parasitoid offspring can occur at any time during parasitoid development and can have a variety of consequences for the host (Fig. 2). We designate two terms for non-reproductive effects caused by parasitoid oviposition followed by unsuccessful offspring development, depending on whether the negative effects for hosts are mediated by the host's immune system ("immune defense costs") or the immature parasitoid prior to its death ("aborted parasitism"). We recognize that these two terms do not necessarily describe mutually exclusive processes because immune responses may sometimes be involved in aborted parasitism.

Immune defense costs are observed in host individuals surviving parasitism, and result from the physiological cost of mounting an immune defense against the immature parasitoid (e.g., Kraaijeveld and Godfray 1997). For example, mounting an immune defense can negatively impact the body size, survival, offspring viability, fecundity, mating capacity, and competitive ability of surviving hosts (Kraaijeveld and Godfray, 1997; Fellowes *et al.* 1998; Fellowes et al. 1999; Carton and David 1983; Hoang et al. 2001; Niogret et al. 2009; Lynch et al. 2016). For host life stages with immune responses, the immune system is activated when female parasitoids insert their ovipositors into hosts (Rivers et al. 2002), with the aim of inhibiting or preventing the development of immature parasitoids. Encapsulation is the most common defense mechanism mounted by host immune system in response to parasitoid eggs, and it can have a variety of consequences for the host (Salt 1968; Strand, 1986; Strand and Pech 1995; Blumberg 1997). In some cases, parasitoid eggs are eliminated with minimal negative fitness consequences (e.g., Hoogendoorn and Heimpel, 2002). In more severe cases, the immune response has negative impacts on surviving host fitness (see above), or the immune system is exhausted and the host becomes more vulnerable to subsequent parasitoid attacks (Blumberg 1997; Tena et al., 2008). For example, when the host immune system is exhausted as the result of a first parasitism event that results in encapsulation, subsequent parasitism attempts (self- or conspecific-superparasitism, or multiparasitism) are more likely to result in successful parasitoid offspring development to adulthood (Blumberg and Goldenberg 1992; Guzo and Stoltz 1985; Ode and Rosenheim 1998; Tena et al., 2008). All studies describing host immune system exhaustion are based on laboratory assays to date; thus, its incidence in the field remains unknown. This non-reproductive effect would likely be common under conditions of high parasitism levels in the field (when super- and multi-parasitism are more likely), when as well as under mass rearing conditions or in laboratory colonies.

Aborted parasitism occurs when developing parasitoids die for reasons other than the activation of the host immune system, and their host also dies due to actions of the immature parasitoid prior to its own death. For example, Gariepy et al. (2008) observed that 30-60% of *Peristenus* spp. offspring developing in plant bugs (Hemiptera: Miridae) advanced to the pupal stage, killing the host, but failed to emerge as adult wasps. Depending on the host-parasitoid association, reasons for host death during aborted parasitism may include some combination of immature parasitoid feeding or the release of chemical compounds or teratocytes. The immature parasitoid may die due to poor host nutritional suitability, adverse environmental conditions, or competition. However, the exact mechanism by which the immature parasitoid causes host death is usually unclear. Quantifying the mortality caused by parasitism abortion can be relatively easy when parasitoids die late in their development because they can be observed directly (Tena et al. 2012) or by performing host dissections (Gariepy et al. 2008; Abram et al. 2016). Aborted parasitoid eggs are often more challenging to detect; however, levels of aborted parasitism can still be estimated by comparing host mortality in the presence and absence of parasitoids (e.g., Abram et al. 2016). Aborted parasitism can represent the main cause of host mortality in many host-parasitoid associations (Desneux et al., 2009; Abram et al., 2016). However, as with other non-reproductive effects, aborted parasitism is not always distinguished from other causes of mortality (e.g., Minot and Leonard 1976; Godwin and Odell 1984; Scholler et al. 1996; Huang et al. 2017).

3. OCCURRENCE, CONDITION-DEPENDENCE, AND COSTS

3.1 Occurence across taxa, host stages, and life history characteristics

Non-reproductive effects of parasitoids on their hosts appear to be general phenomena distributed across diverse host and parasitoid taxa with varying life histories, acting on a variety of host life stages. Significant non-reproductive effects have been observed in holometabolous (Lepidoptera, Coleoptera, Diptera) and heterometabolous host taxa (Hemiptera) acting on host eggs (e.g., Abram et al. 2016; Lashomb et al. 1987), nymphs (Flanders 1953; Tena et al., 2008; Desneux et al. 2009; Ingerslew and Finke 2016; Cebolla et al. 2018), larvae (Campbell 1963; Rahman 1970; Carton et al. 1983; Münster-Swendsen 1994; Liu et al. 2015), pupae (Pimentel et al. 1978; Legner 1979; Geden et al. 2006, Geden and Moon 2009), and adults (Fusco and Hower 1974; Barratt et al. 1996; Goldson et al. 2004; Deas and Hunter 2011, 2013). Some qualitative trends and delimitations are notable:

(i) Non-consumptive effects have been observed in mobile life stages of hosts (nymphs, larvae, adults) whose behaviorcan be altered by the presence of parasitoids (Tamaki et al. 1970; Sloggett and Weisser 2002; Fill et al. 2012; Deas and Hunter 2011, 2013; Ingerslew and Finke 2016); however, immobile host life stages may also be able to detect parasitoid presence and alter defensive morphology or physiology.

(ii) Unequivocal pseudoparasitism has been observed only in koinobiont larval, egg-larval, or adult endoparasitoids (which often inject venom or other regulatory substances but keep their hosts alive) (Brown and Kainoh 1992; Jones et al. 1986b; Munster-Swendsen et al. 2002; Vereijssen et al. 2011). Permanent paralysis is mostly induced by idiobiont ectoparasitoid of lepidopteran larvae (Coudron et al. 1990; Casas, 1989; Charles et al. 2013).

(iii) Immune defense costs should be unique to endoparasitoids of insect life stages with encapsulation or other, similar immune defenses; for example, in dipteran pupae and larvae, hemipteran nymphs, and lepidopteran larvae (Pimentel et al. 1978; Kraaijeveld and Godfray 1997; Hoang et al. 2001; Tena et al., 2008; Niogret et al. 2009).

(iv) Aborted parasitism appears to be the most widespread non-reproductive effect type, occuring any time there is unsuccessful parasitoid development that causes death of any host life stage, in both endoparasitoids (Gariepy et al. 2008; Tena et al. 2012) and ectoparasitoids (Pawson and Peterson 1988; Kapranas et al. 2016), and koinobiont (Ryan 1985; Desneux et al. 2009) as well as idiobiont (Duan et al. 2014; Abram et al. 2016) parasitoid species.

These general observations should be considered preliminary, however, as studies of non-reproductive effects have been focused on a restricted set of host-parasitoid study systems. More taxonomically widespread studies that make use of the unified terminology proposed herein, and take the possibility of non-reproductive effects into account – even in associations where they are less prevalent – would help to identify more systematic, quantitative patterns in the future.

3.2 Condition-dependence

Broadly, the incidence and magnitude of non-reproductive effects are influenced by similar factors as reproductive effects (parasitism). These factors include host characteristics (e.g., species, age), parasitoid characteristics (species, strain, nutritional regime), ecological variables (exposure time or number of attacks, ratios of hosts to parasitoids, presence of mutualist species), environmental factors (temperature), and more applied considerations (number of generations a parasitoid has been in culture, different shipments from commercial supplier) (Table 1). However, the relative impacts of a given factor on reproductive versus non-reproductive effects may differ, or even have different tendencies. For example, *Bactrocera curcurbitae* (Diptera: Tephritidae) pupal mortality from mutilation and/or pseudoparasitism by *Spalangia endius* (Hymenoptera: Pteromalidae) decreases with host age, whereas successful parasitism is highest at intermediate host age (Tang et al. 2015). This negative relationship between host age and mortality from mutilation/pseudoparasitism appears to be a general trend (Table 1). Martinez-Ferrer et al. (2003) found that the presence of host-attending ants reduced the negative effects of probing-induced mutilation of *Aonidiella aurantii* (Hemiptera: Diaspididae) by *Aphytis melinus* (Hymenoptera:

Aphelinidae); however, the reduction in probing was less than the reduction of reproductive parasitism, possibly because parasitism takes longer and is more likely to be interrupted by ants.

3.3 Costs and benefits for parasitoids

By definition, non-reproductive effects do not result in the production of parasitoid offspring (i.e., fitness gain). Thus, from the point of view of the parasitoid, non-reproductive effects can mostly be considered as costly constraints, or inefficiencies, arising from the need to search for hosts and overcome their structural, behavioral, and physiological defenses in the face of varying levels of host availability, host quality, competition, and environmental stress. Depending on the host-parasitoid system, the outcome, and the mechanism underlying the non-reproductive effect – that is, how far along in the host exploitation sequence the mechanism sits (Fig. 2) – these costs could range from minimal to relatively severe for parasitoids. For example, normal parasitoid foraging behaviors that mediate some non-consumptive effects (e.g., antennation; Ingerslew and Finke 2016) would not incur any particular cost to the parasitoid, but probing and drilling behaviors that can cause host mutilation can be extremely costly in time (Beltra et al. 2015) and metabolic energy (Boisseau et al. 2017). Although we are unaware of any direct investigations into the cost of host regulatory chemical substances (e.g., venom) in parasitoids, it is plausible that there is a cost to their production and wasteful use during pseudoparasitism (Casewell et al. 2013; Kapranas et al. 2012). Reduction in host availability for parasitism and host-feeding would be a cost shared by all forms of non-reproductive effects. For example, non-consumptive effects can result in host dispersal from the local patch (e.g. crawling away, dropping) (Fill et al. 2012), and reduced host reproduction (Deas and Hunter 2011). Mutilation, pseudoparasitism, and aborted parasitism, by killing hosts or lowering their quality, directly reduce the proportion of suitable hosts in a given environment (Islam et al. 1997). Finally, the non-reproductive effects of costly host immune defense and aborted parasitism involve a cost for the parasitoid of laying eggs that do not result in offspring production. The fitness cost for the parasitoid of wasted eggs would be more severe if the parasitoid routinely experiences egg limitation (as opposed to time limitation) in a particular environment (Rosenheim 1999).

While most instances of non-reproductive effects of parasitoids on their hosts are certainly costly for parasitoids, a few studies have proposed potential fitness benefits. For example, in some cases, not using a given host for offspring production (while incidentally having non-reproductive effects on the host) may be adaptive; for example, when a parasitoid mutilates a host while probing but determines that it is low-quality or unsuitable for parasitism (Desneux et al. 2009). In another relatively well-known example, parasitoids can exhaust the immune defense system of hosts that otherwise would be unsuitable by superparasitizing them (van Alphen and Visser 1990; Tena et al. 2008). Patel et al. (2003) proposed that host-killing (mutilation/pseudoparasitism) of *Liriomyza trifolii* (Diptera: Agromyzidae) larvae by *Diglyphus intermedius* (Hymenoptera: Eulophidae) could be an adaptive strategy by the parasitoid to prevent excessive leafmining by other unparasitized larvae on the same leaflet, which can cause desiccation, necrosis, and abscission, and thus reduce the chance of parasitoid offspring survival. In addition, killing unparasitized hosts may also increase the amount of resources available to parasitized hosts that continue to feed. Similarly, Vereijssen et al. (2011) argued that once a parasitoid has parasitized a host in a given patch, destroying other hosts in the patch could increase fitness (representation of her genome in the next generation) by reducing host availability for other parasitoid individuals. Pseudoparasitism could be beneficial for parasitoids at the population level if it lengthens the suitable stage of hosts by changing the concentration of juvenile hormone (Jones 1986), making them susceptible to parasitism for longer periods. Further theoretical work, including modeling, and experimental studies are needed to directly demonstrate an adaptive value for parasitoids of inducing non-reproductive effects on hosts.

4. ECOLOGICAL AND EVOLUTIONARY IMPLICATIONS OF NON-REPRODUCTIVE EFFECTS

While non-reproductive effects result from aspects of parasitoid behavior and physiology that generally serve adaptive functions (host location, host feeding, host exploitation), the resulting effects themselves cannot be considered as adaptive strategies evolved by parasitoids to either exploit their hosts or expand their host range. However, from an evolutionary perspective, non-reproductive parasitism events could theoretically create favourable conditions for the establishment of new, viable host-parasitoid associations. Opportunities may occur for parasitoids to expand their host range following encounters with a potential new host species, as long as parasitoids possess pre-adaptations for location, development and reproduction on the novel host (Price 1980, Poulin 2011). These conditions may exist when an invasive species possessing similar ecological and physiological attributes to ‘traditional’ hosts becomes abundant in the environment. At first, maladaptive oviposition decisions by parasitoids causing non-reproductive effects could intensify (Heimpel et al. 2003), leading to an evolutionary trap for indigenous parasitoids (Schlaepfer et al. 2005;

Abram et al. 2014). For example, Abram et al. (2014) showed that the indigenous generalist egg parasitoid *Telenomus podisi* Ashmead (Hymenoptera: Scelionidae) accepts eggs of the newly invasive alien stink bug *Halyomorpha halys* (Stål) (Hemiptera: Pentatomidae) at high rates and causes some host eggs to abort development, but their offspring cannot successfully develop. In this association, parasitoid females not wasting time and eggs in *H. halys* should pass more copies of their genes to the next generation, thereby relaxing the frequency of non-reproductive effects on the host. On a microevolutionary time scale, however, behavioural mechanisms could evolve to minimize oviposition mistakes, and/or physiological attributes could be selected for that would improve the success of offspring development following these mistakes. Non-reproductive effects that formerly only killed the host (e.g., aborted parasitism) could pave the way for mutants in the parasitoid population whose offspring can occasionally undergo successful development in the invasive species. Thus, oviposition events that were formerly highly maladaptive would result in some offspring production, gradually relaxing selective pressure to avoid laying eggs in the invasive host. Ultimately, such a process could, in theory, lead to speciation in populations of parasitoids gaining the capacity to successfully attack novel hosts (Stireman et al. 2006). The role of non-reproductive events in shaping parasitoid host range would be determined by ecological and physiological pre-adaptations to host exploitation, frequency of encounters with potential new hosts, the amount of genetic variation in relevant physiological and behavioural traits, and subsequent fitness benefits.

There is currently little empirical evidence concerning the quantitative impacts of non-reproductive effects of parasitoids on host-parasitoid interactions at population and community scales (but see Munster-Swendsen 1994; Munster-Swendsen 2002). However, a theoretical framework, mostly through modeling, is currently being developed and will help to acknowledge the relative importance played by non-reproductive parasitoid mortality in mediating direct and indirect effects at the ecosystem level (Abram et al. 2016; Kaser et al. 2018). Abram et al. (2016) first developed a heuristic population dynamic model of egg-limited parasitoids inducing aborted parasitism on host eggs. They concluded that abortion negatively impacts population growth of the host (and to a lesser extent, the parasitoid), and that the consequences for parasitoids are worst when parasitoid females are egg-limited. Kaser et al. (2018) investigated the population-level consequences of non-reproductive effects of parasitoids in the context of novel associations between invasive and native hosts attacked by native or exotic parasitoids. Indirect effects mediated by parasitoids were examined by constructing models examining population-level outcomes of non-reproductive mortality for suitable (co-evolved associations) and unsuitable hosts (novel associations) attacked by either egg-limited or time-limited parasitoids. From model outputs, they concluded that non-reproductive parasitoid induced host mortality would influence indirect interactions in varying and often complex ways including apparent competition, apparent parasitism, apparent amensalism and apparent commensalism, depending on the mechanism of aborted parasitism and the degree of parasitoid egg limitation.

Parasitoids are ubiquitous in terrestrial ecosystems and parasitism can exert a significant influence on food webs (Hawkins 1994; Quicke 2015). However, to our knowledge, there is no evidence concerning the contribution of non-reproductive effects to the structure and stability of ecological communities. Investigating the functional role played by this largely 'hidden dimension' (*sensu* Condon et al. 2014) represents a new challenge for population ecologists. Broadening current host-parasitoid population and community models by adding the non-reproductive dimension could lead to a more comprehensive appreciation of the true range of interactions that determine community structure and function, and help to understand the potential of these effects to cascade through food chains and influence ecosystem services such as biological control (see section 5).

5. IMPLICATIONS FOR BIOLOGICAL CONTROL OF INSECT PESTS

Over the past several decades, a number of researchers have argued that only measuring host death from host feeding or resulting in parasitoid offspring emergence underestimates biological control impact of parasitoids (e.g., Campbell 1963; Legner 1979; Van Driesche 1983; Neuenschwander and Madojemu 1986; Munster-Swendsen 1994; Abram et al. 2016; Cebolla et al. 2018). Our integrative review of a large body of literature further emphasizes this point, showing that non-reproductive effects are widespread, diverse, condition-dependent (Table 1), and often comprise a large component of the impact of parasitoids on host populations. Thus, it is critical for biological control researchers to measure these effects in order to fully appreciate the magnitude of ecosystem services provided by these important natural enemies. However, the significance of non-reproductive effects for the applied aims of biological control programs depends on the approach (see also Abram et al. 2016); for example:

Inundative/Augmentative biological control: Inundative releases of parasitoids aiming for short-term pest control can be successful even if most of the mortality caused to hosts is the result of non-reproductive effects (Huang et al. 2017). In mass-rearing systems for inundative and augmentative biological control programs, non-reproductive effects can be ambiguous: on one hand, host immune system exhaustion due to superparasitism can allow the use of host material for rearing that would otherwise be unsuitable (Tena et al. 2008); on the other hand, they can lead to wasted or low-quality host material (e.g., dead or low-quality hosts that yield poor-quality parasitoids or no parasitoids at all).

Conservation biological control: In our literature search, we did not find any case where non-reproductive effects of parasitoids were explicitly considered as part of conservation biological control programs (i.e., habitat management to provide nectar, shelter, alternative hosts, etc.; see Begg et al. 2017). However, conservation biological control strategies that provide alternative hosts to parasitoids (e.g., Bernal et al., 2001; Kapranas 2007) could also consider conserving parasitoids that cause non-reproductive effects on the target host.

Classical biological control: Ecological risk assessments for the introduction of parasitoids include estimating risk of candidate parasitoids to non-target host species in the proposed area of introduction. It is generally assumed that if a given host species is attacked by a candidate parasitoid (i.e., the parasitoid lays eggs in them) but is of low suitability for the immature parasitoid development, then this host is not under any real threat because it will be unable to support parasitoid populations (Heimpel and Mills 2017). This assumption may not be valid if the severity of non-reproductive effects are large, and the suitable target species that would support parasitoid populations often co-occurs (e.g., on the same host plant in the same habitat) with the unsuitable non-target species (see Condon et al. 2014). When non-target impact as a result of non-reproductive effects of parasitoids could potentially be severe and sustained, we suggest that these effects should be included as additional parameters used in non-target host range evaluation.

To support a better understanding of the contribution of non-reproductive effects to the various types of biological pest control, we advocate for standardized measurement, calculation, and reporting. For example, non-reproductive effects should be: (i) reported with clear terminology along with known or hypothetical mechanisms and outcomes, according to our framework; (ii) reported separately from reproductive effects; (iii) reported as a percentage of the total number of hosts exposed to parasitism, and, when behavioral observations are available, the percentage of hosts attacked by the parasitoid; (iv) measured in assessments of parasitoid impact under various conditions by including unexposed controls in the experimental design to allow correction of mortality rates of exposed individuals (i.e. with Abbott's formula; see Abram et al. 2016); (v) estimated in the field by applying corrections derived from laboratory studies, when direct measurement in the field is not feasible (e.g. Van Driesche et al. 1987; Van Driesche et al. 1990). Sub-lethal non-reproductive effects (e.g., reduction of host fecundity or longevity) could similarly be included in estimates of population-level impacts of parasitoids following calculations used to account for sub-lethal effects of pesticides (Sterk et al., 1999). Efforts should also be made to validate the accuracy of lab estimates in predicting field estimates of non-reproductive effects. Use of molecular diagnostic tools to assess levels of unsuccessful parasitoid attack could prove very useful in this regard (Gariepy et al. 2014; Condon et al. 2014; Gómez-Marco et al., 2015).

FUTURE ISSUES

1. Non-reproductive effects should be included in models of host-parasitoid population dynamics and trophic networks.
2. Theory and empirical data are needed to determine whether non-reproductive effects could increase parasitoid fitness under some conditions; for example, as a competitive tactic to increase resource availability for parasitized hosts.
3. The role of community interactions (e.g., interactions with host mutualists, predators, competitors) on the frequency and magnitude of non-reproductive effects should be further examined.
4. Quantitative and molecular methods should continue to be developed and implemented to evaluate the occurrence of non-reproductive effects in the field, and resulting impact on host populations.
5. The role of symbiotic and pathogenic microorganisms in non-reproductive effects of insect parasitoids on their hosts should be explored.

DISCLOSURE STATEMENT

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Augmentative biological control: Intentionally increasing populations of an established biological control agent through the release of additional individuals.

Classical biological control: The intentional introduction of an exotic biological agent for long-term pest control, involving the agent's permanent establishment of self-sustaining populations.

Conservation biological control: Manipulative management of habitats that increases population sizes of biological control agents and their impact on pests.

Ectoparasitoid: Species in which eggs are laid and larvae develop on the exterior of hosts.

Encapsulation: Cellular immune defense of insects against their parasitoids.

Endoparasitoid: Species in which egg laying and development occurs within the host.

Idiobiont parasitoids: Species that prevent further development of their host after oviposition by the maternal parasitoid.

Inundative biological control: Application of large numbers of a biological agent to immediately reduce a pest population.

Koinobiont parasitoids: Species whose hosts continue to feed and develop after parasitization, before eventually being killed by the immature parasitoid.

Table 1. Condition-dependence of non-reproductive effects by effect type. Mutilation, pseudoparasitism, and aborted parasitism are presented as one category because definitive categorization of many studies was not possible due to lack of mechanistic understanding.

Effect type(s)	**Factor**	**Relationship to strength of non-reproductive effects [References]**
Non-consumptive effects	Host age/stage	positive [1]; negative [2]
	Presence of parasitized hosts	positive [3]
	Presence of female versus male parasitoids	positive [4]
	Extreme high temperatures	positive [5]
	Host plant resistance	positive [6]
	Presence of other host species	positive [7]
Mutilation/ Pseudoparasitism/ Aborted Parasitism*	Host species	varies among species [8–15]
	Host age/stage	positive [16–18]; negative [19–26]; hump-shaped [27,28]; no effect [29,30]
	Smaller host size distribution	positive [31]
	Host rearing substrate	varies among substrates [32,33]
	Parasitoid age	no effect [29]
	Parasitoid species	varies among species [8,12,15,23,34–37]; does not vary among strains [10]
	Parasitoid strain within species	varies among strains [8]; does not vary among strains [12,14]
	Parasitoid individual within species	varies among individuals [8]
	Parasitoid shipment from commercial supplier	varies among shipments [36]
	Number of generations parasitoid in culture	no effect [10]
	Parasitoid heterosis	positive [38]
	Parasitoid nutrition	varies among nutritive regimes [39], no effect [40]
	Parasitoid:host density ratio	positive [15,41–47], hump-shaped [48]
	Exposure time with parasitoid	positive [47]
	Number of parasitoid attacks	positive [33,49–51]
	Order of multiparasitism	varies with species order and combination [52]
	Pre-exposure of parasitoids to host adults	positive [53]
	Presence of host-attending ants	negative [37]
	Temperature	positive [17], negative [54], hump-shaped [55,56]
Immune defense costs	Density of competitors	positive [57,58]
	Increased desiccation risk	positive [59]
	Decreased nutrient availability for host	positive [59]
	Host species	does not vary among host species [58]
	Parasitoid species	varies among species (longevity) [60], does not vary among species (fecundity) [60]

*Effects on hosts may include results of host feeding in some studies where mechanisms were not distinguished.

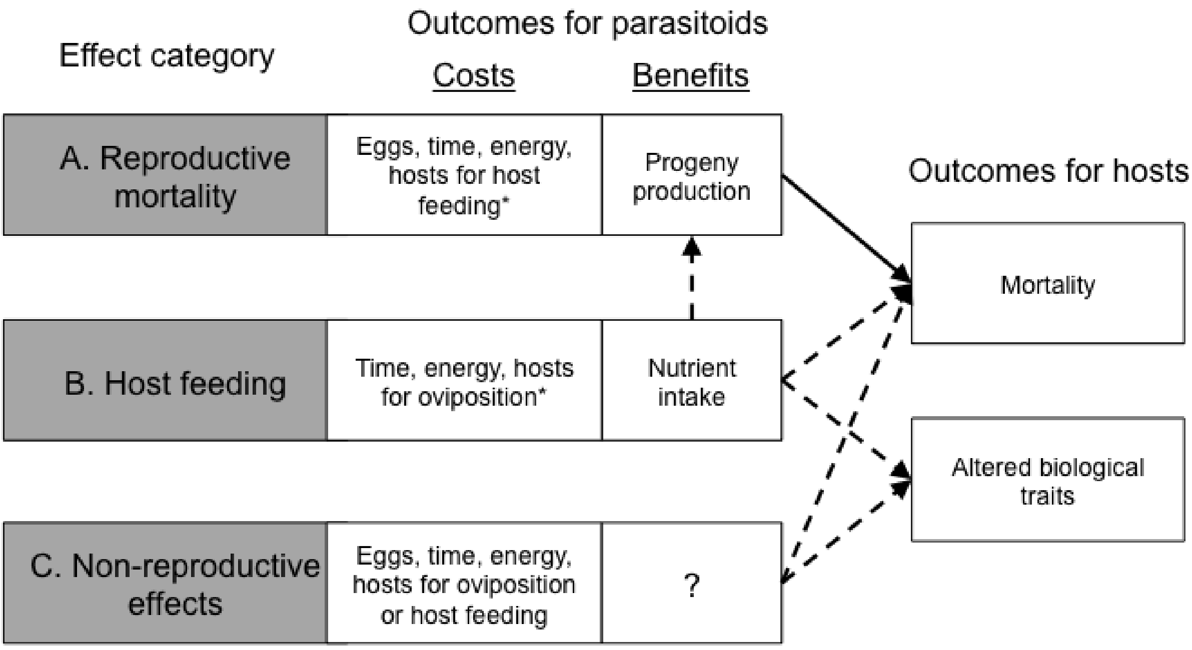

Figure 1. Categories of the three different ways that parasitoids can negatively affect host populations, with the resulting benefits and costs for parasitoids and outcomes for hosts. Solid arrows show direct relationships; dashed arrows show indirect or conditional relationships. "Altered biological traits" are listed in Fig. 2. Asterisks (*) indicate that there is no cost for parasitoid species with concurrent host feeding (i.e., that can use the same host for progeny production and host feeding).

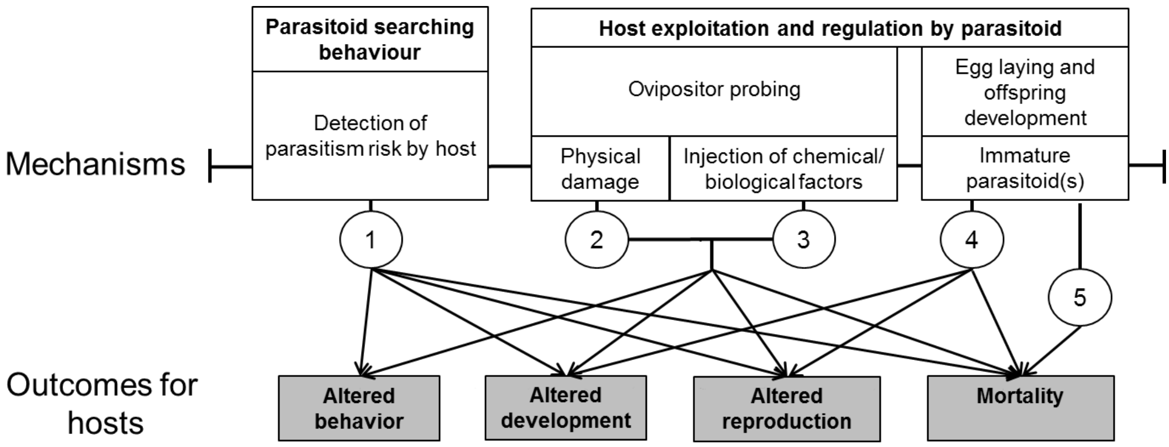

Figure 2. Mechanisms and outcomes of non-reproductive effects of parasitoids on their hosts, with suggested terminology: (1) non-consumptive effects; (2) mutilation; (3) pseudoparasitism; (4) immune defense costs; (5) aborted parasitism.

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