Supplementary Materials

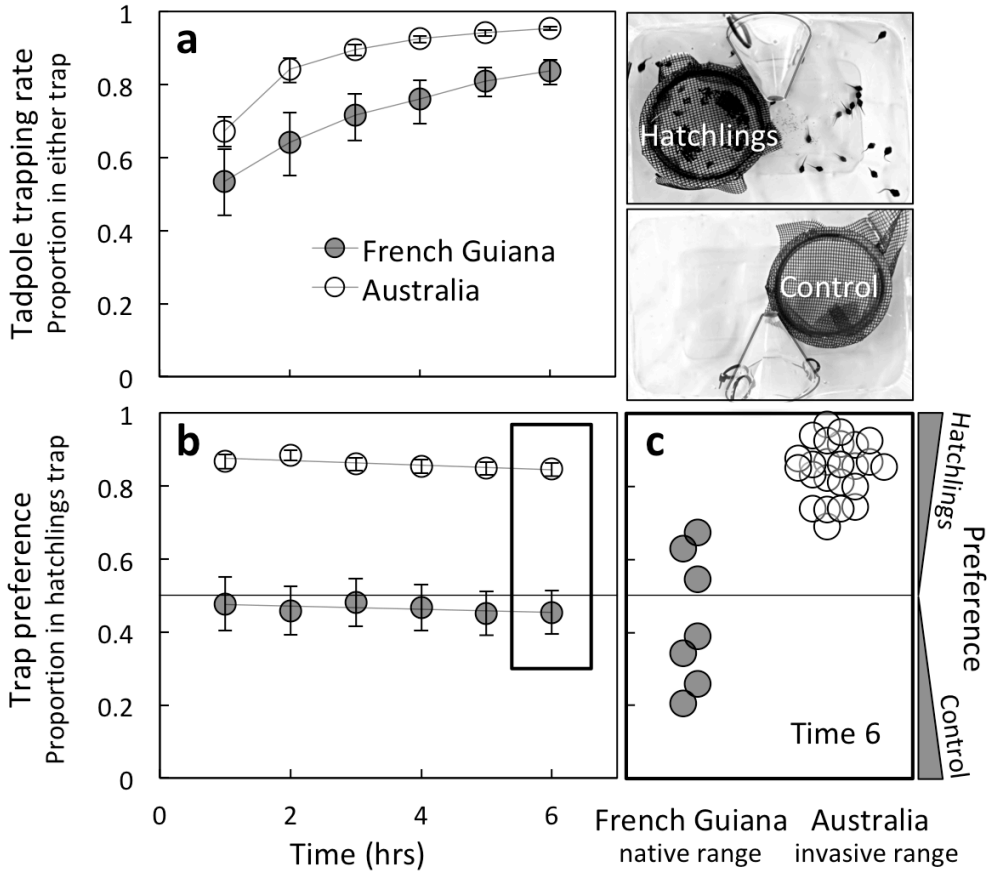

Figure S1. The effect of population on tadpole trapping rate and trap preference. In trapping trials where 50 cane toad tadpoles were given a choice between a trap containing conspecific hatchlings and an empty control trap the proportion of tadpoles trapped in either trap type (i.e., the trapping rate) was affected by an interaction between time and population (a; means ± SE, df=343, t=2.80, p=0.0054). Although the trapping rate did not initially differ between native French Guiana populations and invasive Australian populations (odds ratio at hour 1: 1.79, SE: 1.18 to 2.71, df=29, t=1.40, p=0.17), Australian tadpoles were more likely to enter a trap by the end of the trial period (odds ratio at hour 6: 3.89, SE: 2.74 to 5.52, df=29, t=3.90, p<0.0001). In contrast, the effect of population on trap preference was consistent across time (b; means ± SE), such that the odds that an Australian tadpole would select the hatchlings trap were 8.90 times those of a native range tadpole (SE: 6.35 to 12.48, df=29, t=-4.15, p<0.0001; the solid black line represents a theoretical scenario in which tadpoles are equally likely to enter either trap type). Mean data after six hours ("Time 6") for each of the 31 tadpole clutches is also depicted in panel c, where the proportion of trapped tadpoles that selected the hatchlings trap is depicted for each clutch (averaged across 1 to 6 trials per clutch), demonstrating that attraction to hatchlings was ubiquitous among the Australian clutches. The photo insets provide visual examples of these traps; here the hatchlings trap and the paired control trap are depicted one hour into a trial period. Photo credit: M. Crossland.

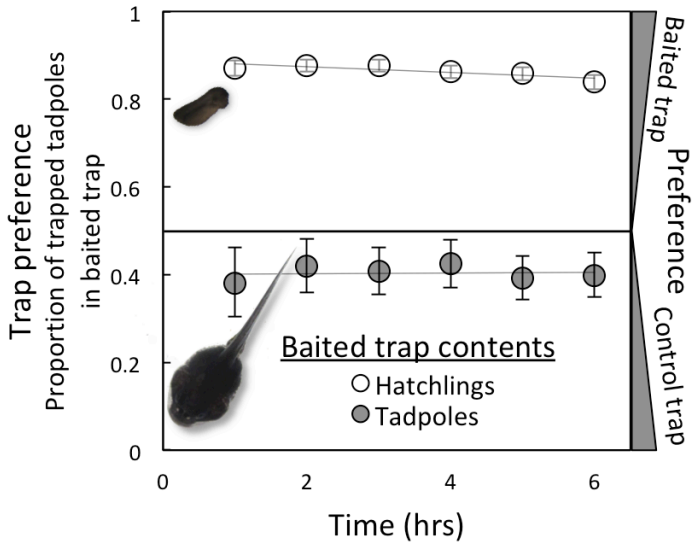

Figure S2. Cannibalistic tadpoles from invasive populations are attracted to conspecifics during the vulnerable hatchling stage, but not during the invulnerable tadpole stage.

The proportion of Australian cane toad tadpoles that entered a baited trap (vs. a paired control trap) differed depending on the life stage of the conspecific larvae used as bait. Here, the proportion of tadpoles that selected the baited trap is shown for each bait type (Hatchlings vs. Tadpoles). In 56 Hatchling trials using 24 tadpole clutches and 10 non-sibling "attractant" hatchling clutches, 300 live hatchlings (~1.0g wet mass; Gosner stage 18±1) were used as bait (see also Figure S1). In 22 Tadpole trials using 5 tadpole clutches and 7 non-sibling "attractant" tadpole clutches (in 22 combinations), 1.20 to 1.27g of live tadpoles (Gosner stage 28 to 38) were used as bait. Although Australian tadpoles were attracted to conspecifics during the vulnerable hatchling stage (Figure S1, Table S2), they were not attracted to conspecifics during the invulnerable tadpole stage (instead, tadpoles tended to avoid the trap that contained tadpoles from other clutches, Table S3). Overall, the odds that a tadpole would enter a baited trap if it were baited with conspecific hatchlings were 11.12 times those if it were baited with conspecific tadpoles (SE: 8.47 to 14.62, $p<0.0001$, N=29 clutches, Table S3). Since it is the maternally-invested bufadienolide toxins present in cane toad eggs and hatchlings that attract cannibalistic tadpoles (1), tadpole differentiation between conspecifics from edible and inedible life stages may be facilitated by the ontogenetic shifts in toxin profiles that occur during larval development (2). Means ± SE; the solid line indicates a theoretical scenario in which tadpoles are equally likely to enter a baited trap as a control trap.

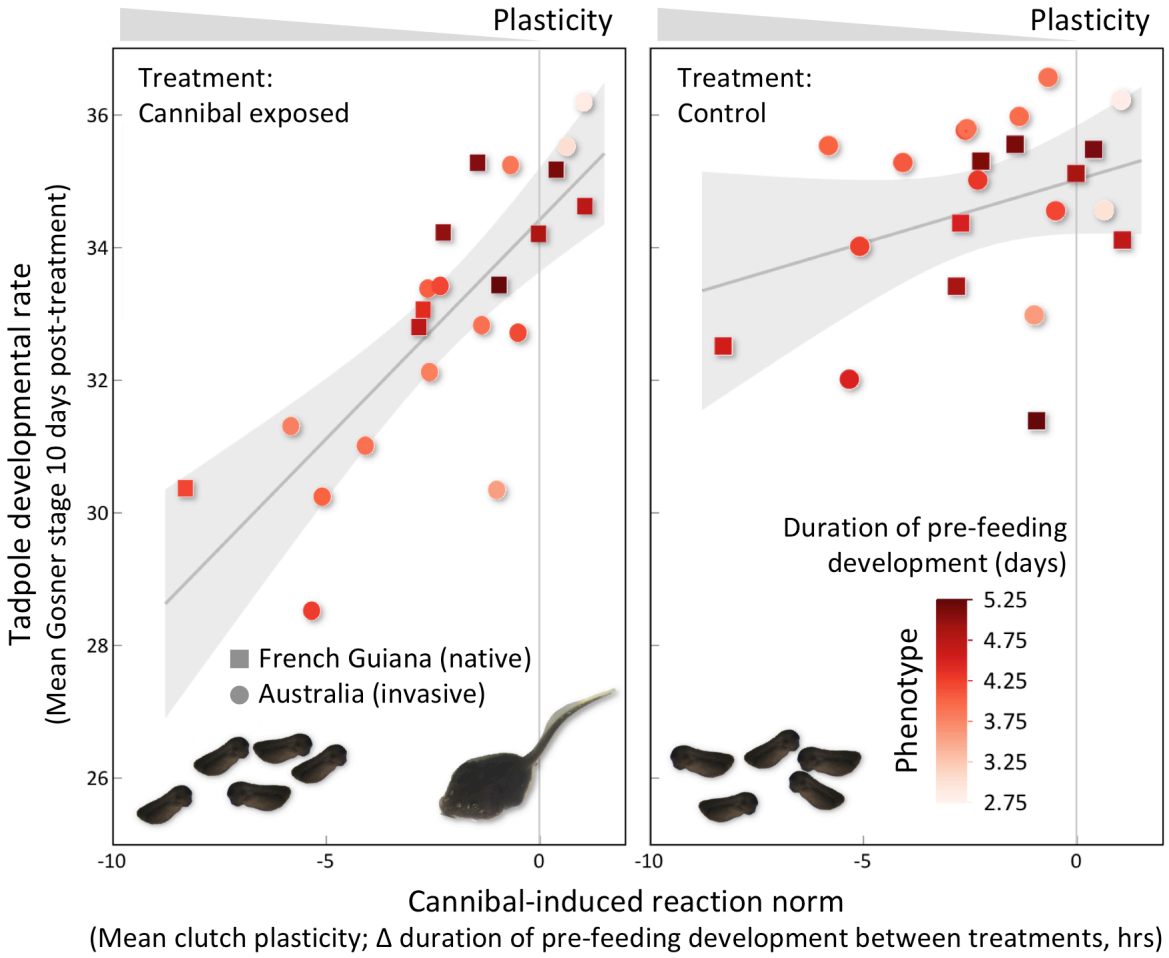

Figure S3. The relative influences of plasticity and phenotype during pre-feeding development on subsequent tadpole performance.
Plasticity, rather than phenotype, was associated with the mean rate at which tadpoles from each clutch developed following the pre-feeding treatments. For tadpoles that had been exposed to cannibal cues during the vulnerable pre-feeding stages, those from clutches that accelerated development to achieve a given pre-feeding developmental rate subsequently performed poorly relative to clutches that achieved the same developmental rate via non-plastic development (a; plasticity $p<0.0001$). Conversely, clutches that developed at substantially different rates (e.g., 3.05 vs. 5.16 days), but did not exhibit a plastic response (<1hr), performed similarly as tadpoles (phenotype $p=0.855$). This negative effect of plasticity was also significant in control conditions; tadpoles from more plastic clutches also developed more slowly in treatments where cannibals were absent (b; $p=0.045$). Here, the mean Gosner stage of the tadpoles from each clutch 10 days into the tadpole stage is related to the mean plastic response their clutch exhibited if exposed to cannibals (a value of 0 indicates that the duration of pre-feeding development did not differ between siblings from control and cannibal-exposed treatments, increasingly negative values indicate stronger inducible adaptive responses). Color variation indicates variation in the duration of pre-feeding development within the focal environment (i.e., the mean duration of the vulnerable period in cannibal-exposed [left] or control [right] treatments).

Note that these are simply the mean values for each of the 22 clutches. Unlike the final analysis, these values do not correct for variation attributable to experimental block. The line is a linear regression of these uncorrected means (±95% confidence intervals). Photo insets represent treatments.

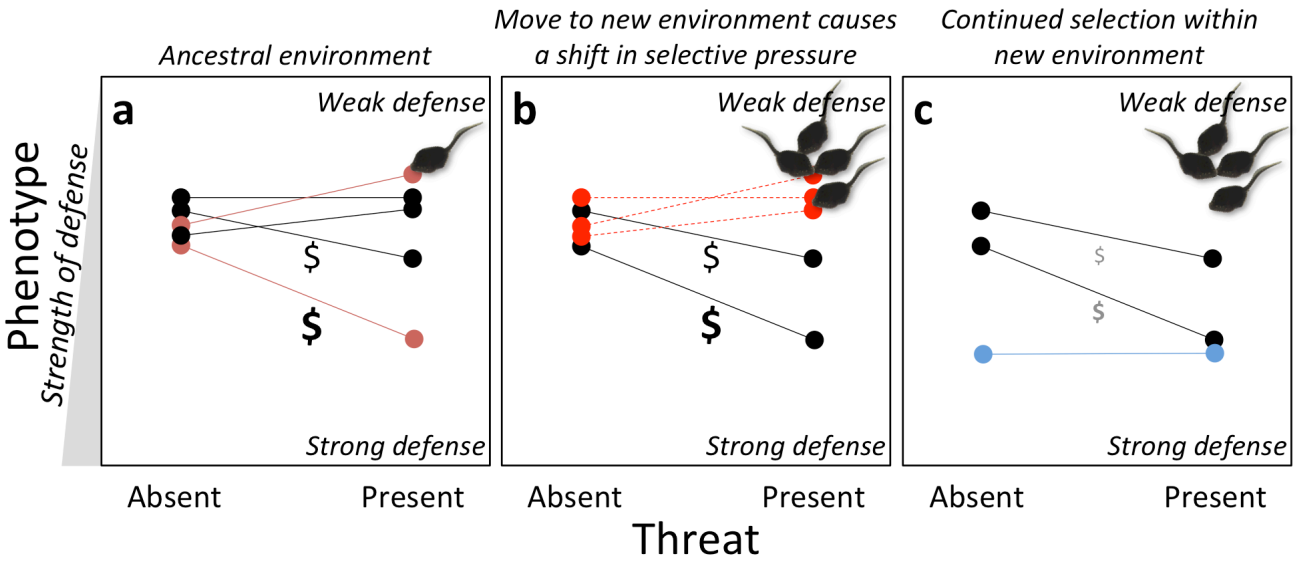

Figure S4. The influence of plasticity costs on the evolution of inducible and canalized defenses following a shift in selective pressure- theoretical figure.
Even costly inducible defenses may initially be favoured following a shift in selective pressure, as individuals that produce an adaptive response are more likely to survive the threat. However, evolutionary processes may eventually offset these costs, or costly plastic responses may be replaced by canalized, non-plastic defenses. As a result, plasticity costs may be difficult to detect for well-established plastic responses. This process is illustrated above; brown lines indicate genotypes with lower relative fitness, red lines indicate maladapted genotypes with the lowest relative fitness being eliminated under increased selective pressure, and blue lines indicate novel strategies with greater relative fitness. Dollar signs indicate that the plastic response is associated with a fitness cost (i.e., a cost of being plastic); larger signs indicate greater costs.
a) Under weak selective pressure, a variety of responses may persist. Costly inducible defenses are likely to be rare, as the costs of the defense may frequently exceed the benefits. In cane toad hatchlings, this situation is apparent in the native range, where costly cannibal-induced defenses are present, but at a low frequency.
b) If selective pressure increases, any maladaptive responses that were able to persist within the ancestral environment will be eliminated (here, by cannibalistic conspecific tadpoles). The costs of producing the inducible defense may now be lower than the costs of not responding, in which case even costly defenses will be favored if they decrease mortality risk. As a result, costly inducible defenses may become common, as they are preferable to no defense. In cane toads, the increased risk of cannibalism in the invasive Australian range has produced a similar scenario, with the majority of cane toad clutches exhibiting a costly inducible defense when exposed to cannibals.
c) Over time, as the inducible defense is frequently produced in the new environment, plasticity costs may be reduced by evolutionary processes. If these plasticity costs are reduced, increased plasticity and/or the maintenance of plasticity accompanied by further shifts toward rapid development could be favored. Alternatively, if plasticity itself is costly and/or relatively ineffective, canalized defenses may replace inducible defenses. As a

result, costly plastic responses may be ephemeral, and easiest to detect immediately following a shift in selective pressure (before plasticity costs are reduced or defenses become canalized). This process may be underway in Australia, where some clutches exhibit canalized defenses that are both more effective and less costly than the inducible defense, and plasticity is greatest in clutches with weak constitutive defenses. Under continued selective pressure, these canalized defenses may replace the inducible defense.
- Note that the costs found in this system and illustrated here are plasticity costs, which can have different effects on the evolution of plasticity than phenotypic costs, eventually resulting in a different "panel c" outcome. For example, if the production of a defended phenotype is costly, regardless of whether it was produced by plastic or non-plastic development (e.g., a cost of phenotype in which the costs of producing a defended phenotype increase with the strength of the defense), increased plasticity can be both initially favored and subsequently maintained. In this scenario, plastic genotypes would experience greater fitness than non-plastic genotypes in threat-free environments because they only incur the costs of producing a costly, well-defended phenotype if the threat is present. For example, in the example above, the genotype in blue (panel c) would have lower overall fitness than the plastic genotype that produces the same, defended phenotype in the presence of cannibals, because the blue genotype also produces the costly, defended phenotype when cannibals are absent (reducing its performance in threat-free environments). Therefore, phenotypic costs would generally favor the plastic genotype, whereas plasticity costs favor the blue, non-plastic genotype.

Table S1. Cannibalism propensity in native and invasive populations
43 tadpole clutches and 32 hatchling clutches in 114 combinations across 514 trials.

Cannibalism rates differed between the native range and Australia, such that the odds that a hatchling exposed to a conspecific tadpole would be cannibalized during an Australian trial were 2.55 times those of being cannibalized during a native range trial. The odds of being cannibalized also increased with cannibal mass (this variable was centered at 100mg). Note that the mass of each cannibal tadpole was taken, and cannibal mass is included as a covariate for each of the 514 trials. Cannibal clutch and hatchling clutch were included in this quasibinomial model as nested random effects.

Model S1	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*-0.36289*	*0.14629*	*399*	*-2.481*	*0.0135*		
Population (Australia)	0.93634	0.17023	41	5.500	**<0.0001**	2.5506	2.1514 to 3.0240
Cannibal mass (mg)	0.029984	0.001385	399	21.65	**<0.0001**	1.0304	1.0290 to 1.0319

Random effect (SD): Cannibal clutch (0.214), Egg clutch:Cannibal clutch (0.613), Residual (1.021)

```
y<-cbind(Cannibalized,Alive)
model<-glmmPQL(y~Country+CannibalMass,random=~1|CannibalClutch/HatchlingClutch, family=quasibinomial)
```

Table S2. Tadpole attraction to conspecifics during the vulnerable hatchling stage in native and invasive populations

31 tadpole clutches and 14 hatchling clutches in 69 combinations across 69 trials.

Across populations:

The effect of population on trapping rate

In trials where tadpoles were offered a choice between a trap containing 300 conspecific hatchlings and an empty control trap, there was a significant interaction between source population and time on the proportion of the 50 tadpoles that were trapped (in either trap). Trapping rates did not initially vary between native and Australian populations. However, later in the 6-hour trial period, Australian tadpoles were more likely to be trapped than were native range tadpoles (Figure S1a). Tadpole clutch and trial ID were included in this quasibinomial model as nested random effects. (Note that this analysis focused the proportion trapped; this binomial response compared the total number of tadpoles in both traps with the number of tadpoles that remained untrapped, such that trapped + untrapped = 50.)

Model S2a	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*-0.03538*	*0.53367*	*343*	*-0.0663*	*0.9472*		
Population (Australia)	0.81102	0.60145	29	1.348	0.188	2.2502	1.2332 to 4.1061
Time (hrs)	0.46794	0.03754	343	12.467	**<0.0001**	1.5967	1.5379 to 1.6578
Population * Time	0.12434	0.04443	343	2.799	**0.0054**	1.1324	1.0832 to 1.1838

Random effect (SD): Tadpole clutch (0.754), Trial ID:Tadpole clutch (1.496), Residual (1.286)

```
t<-cbind(EitherTrap,Untrapped)
model<-glmmPQL(t ~ Country*Time, random= ~1|TadpoleClutch/TrialID,family=quasibinomial)
```

The effect of population on trap preference

Trap preference (i.e., the proportion of the trapped tadpoles that selected the hatchlings trap vs. the control trap) differed between countries; Australian tadpoles were more likely than native range tadpoles to select the trap containing hatchlings. This effect of population was consistent across time (Time*Country p=0.22). However, the magnitude of the preference for the hatchlings trap decreased slightly over time (Figure S1b). Tadpole clutch and trial ID were included in this quasibinomial model as nested random effects. (Note that this analysis focused on the trapped tadpoles; this binomial response was created by comparing the number of tadpoles in the hatchlings trap with the number in the control trap. In one trial where no tadpoles had been trapped at the first time period, one tadpole was taken to be present in each trap to allow model convergence.) Attraction to the hatchling stage was apparently ubiquitous among Australian clutches, such that tadpoles from all of the Australian clutches tested preferentially entered the hatchlings trap (Figure S1c).

Model S2b	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*-0.05616*	*0.30069*	*344*	*-0.1868*	*0.8519*		
Population (Australia)	2.1866	0.3377	29	6.474	**<0.0001**	8.905	6.352 to 12.483
Time (hrs)	-0.027397	0.006599	344	-4.152	**<0.0001**	0.9729	0.9666 to 0.9794

Random effect (SD): Tadpole clutch (0.449), Trial ID:Tadpole clutch (0.862), Residual (0.517)

```
y<-cbind(HatchlingsTrap,ControlTrap)
model<-glmmPQL(y ~ Country +Time, random= ~1| TadpoleClutch/TrialID,family=quasibinomial)
```

Within populations:

The effect of the presence of hatchlings on the trap preference of tadpoles

Native range tadpoles were equally likely to enter a trap containing hatchlings as the control trap (Model S2c). In contrast, Australian tadpoles were attracted to conspecific hatchlings, such that, overall, the odds that a tadpole would enter a trap containing hatchings were 29.46 times those of entering a control trap (Model S2d). Here, the proportion of the 50 tadpoles within each trap is analyzed as a response to treatment (i.e., control vs. hatchlings). The nested random effects of tadpole clutch, trial ID, and trap ID were also included in the quasibinomial model.

French Guiana trap preference (quasi-binomial)

Model S2c	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*-0.62694*	*0.29476*	*130*	*-2.127*	*0.0353*		
Treatment (Hatchlings)	-0.19968	0.38061	12	-0.525	0.6094	0.8190	0.5597 to 1.1983

Random effect (SD): Tadpole clutch (1.112E-4), Trial ID:Tadpole clutch (0.432), Trap:Trial ID:Tadpole Clutch (0.948), Residual (1.192)

```
f<-cbind(InFocalTrap,NotInFocalTrap)
model<-glmmPQL(f ~ Treatment, random= ~1| TadpoleClutch/TrialID/Trap,family=quasibinomial)
```

Australia trap preference (quasi-binomial)

Model S2d	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*-2.1293*	*0.1255*	*560*	*-16.97*	*<0.0001*		
Treatment (Hatchlings)	3.3832	0.1756	55	19.26	**<0.0001**	29.464	24.718 to 35.122

Random effect (SD): Tadpole clutch (2.297E-5), Trial ID:Tadpole clutch (5.572E-5), Trap:Trial ID:Tadpole Clutch (0.891), Residual (1.393)

```
a<-cbind(InFocalTrap,NotInFocalTrap)
model<-glmmPQL(a~Treatment, random=~1|TadpoleClutch/TrialID/Trap, family=quasibinomial)
```

Table S3. Tadpole attraction to conspecifics during the invulnerable tadpole stage (vs. the vulnerable hatchling stage) in invasive populations
5 tadpole clutches and 7 non-sibling "attractant" tadpole clutches in 22 combinations across 22 trials.

When Australian tadpoles were offered a choice between a trap containing non-sibling conspecific tadpoles and an empty control trap, their response differed from that demonstrated for non-sibling conspecific hatchlings (Figure S2). Overall, there was a marginally significant interaction between trap type (control vs. tadpoles) and time (Model S3a); tadpoles exhibited a tendency to preferentially enter the control trap that was marginally significant 1hr into the trapping period (df=25, t=1.95, p=0.0628), but was significant later in the trapping period (e.g., 6hrs OR: 1.76, SE: 1.41 to 2.19, df=25, t=2.58, p=0.0160). Therefore, whereas Australian tadpoles were strongly attracted to vulnerable conspecific hatchlings (Model S2c), they tended to avoid invulnerable conspecific tadpoles.

Australia (Attraction to conspecifics during the invulnerable tadpole stage; quasi-binomial)

Model S3a	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	-3.3227	0.2474	258	-13.433	<0.0001		
Treatment (Tadpoles)	-0.29907	0.27387	25	-1.092	0.2852	0.7415	0.5639 to 0.9751
Time (hrs)	0.33302	0.01636	258	20.352	**<0.0001**	1.3952	1.3725 to 1.4182
Treatment*Time	-0.04665	0.02511	258	-1.858	0.0644	0.9544	0.9308 to 0.9787

Random effect (SD): Tadpole clutch (0.345), Trial ID:Tadpole clutch (1.105E-4), Trap:Trial ID:Tadpole Clutch (0.888), Residual (0.774)
at<-cbind(InFocalTrap,NotInFocalTrap)
*model<-glmmPQL(at~Treatment*Time, random=~1|TadpoleClutch/TrialID/Trap, family=quasibinomial)*

Effect of bait type (hatchlings vs. tadpoles) on trapping rate and trap preference in Australia
Both trapping rate (Model S2f) and trap preference (Model S2g) were significantly affected by the life stage of the tadpoles used as bait. Tadpoles were trapped (in either trap type) more quickly during trials in which hatchlings were used as bait than those in which tadpoles were used, but the magnitude of this difference varied across time (e.g., 1hr into the trapping period, the relative odds of being trapped during a hatchling trial were 18.0 times those during a tadpole trial [SE: 12.9 to 25.0, df=27, t=8.77, p<0.0001], whereas the odds ratio increased to 29.5 times 6hrs into the trapping period [SE: 21.3 to 40.7, df=27, t=10.47, p<0.0001].) Trapped tadpoles were also more likely to select the baited trap during hatchling trials than during tadpole trials, such that, overall, the odds of selecting the baited trap if it contained hatchlings were 11.1 times those if it contained tadpoles (SE: 8.5 to 14.6). There was no significant interaction between bait type and time on the proportion of tadpoles that selected the baited trap (p=0.36).

Effect of bait type on trapping rate in Australia (quasi-binomial)

Model S3b	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*0.69102*	*0.25095*	*388*	*2.753*	*0.0062*		
Bait type (Tadpoles)	-3.3381	0.5275	27	-6.328	**<0.0001**	0.03550	0.02095 to 0.06017
Time (hrs)	0.58980	0.02299	388	25.655	**<0.0001**	1.8036	1.7626 to 1.8456
Bait type * Time	-0.21609	0.03312	388	-6.523	**<0.0001**	0.8057	0.7794 to 0.8328

Random effect (SD): Tadpole clutch (0.864), Trial ID:Tadpole clutch (1.069), Residual (1.249)
t<-cbind(InEitherTrap,NotTrapped)
*model<-glmmPQL(t~Treatment*Time, random=~1|TadpoleClutch/TrialID, family=quasibinomial)*

Effect of bait type on trap preference in Australia (quasi-binomial)

Model S3c	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
Intercept	*2.1309*	*0.1443*	*389*	*14.773*	*<0.0001*		
Bait type (Tadpoles)	-2.4092	0.2730	27	-8.826	**<0.0001**	0.08989	0.06841 to 0.11810
Time (hrs)	-0.02934	0.00745	389	-3.940	**0.0001**	0.9711	0.9639 to 0.9783

Random effect (SD): Tadpole clutch (0.223), Trial ID:Tadpole clutch (0.968), Residual (0.544)

```
y<-cbind(InFocalTrap,NotInFocalTrap)
model<-glmmPQL(y~Treatment+Time, random=~1|TadpoleClutch/TrialID, family=quasibinomial)
```

Table S4. Effects of population and exposure to cannibal cues on rates of pre-feeding development

23 egg/hatchling clutches split between 189 exposure and 109 control tanks (5 eggs/tank). Trials conducted over 8 temporal blocks.

The effect of exposure to cannibal cues on the duration of pre-feeding development varied between the native range and Australia. In the native range, exposure to cannibal cues did not significantly affect the rate of pre-feeding development (though hatchlings tended to accelerate development in exposed treatments; Model S4b, p=0.06). However, in the invasive Australian range, exposure to cannibal cues induced developmental acceleration (Model S4c). Mean development time within each tank was calculated by averaging the development time of all of the individuals within the tank that successfully reached the tadpole stage, then was modeled as a response to source population (i.e., country), treatment, and their interaction. Experimental block and clutch ID were included in all models as nested random effects.

Model S4a	Value	Std.Error	DF	t-value	p-value
(Intercept)	*115.328*	*5.558*	*6.040*	*20.75*	*7.61E-07*
Treatment (Exposed)	-1.241	0.517	271.6	-2.40	0.017
Country (Australia)	-23.184	7.811	5.900	-2.97	0.0255
Treatment*Country	-1.096	0.633	271.4	-1.73	0.0846

Random effect (variance, SD): Block (111.688, 10.568), Clutch:Block (22.832, 4.778), Residual (5.724, 2.392)

*model<-lmer((TankMeanDevelopTimeHrs)~Treatment*Country+(1|Block/ClutchID))*

<u>Within populations (treatment effects):</u>

French Guiana cannibal-induced plasticity (hrs)

Model S4b	Value	Std.Error	DF	t-value	p-value
(Intercept)	*115.487*	*4.103*	*2.942*	*28.15*	*0.000113*
Treatment (Exposed)	-1.226	0.638	108.4	-1.922	0.0573

Random effect (variance, SD): Block (56.263, 7.501), Clutch:Block (20.655, 4.545), Residual (8.721, 2.953)

model<-lmer((FgTankMeanDevelopTimeHrs)~Treatment+(1|Block/ClutchID))

Australia cannibal-induced plasticity (hrs)

Model S4c	Value	Std.Error	DF	t-value	p-value
(Intercept)	*92.102*	*6.611*	*2.932*	*13.93*	*0.000901*
Treatment (Exposed)	-2.341	0.295	163.0	-7.927	**3.35E-13**

Random effect (variance, SD): Block (165.586, 12.868), Clutch:Block (24.264, 4.926), Residual (3.739, 1.934)

model<-lmer((AusTankMeanDevelopTimeHrs)~Treatment+(1|Block/ClutchID))

<u>Within treatments (population effect):</u>

Clutches from the native range developed more slowly during the pre-feeding stages than invasive range clutches in both control (Model S4d) and exposed (Model S4e) treatments. Experimental block and clutch ID were included in the models as nested random effects.

Control development time (hrs)

Model S4d	Value	Std.Error	DF	t-value	p-value
(Intercept)	*115.746*	*5.755*	*6.008*	*20.113*	*9.67E-07*
Country (Australia)	-23.784	8.093	5.884	-2.939	**0.0266**

Random effect (variance, SD): Block (120.61, 10.982), Clutch:Block (23.42, 4.84), Residual (2.86, 1.691)

model<-lmer((ContTankMeanDevelopTimeHrs)~Country+(1|Block/ClutchID))

Exposed development time (hrs)

Model S4e	Value	Std.Error	DF	t-value	p-value
(Intercept)	*113.798*	*5.412*	*5.986*	*21.028*	*7.72E-07*
Country (Australia)	-23.828	7.609	5.859	-3.132	**0.0209**

Random effect (variance, SD): Block (105.553, 10.274), Clutch:Block (23.118, 4.808), Residual (6.111, 2.472)

model<-lmer((ExpTankMeanDevelopTimeHrs)~Country+(1|Block/ClutchID))

Table S5. Carry-over effects of exposure to cannibal cues on tadpole performance in native and invasive populations

1,190 individually raised tadpoles from 22 clutches sourced from 278 of the egg/hatchling exposure tanks in which the duration of pre-feeding development was measured. Clutches raised within 7 temporal blocks.

Do the carry-over effects that follow exposure to cannibal cues differ between populations? In Australia, exposure to cannibal cues during the pre-feeding stages significantly reduced subsequent rates of development and growth during the tadpole stage. However, in the native range, exposure to conspecific cues did not significantly affect subsequent performance. Performance measures were calculated as the mean value for all of the individually raised tadpoles sourced from a given egg/hatchling tank. These mean values were then modeled as a response to the fixed effects of treatment, source population (i.e., country), and their interaction. Experimental block and clutch ID were included in the models as nested random effects. Survival was similarly modeled, but as a binomial response using a quasibinomial model. Survival was not significantly affected by treatment, source population, or their interaction (country* treatment p=0.456), though it tended to be lower in Australian tadpoles (p=0.086; Model S5g).

Development (Gosner stage)

Model S5a	Value	Std.Error	DF	t-value	p-value
(Intercept)	*34.1439*	*0.9795*	*4.8817*	*34.859*	*4.83E-07*
Treatment (Exposed)	-0.2578	0.2676	256.55	-0.963	0.336
Country (Australia)	0.6073	1.2912	4.8008	0.47	0.659
Treatment*Country	-2.0788	0.3198	255.8122	-6.501	**4.15E-10**

Random effect (variance, SD): Block (2.367, 1.538), Clutch:Block (1.067, 1.033), Residual (1.314, 1.146)

*model<-lmer(MeanTankStage~Treatment*Country+(1|Block/ClutchID))*

French Guiana

Model S5b	Value	Std.Error	DF	t-value	p-value
(Intercept)	*34.1256*	*0.611*	*2.1775*	*55.856*	*0.000177*
Treatment (Exposed)	-0.2693	0.1765	91.2712	-1.525	0.1306

Random effect (variance, SD): Block (0.6295, 0.7934), Clutch:Block (1.2675, 1.1258), Residual (0.5699, 0.7549)

model<-lmer(FgMeanTankStage~Treatment+(1|Block/ClutchID))

Australia

Model S5c	Value	Std.Error	DF	t-value	p-value
(Intercept)	*34.7425*	*1.0211*	*2.877*	*34.03*	*7.72E-05*
Treatment (Exposed)	-2.3372	0.2007	163.3	-11.65	**< 2e-16**

Random effect (variance, SD): Block (3.7175, 1.9281), Clutch:Block (0.9619, 0.9808), Residual (1.7286, 1.3147)

model<-lmer(AusMeanTankStage~Treatment+(1|Block/ClutchID))

Growth (mass, mg)

Model S5d	Value	Std.Error	DF	t-value	p-value
(Intercept)	*118.9*	*14.305*	*4.753*	*8.312*	*0.000524*
Treatment (Exposed)	2.246	5.141	256.34	0.437	0.6626
Country (Australia)	8.198	18.79	4.57	0.436	0.6825
Treatment*Country	-56.067	6.142	255.63	-9.128	**< 2e-16**

Random effect (variance, SD): Block (412.2, 20.3), Clutch:Block (432.3, 20.79), Residual (484.8, 22.02)

*model<-lmer(MeanTankMass~Treatment*Country+(1|Block/ClutchID))*

French Guiana

Model S5e	Value	Std.Error	DF	t-value	p-value
(Intercept)	*118.592*	*10.163*	*2.46*	*11.669*	*0.00325*
Treatment (Exposed)	2.056	4.523	91.415	0.454	0.65056

Random effect (variance, SD): Block (43.63, 6.605), Clutch:Block (665.08, 25.789), Residual (374.47, 19.351)

model<-lmer(FgMeanTankMass~Treatment+(1|Block/ClutchID))

Australia

Model S5f	Value	Std.Error	DF	t-value	p-value
(Intercept)	*126.532*	*14.9*	*2.835*	*8.492*	*0.00424*
Treatment (Exposed)	-53.816	3.566	163.44	-15.093	**< 2e-16**

Random effect (variance, SD): Block (754.4, 27.47), Clutch:Block (280, 16.73), Residual (545.7, 23.36)

model<-lmer(AusMeanTankMass~Treatment+(1|Block/ClutchID))

Survival (quasi-binomial)

Model S5g	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
(Intercept)	*3.892*	*0.441*	*255*	*8.818*	*0*		
Treatment (Exposed)	-0.3788	0.2578	255	-1.469	0.143	0.6847	0.5291 to 0.8860
Country (Australia)	-0.9837	0.4610	5	-2.134	0.086	0.3739	0.2358 to 0.5929

Random effect (SD): Block (9.71E-5), Clutch:Block (0.6863), Residual (0.9494)

y<-cbind(TankAlive,TankDead)

model<-glmmPQL(y~ Treatment+Country, random= ~1|Block/ClutchID,family=quasibinomial)

French Guiana

Model S5h	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
(Intercept)	*3.5361*	*0.5990*	*91*	*5.903*	*0*		
Treatment (Exposed)	0.01206	0.70237	91	0.01718	0.9863	1.0121	0.5014 to 2.0430

Random effect (SD): Block (6.92E-9), Clutch:Block (3.265E-4), Residual (1.0126)

f<-cbind(FgTankAlive,FgTankDead)

model<-glmmPQL(f~ Treatment, random= ~1|Block/ClutchID,family=quasibinomial)

Australia

Model S5i	Value	Std.Error	DF	t-value	p-value	Odds ratio	SE of odds ratio
(Intercept)	*2.9635*	*0.3052*	*163*	*9.709*	*0*		
Treatment (Exposed)	-0.4512	0.2777	163	-1.625	0.1061	0.6369	0.4825 to 0.8407

Random effect (SD): Block (9.55E-5), Clutch:Block (0.7222), Residual (0.9493)

a<-cbind(AusTankAlive,AusTankDead)

model<-glmmPQL(a~ Treatment, random= ~1|Block/ClutchID,family=quasibinomial)

Table S6. Phenotypic vs. plasticity costs

Mean clutch values per treatment for tadpoles from 22 clutches that were either exposed or naïve to cannibal cues during pre-feeding development. Trials conducted over 8 temporal blocks.

Associations between performance during the tadpole stage and plasticity and phenotype during pre-feeding development; across clutches, greater plasticity was associated with poorer performance. Here, plasticity is the mean reduction in pre-feeding development time induced by exposure to conspecific cues (-hrs) and phenotype is the total duration of pre-feeding development in the focal treatment (hrs). Note that an inducible reduction in development time was given a positive plasticity value. Experimental block was included as a random effect. A significant negative coefficient for the plasticity term indicates that the ability to accelerate development is costly within the focal environment, such that stronger inducible reductions in pre-feeding development time were associated with poorer tadpole performance. A significant coefficient for phenotype would indicate that it is the rate of development through the pre-feeding stages that affects subsequent performance; here, a positive coefficient would indicate that rapid pre-feeding development was associated with poor tadpole performance. A significant interaction between plasticity and phenotype could indicate that production costs are greater for more plastic phenotypes; however, this interaction term was never significant ($p>0.1$ in all cases) and was removed from the final models. In addition, adding population (i.e., native vs. invasive) to these models as a fixed effect did not improve model fit, nor was the effect of population significant ($p>0.15$ in all cases). Population was therefore removed from the final models. Note that, in many systems, strong correlations between trait plasticities and trait values can bias estimates of plasticity costs (for example, if an extreme, predator-induced morph is never produced by non-plastic genotypes (3, 4)). However, phenotype and plasticity were not significantly correlated in our dataset (Pearson's correlation tests: control $r=0.149$, $N=23$, $p=0.499$; exposed $r=-0.004$, $N=23$, $p=0.986$), such that the performance of plastic and non-plastic clutches could be compared when both produced the same phenotype. This allowed us to separate costs of plasticity from costs of phenotype. Mean values from each egg/hatchling exposure tank were averaged to calculate the mean clutch values.

Treatment: Exposed

Response: Development (Gosner stage)

Model S6a	Value	Std.Error	DF	t-value	p-value
Intercept	*33.54*	*2.77*	*13.265*	*12.099*	*1.52E-08*
Phenotype	0.004954	0.026557	13.779	0.187	0.855
Plasticity	-0.5298	0.0775	14.844	-6.836	**5.96E-06**

Random effect (variance, SD): Block (2.0696, 1.4386), Residual (0.5463, 0.7391)

model<-lmer(StageExposed~ DevelopTimeExposedHrs+PlasticityHrs+(1|Block))

Response: Growth (mass, mg)

Model S6b	Value	Std.Error	DF	t-value	p-value
Intercept	*21.43*	*45.73*	*9.059*	*0.469*	*0.6503*
Phenotype	0.8807	0.4401	9.254	2.001	0.0755
Plasticity	-8.942	1.570	15.36	-5.696	**3.87E-05**

Random effect (variance, SD): Block (395.7, 19.89), Residual (234.2, 15.3)

model<-lmer(MassExposed~ DevelopTimeExposedHrs+PlasticityHrs+(1|Block))

Response: Survival (quasi-binomial)

Model S6c	Value	Std.Error	DF	t-value	p-value
Intercept	*0.5495*	*1.3437*	*13*	*0.409*	*0.6892*
Phenotype	0.02389	0.01373	13	1.739	0.1056
Plasticity	-0.1212	0.0803	13	-1.509	0.1552

Random effect (SD): Block (2.01E-5), Residual (1.208)

e<-cbind(ExposedAlive,ExposedDead)

model<-glmmPQL(e ~ DevelopTimeExposedHrs+PlasticityHrs, random=~1|Block, family=quasibinomial)

<u>Treatment: Control</u>

Response: Development (Gosner stage)

Model S6d	Value	Std.Error	DF	t-value	p-value
Intercept	*38.68*	*2.54*	*10.83*	*15.21*	*1.19E-08*
Phenotype	-0.03833	0.02441	11.18	-1.57	0.1442
Plasticity	-0.17135	0.07814	14.47	-2.193	**0.0451**

Random effect (variance, SD): Block (1.4965, 1.223), Residual (0.546, 0.739)

model<-lmer(StageControl~ DevelopTimeControlHrs+PlasticityHrs+(1|Block))

Response: Growth (mass, mg)

Model S6e	Value	Std.Error	DF	t-value	p-value
Intercept	*126.58*	*56.73*	*9.293*	*2.231*	*0.0517*
Phenotype	0.01913	0.54611	9.441	0.035	0.9728
Plasticity	-3.715	2.077	15.95	-1.788	0.0928

Random effect (variance, SD): Block (564.3, 23.76), Residual (399.9, 20)

model<-lmer(MassControl~ DevelopTimeControlHrs+PlasticityHrs+(1|Block))

Response: Survival (quasi-binomial)

Model S6f	Value	Std.Error	DF	t-value	p-value
Intercept	*0.5246*	*2.0037*	*13*	*0.262*	*0.7976*
Phenotype	0.02565	0.02214	13	1.158	0.2676
Plasticity	-0.01665	0.14421	13	-0.115	0.9099

Random effect (SD): Block (2.61E-5), Residual (1.203)

c<-cbind(ControlAlive,ControlDead)

model<-glmmPQL(c ~ DevelopTimeControlHrs+PlasticityHrs, random=~1|Block, family=quasibinomial)

Table S7. Association between developmental rates and plasticity
Mean clutch values per treatment for tadpoles from 23 clutches that were exposed or naïve to cannibal cues during pre-feeding development.

There was a quadratic relationship between the rate of pre-feeding development and the magnitude of the plastic response induced by exposure to cannibal cues, such that clutches with intermediate phenotypes were the most plastic. This relationship was significant in both control and exposed conditions. Country was not a significant predictor in either quadratic model (p>0.5), and was removed from the final analyses. However, this curve apparently occurs because the relationship between phenotype and plasticity differed between the native and invasive range (Models S7c and S7f); within the native range, rapidly developing clutches were the most plastic, whereas the opposite relationship was found within the invasive range. Within a given country the quadratic term was not significant. Plasticity was calculated for each clutch as the mean difference in development time between control and exposed treatments- greater inducible reductions in development time indicate greater adaptive plastic responses. Development time was calculated for each clutch as the mean duration of pre-feeding development in control or exposed conditions.

Quadratic relationships between phenotype and plasticity across populations
Treatment: Control

Model S7a	Value	Std.Error	t-value	p-value
(Intercept)	*-40.156*	*14.687*	*-2.734*	*0.0128*
Phenotype (hrs)	0.8439	0.2968	2.844	**0.0100**
Phenotype^2	-0.00412	0.00148	-2.779	**0.0116**

Residual SE: 2.083 on 20 DF, $F_{2,20}$=4.176, p=0.0305
model<-lm(PlasticityHrs~DevelopTimeControlHrs+QuadraticDevelopTimeControlHrs)

Treatment: Exposed

Model S7b	Value	Std.Error	t-value	p-value
(Intercept)	*-45.756*	*15.243*	*-3.002*	*0.0071*
Phenotype (hrs)	0.9801	0.3070	3.192	**0.0046**
Phenotype^2	-0.0049	0.0015	-3.208	**0.0044**

Residual SE: 2.015 on 20 DF, $F_{2,20}$=5.146, p=0.0157
model<-lm(PlasticityHrs~DevelopTimeExposedHrs+QuadraticDevelopTimeExposedHrs)

Differences between countries in the phenotype/plasticity association
Treatment: Control

Model S7c	Value	Std.Error	t-value	p-value
(Intercept)	*21.286*	*11.229*	*1.896*	*0.0733*
DevelopmentTimeControl (hrs)	-0.1645	0.0957	-1.719	0.1018
Country (Australia)	-32.00	12.19	-2.626	**0.0166**
DevelopmentTimeControl*Country	0.3029	0.1080	2.806	**0.0113**

Residual SE: 2.034 on 19 DF, $F_{3,19}$=3.576, p=0.0333
*model<-lm((PlasticityHrs)~DevelopTimeControlHrs*Country)*

French Guiana

Model S7d	Value	Std.Error	t-value	p-value
(Intercept)	*21.286*	*13.755*	*1.548*	*0.1600*
DevelopmentTimeControl (hrs)	-0.1645	0.1172	-1.404	0.1980

Residual SE: 2.491 on 8 DF, $F_{1,8}$=1.97, p=0.198
model<-lm(FGPlasticityHrs~FGDevelopTimeControlHrs)

Australia

Model S7e	Value	Std.Error	t-value	p-value
(Intercept)	*-10.719*	*3.775*	*-2.839*	*0.0161*
DevelopmentTimeControl (hrs)	0.1383	0.0399	3.471	**0.0052**

Residual SE: 1.622 on 11 DF, $F_{1,11}$=12.05, p=0.0052

model<-lm(AusPlasticityHrs~AusDevelopTimeControlHrs)

Treatment: Exposed

Model S7f	Value	Std.Error	t-value	p-value
(Intercept)	*25.480*	*8.724*	*2.921*	*0.00877*
DevelopmentTimeExposed (hrs)	-0.2039	0.0756	-2.697	**0.0143**
Country (Australia)	-35.492	10.084	-3.520	**0.0023**
DevelopmentTimeExposed*Country	0.338	0.0934	3.619	**0.0018**

Residual SE: 1.949 on 19 DF, $F_{3,19}$=4.459, p=0.0156

*model<-lm((PlasticityHrs)~DevelopTimeExposedHrs*Country)*

French Guiana

Model S7g	Value	Std.Error	t-value	p-value
(Intercept)	*25.480*	*9.263*	*2.751*	*0.0250*
DevelopmentTimeExposed (hrs)	-0.2039	0.0803	-2.540	**0.0347**

Residual SE: 2.069 on 8 DF, $F_{1,8}$=6.452, p=0.0347

model<-lm(FGPlasticityHrs~FGDevelopTimeExposedHrs)

Australia

Model S7h	Value	Std.Error	t-value	p-value
(Intercept)	*-10.0124*	*4.8185*	*-2.078*	*0.0619*
DevelopmentTimeControl (hrs)	0.1341	0.0522	2.568	**0.0261**

Residual SE: 1.856 on 11 DF, $F_{1,11}$=6.595, p=0.0261

model<-lm(AusPlasticityHrs~AusDevelopTimeExposedHrs)

Table S8. Is plasticity an effective strategy for reducing the period of vulnerability?
Mean clutch values per treatment for tadpoles from 23 clutches that were exposed or naïve to cannibal cues during pre-feeding development.

To determine whether plasticity is an effective strategy for reducing the duration of the vulnerable period, we related the mean plasticity of each clutch to the mean phenotype it produced during cannibal exposure. We found that, in the native range, the most plastic phenotypes also developed the fastest when cannibals were present. In contrast, increasingly plastic clutches had slower development times in Australia. Therefore, although inducible developmental acceleration may reduce the duration of the vulnerable period for a given clutch, within the invasive range clutches that utilize inducible defenses may be more vulnerable than clutches that exhibit non-plastic, rapid development. Phenotype is taken as the mean total duration of the vulnerable, pre-feeding period where cannibals were present (hrs), and plasticity is the mean reduction in the duration of this period induced by cannibal cue exposure (hrs). See also Models 6f-6h.

Across populations

Model S8a	Value	Std.Error	t-value	p-value
(Intercept)	*119.48*	*3.174*	*37.643*	*< 2e-16*
Plasticity (hrs)	-2.189	0.993	-2.206	**0.0399**
Country (Australia)	-34.115	4.477	-7.62	**3.43E-07**
Plasticity*Country	4.985	1.411	3.533	**0.00222**

Residual SE: 7.808 on 19 DF, $F_{3,19}$=21, p=2.97E-6
*model<-lm(DevelopTimeExposedHrs~PlasticityHrs*Country)*

French Guiana

Model S8b	Value	Std.Error	t-value	p-value
(Intercept)	*119.485*	*2.756*	*43.35*	*8.84E-11*
Plasticity (hrs)	-2.189	0.862	-2.54	**0.0347**

Residual SE: 6.78 on 8 DF, $F_{1,8}$=6.452, p=0.0347
model<-lm(FGDevelopTimeExposedHrs~FGPlasticityHrs)

Australia

Model S8c	Value	Std.Error	t-value	p-value
(Intercept)	*85.37*	*3.428*	*24.904*	*5.03E-11*
Plasticity (hrs)	2.796	1.089	2.568	**0.0261**

Residual SE: 8.478 on 11 DF, $F_{1,11}$=6.595, p=0.0261
model<-lm(AusDevelopTimeExposedHrs~AusPlasticityHrs)

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