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HERITABILITIES AND CORRELATIONS OF THE HONEY BEE : RESPONSE TO *NOSEMA APIS*, LONGEVITY, AND ALARM RESPONSE TO ISOPENTYL ACETATE

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SUMMARY

Heritabilities, phenotypic correlations, and genotypic correlations for honey bee response to *Nosema apis*, longevity, and three alarm responses to isopentyl acetate are presented. These measurements indicate that both resistance to *N. apis* and longevity can be improved by selective breeding without producing bees unacceptably quick to defend their colonies providing the selection and breeding programs consider effects of dominance and epistasis.

INTRODUCTION

Honey bee breeding technology is considerably less developed than the breeding technologies used to improve other agriculturally important animals. Currently, most bee breeding programs rely exclusively on the evaluation of field performance of colonies. Such procedures are not entirely desirable since most characteristics of economic interest are strongly influenced by environment in field settings. Thus, genetic differences are difficult to ascertain and selections are imprecise. Also, little is known of the genetics of most economically important characteristics or the genetic and phenotypic relationships between these characteristics.

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In light of these problems, a long range program has been proposed for the development of technology used to breed stocks of bees (RINDERER, 1977 a). This proposal is aimed at the design of selection-index approaches to selecting breeding stock, the development of laboratory procedures which will more precisely assess genotype, and the acquisition of basic genetic information concerning the heritabilities of characteristics and the genetic and phenotypic correlations between these characteristics.

As a contribution to these general goals, this study was undertaken to determine the heritabilities of and relationships between five characteristics of the southern Louisianan population of honey bees as assessed through laboratory measurement. These characteristics were longevity, longevity after infection with the parasitic microsporidian *Nosema apis* (response to Nosema), and three measures of response to isopentyl acetate (IPA), a major component of honey bee alarm pheromone.

MATERIALS AND METHODS

Approximately 300 colonies from the free-mating population of honey bees in southern Louisiana were used to develop a collection of super-sibling ($r = 0.75$; coefficient of relationship) and half-sibling ($r = 0.25$) families according to the sibling analysis format developed by RINDERER (1977 b). Eight free-mated queens were randomly selected from the 300 colonies to serve as sire-queens (drone mothers). Virgin queens were reared from 40 randomly selected colonies and divided into 8 groups of 5 queens. In each group, single drones from 1 of the 8 sire-queens were used to inseminate each virgin queen. Characteristics were measured in the worker progeny of the resulting colonies.

Worker brood from each colony was emerged separately in an incubator. Thirty newly emerged bees were caged in small glass-fronted wooden cages with comb, sugar syrup and water (KULINČEVIĆ *et al.* 1973). Response to *N. apis* and longevity were measured by counting the days until 15 bees had died (RINDERER and SYLVESTER 1978).

Separate cages were used to measure response to IPA. Response to IPA was quantified as the initial activity level of the bees prior to exposure to the pheromone (no. of bees moving about the cage), the time until a general response was seen following presentations of IPA in the cage, and the duration of the response (COLLINS and ROTHENBUHLER 1978).

Analysis of the data was by nested analysis of variance. The linear model included sire-queen, colony nested in sire-queen, and observation nested in sire-queen and colony. The characters were separated into two groups based on number of observations per colony. Initial activity level, time to react to isopentyl acetate, and the duration of the response were measured up to 27 times per colony. Longevity and response to *N. apis* were measured 4 times per colony. All observations were used in estimating heritabilities and genetic and phenotypic correlations within groups. For correlations between characters in different groups, colony means were used to accomodate the different numbers of observations. Heritability was estimated by :

$$h^2 = \frac{4\sigma_S^2}{\sigma_S^2 + \sigma_C^2 + \sigma_E^2}$$

where σ_s^2 is the sire-queen variance component, σ_c^2 is the colony variance component and σ_e^2 is the sampling variance component. The standard error of h^2 was estimated by the methods of HAZEL and TERRILL (1945). Genetic correlations were estimated by :

$$r_g = \frac{\sigma_{xy}}{\sigma_x \sigma_y}$$

where σ_{xy} is the sire-queen covariance component and σ_x and σ_y are the square roots of the sire variance components for the covariate responses x and y. The standard error was estimated by the method of FALCONER (1960).

RESULTS

Not all of the 40 colonies produced sufficient brood for evaluation. However, the average number of full-sibling families per sire-queen tested for responses to I.P.A. was 2.4 and the number of observations ranged from averages of 37.9 to 43.5 per sire-queen family. An average of 3.6 full-sibling families per sire-queen was measured for longevity and response to *N. apis* and each colony was measured an average of 3.9 and 4 times, respectively. These measurements, with the varied numbers of observations, were considered sufficient to estimate heritabilities and genetic correlations.

Analysis of variance values are shown in Table 1. In the analyses for initial activity level, duration of response, and response to *N. apis*, the sire-queen mean

TABL. 1. — *Analysis of variance values for the five characters measured on caged honey bee workers.*

Character	Source	d.f.	Mean Square	$\bar{x} \pm$ S.D.
Initial Activity Level (no. of bees)	Sire queen	7	261.8	5.2 ± 3.6
	Colony (S.Q.)	11	451.3	
	Error	704	22.2	
Time to react to I.P.A. (sec)	Sire queen	7	327.0	6.7 ± 2.4
	Colony (S.Q.)	11	298.1	
	Error	704	35.6	
Duration of response (sec)	Sire queen	7	2 085.8	39.7 ± 10.2
	Colony (S.Q.)	11	3 314.0	
	Error	704	283.3	
Longevity (days)	Sire queen	7	115.2	17.2 ± 4.4
	Colony (S.Q.)	21	71.7	
	Error	82	24.2	
Response to *Nosema apis* (days)	Sire queen	7	25.6	16.8 ± 4.9
	Colony (S.Q.)	21	77.1	
	Error	82	19.2	

squares (MS) were smaller than the colony mean squares. Since sire-queen variance components in half-sibling analyses are calculated as

$$\text{MS sire} - \frac{\left(\frac{\text{MS colony} - \text{MS observations}}{K_1}\right) * K_2 - (\text{MS observations})}{K_3}$$

where K_1 = the number of colonies per sire-queen, K_2 = the number of sire queens, and $K_3 = K_1 * K_2$, negative sire-queen variance estimates would result. Such negative sire-queen variance components preclude estimates of either heritabilities or genetic correlations.

Analysis of measurements of longevity and time to react to I.P.A. yielded heritability estimates of 0.32 ± 0.27 and 0.03 ± 0.006, respectively (Table 2). The estimate of genetic correlation between these characteristics was 0.72 ± 0.87.

TABL. 2. — *Estimates of heritability and genetic and phenotypic correlations*[a].

	Initial activity level[b]	Time to react	Duration	Longevity
Time to react to I.P.A.	— .61**	0.3 ± .006	NE	.72 ± .87
Duration of response	.15	— .66*	NE	NE
Longevity	— .08	— .06	— .17	.32 ± .27
Response to *Nosema apis*[b]	— .15	— .09	— .001	.76**[c]

a. Heritabilities (h^2s) are underlined. Genetic correlations are above and to the right of the h^2s. Phenotypic correlations are below and to the left of the h^2s.

b. Heritabilities and thus genic correlations for initial activity level and response to *Nosema apis* were not estimable and thus do not appear in the table.

c. N = 29, all others are N = 19.

NE Not estimable.

* $P < 0.05$.

** $P < 0.01$.

Of the 10 phenotypic correlations, three were significant. Time to react to I.P.A. was negatively correlated with both the duration of response (— 0.66, $P < 0.05$) and the initial activity level (— 0.61, $P < 0.01$). This agrees with phenotypic correlations found by COLLINS, *et al.* (1982). Response to *N. apis* was positively correlated with longevity (0.76, $P < 0.01$).

DISCUSSION

Those characteristics for which heritabilities were not estimable essentially showed greater variation within half-sibling families than between them. Apparently, non-additive effects (dominance, epistasis, environment, or the interaction of these effects) accounted for major portions of the total variance. This collective variance was sufficiently large that the half-sibling analysis, which assays 1/4 of the additive genetic variance, could not distinguish a clear additive component in the total variance. Such results can be expected if additive genetic variance is relatively small, or if the sample size is very small. Thus, our results suggest that the additive genetic features of the systems regulating initial activity levels in evaluations of response to I.P.A., the durations of response to I.P.A., and response to *N. apis* are substantially less important than the non-additive genetic or environmental effects. This suggests that genetic improvement in these traits will require breeding methods that can increase the accuracy of selection such as progeny testing or those that utilize non-additive genetic variance such as cross-breeding or reciprocal recurrent selection.

Longevity, with a h^2 of 0.32 ± 0.27, shows good prospects for improvement through selection. The time to react to I.P.A. has a genetic correlation with longevity of 0.72. Thus, many additive genetic effects regulating longevity also probably effect the time to react to I.P.A. However, the apparent low genetic variation of the time to react to I.P.A. suggests overriding effects of dominance, epistasis, or environment. Attention to these effects during a selection program would probably permit the development of long-lived bees not unacceptably quick to defend their colonies.

Selecting for longevity might also improve response to *N. apis* if this characteristic's strong phenotypic correlation with longevity is largely based on additive genetic effects. The present evidence, in concert with that of RINDERER and SYLVESTER (1978) suggests that adverse dominance or epistatic effects on response to *N. apis* may be eliminated from a breeding population by inbreeding, outcrossing, or reciprocal recurrent selection, and that the additive effects commonly influencing longevity and response to *N. apis* may be used to improve both characteristics.

Thus, these data suggest that longevity would respond to selection and that correlated responses in defensive behaviour and resistance responses to *N. apis* would probably be favorable. Further work with these traits is needed to more precisely estimate the genetic parameters and relationships with other traits of interest and to compare predicted and actual responses to selection in selection experiments.

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RÉSUMÉ

RÉACTION DE L'ABEILLE A *NOSEMA APIS*,
LONGÉVITÉ ET RÉACTION D'ALARME A L'ACÉTATE D'ISOPENTYL :
HÉRITABILITÉS ET CORRÉLATIONS

On a séparé en 8 groupes de 5 des reines vierges prises dans 40 colonies choisies au hasard. Chaque reine a été inséminée artificiellement par un seul mâle provenant des 8 « reines-pères ». On a mesuré les caractéristiques suivantes sur les ouvrières fraîchement écloses de cette série de familles super-sœurs/demi-sœurs : réaction à *Nosema apis*, longévité (nombre de jours nécessaires pour que la moitié de l'échantillon soit mort) et la réaction à la phéromone, l'acétate d'isopentyl (niveau initial d'activité, temps nécessaire pour obtenir une réaction, durée de la réaction). On a traité les données par une analyse hiérarchisée de la variance et calculé l'héritabilité h^2, les corrélations génétiques et phénotypiques. Les héritabilités sont les suivantes : temps de latence à l'I.P.A. = 0,03 ± 0,006; longévité : 0,32 ± 0,27. On n'a pas pu estimer les autres valeurs d'h^2 en raison de la grande variance non additive des facteurs génétiques ou des facteurs du milieu.

ZUSAMMENFASSUNG

REAKTION DER HONIGBIENE AUF *NOSEMA APIS*,
LANGLEBIGKEIT UND ALARMREAKTION AUF ISOPENTYLAZETAT :
HERITABILITÄTEN UND KORRELATIONEN

Unbegattete Königinnen von 40 nach Zufall ausgewählten Völkern wurden in 8 Gruppen zu je 5 aufgeteilt. Jede Königin in einer Gruppe wurde mittels instrumenteller Besamung mit einem einzigen Drohnen von einer der 8 Eltern-Königinnen gepaart. Frischgeschlüpfte Arbeitsbienen aus jedem Volk dieser Reihe von Supergeschwister- Halbgeschwisterfamilien wurde hinsichtlich folgender Eigenschaften gemessen : Reaktion auf *Nosema apis*, Lebensdauer (Anzahl der Tage, bis die halbe Versuchsgruppe tot war) und Reaktion auf das Alarmpheromon Isopentylazetat (anfängliche Aktivitätsstufe, Zeit bis zum Sichtbarwerden einer Reaktion und Dauer der Reaktion). Die Daten wurden mit Hilfe einer hierarchischen Varianzanalyse bearbeitet und die Heritabilität (h^2) sowie die genetischen und phänotypischen Korrelationen berechnet.

Die Heritabilitäten waren : Zeit bis zur Reaktion auf I.P.A. = 0,3 ± 0,006 und Lebensdauer = 0,32 ± 0,27. Andere h^2-Werte konnten wegen der großen nicht-additiven genetischen und Umwelt-Varianz nicht geschätzt werden.

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