large wing surface and a consequently high susceptibility to entrapment. When *Papilio* was thrown by hand into large orbs of *Argiope florida*, it stuck to the webs and was caught by the spiders. Even *Danaus* (Table 1, No. 16), whose scales are more loosely attached, suffered the same fate.

There exist insects whose wings and bodies possess a flaky or powdery covering, supposedly of wax. Among them are the so-called white flies (Hemiptera, Aleurodidae). Minute in size, their wings could not be tested by our technique. However, when they were held in forceps and brushed against spider thread, they did not adhere at all. Subsequent examination of the thread showed it to be densely laden with the "waxy" powder (Fig. 1, part *G*).

Tests were also made to determine the adhesiveness of viscid thread to the cuticle of the spider itself. The two pieces of cuticle (of *Nephila clavipes*) that were tested, from the ventral opisthosoma and the patella of a leg, respectively, were shown to adhere with a strength comparable to that of "naked" insect wings (13 to 18 dyne/mm for the opisthosoma and 11 to 14 dyne/mm for the patella). Of course, a spider ordinarily restricts contact with the web to the claws at the tips of its legs, and generally clings only to nonadhesive fibers. Nevertheless, the possibility exists that the claws or tarsi, or their accessory structures, are actually nonadhesive, but this remains to be investigated.

In conclusion, it seems clear that an outer coating of detachable and partly dispensable structures—of which the scales of moths are but one example—is a distinct adaptive asset to any flying insect. It would be improper to conclude that a decreased vulnerability to capture by spiders is the sole advantage to be derived from the possession of such a coating. Certainly in the case of moths, the dense scales might also play a subtle aerodynamic role. But orb-weavers are an ever-present hazard to any insect on the wing, and their role in forcing upon their prey the evolutionary aquisition or refinement of any mechanism that reduces chances of entrapment can certainly not be dismissed.

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3. Admittedly, a more sophisticated apparatus could have been used, but this research was conceived and carried out under field conditions, and all techniques were improvised from readily available materials. We thank Professor Philip Morison (Cornell University) who some years back showed Eisner the soda-straw balance while the two were working at Educational Services Inc., Watertown, Mass.
4. This calculation involved converting the corrected load to a force in dynes, and then dividing by 2 (since the length of thread attached to cuticle was 2 mm).

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Histamine: Differences in Amount Available for Release in Lungs of Guinea Pigs Susceptible and Resistant to Acute Anaphylaxis

Abstract. *Susceptibility to acute anaphylaxis in guinea pigs is related to the quantity of liberable histamine available for release in the lung. In highly susceptible Hartley animals this amount can be more than 10 times greater than in the resistant strain 2. Strain 2 and Hartley guinea pigs are equally susceptible to histamine toxicity, and their organs fix I^{131}-labeled antibody equally well.*

There has long been interest in the genetics of allergic reactions in man despite limited possibilities for experimentation. Although rats and mice are not incapable of reactivity, guinea pigs continue to be preferred for research in hypersensitivity. Recent availability of inbred, histocompatible guinea pigs (*1*) of the Sewall Wright strains 2 and 13 (*2*) now opens the way to study of the genetics of allergic reactions (*3, 4*).

Individual and strain differences in susceptibility to contact agents have been investigated by Chase (*5*). In other kinds of experiments on delayed hypersensitivity it has been reported that strain 2 is more resistant to induction of allergic encephalomyelitis than either strain 13 or Hartley guinea pigs (*4*). Concerning immediate-type reactions, studies on differences in susceptibility to anaphylactic reactions were undertaken years ago by Zinsser and Enders (*6*) who compared their susceptible guinea pigs from dealer J with resistant animals from dealer M; Zinsser and Enders used a reversed passive anaphylaxis system (rabbit antiserum against horse serum).

During studies on protracted anaphylactic shock, the finding that strain 2 guinea pigs were considerably more resistant than Hartley animals to the acute (bronchospasm) phase of anaphylactic shock (*3*) led us to try to elucidate the mechanism of the bronchospasm and the basis of the differences in susceptibility.

The possibility that the strain difference in susceptibility to acute anaphylaxis was due to differences in susceptibility to histamine was investigated by intravenous injection of histamine and use of histamine aerosols. No differences were found between strain 2 and Hartley animals either in the amount of an intravenous lethal dose of histamine or in susceptibility to histamine aerosols. The intravenous minimum lethal dose for strain 2, Hartley, and strain 13 is roughly the same as that reported previously—about 0.3 mg/kg (*7*).

The possibility that the strain differences reflected differences in the sequestering of passively administered antibody was investigated by measurement of rabbit I^{131}-labeled antibody to egg albumin in the various tissues 19 to 24 hours after intravenous injection of the labeled antibody in Hartley and strain 2 guinea pigs. No differences were discernible in the sequestration of antibody in lung, intestine, spleen, skin, liver, or muscle.

Because there might be genetic differences in the amount of histamine present in the lungs of guinea pigs, we determined by bioassay (*8*) the histamine content of tissues from the Hartley strain, strain 2, and strain 13 (Table 1). There were significant differences in the histamine contents of certain tissues. Most striking was the threefold difference in lung histamine between strain 2 (10.5 ± 4.5 μg/g) and Hartley

Table 1. Histamine in guinea pig tissue (μg/g).

Lung	Intestine	Skin	Spleen	Liver
		Hartley		
31.2±15.8*	15.1±5.7	4.3±1.5	6.8±2.0	2.4±1.8
		Strain 2		
10.5± 4.5	7.5±3.1	3.4±1.3	3.9±1.7	0.8±0.9
		Strain 13		
17.6± 4.3	16.9±5.5		7.7±6.4	2.9±0.8

* Standard deviation.

Table 2. Anaphylaxis in isolated lung preparations.

Antibody (μg N)	Intensity* of bronchospasm†	Histamine released (μg hist. base)
	Hartley	
5	0, +, +++, ++	0.0, 0.0, 0.47, 0.04
20	+++, +++, +++, +++, +++	3.8, 2.3, 19.5, 5.5, 0.9
33	+++, +++, +++, +++, +++, +++	2.1, 10.5, 31.8, 1.6, 14.2, 1.2
100	+++, +++, +++, +++, +++	86.2, 0.6, 3.3, 2.3, 5.5
500	++, +++, +++, +++	3.2, 11.8, 4.0, 9.9
	Strain 2	
20	++, +++B, +R	0.8, 0.0, 0.0
33	+++B, +++, +++B	1.1, 0.5, 0.6
100	+++, +	1.1, 0.6
130	+, +R, +R	1.9, 1.1, 0.5
260	++B, ++, +B, +++B	1.6, 0.7, 0.6, 0.08
500	0, +++, ++, 0	1.4, 1.1, 0.8, 0.07

* Scoring of bronchospasm: +++, complete; ++, medium; + minimal; 0, nil; R, retarded; B, brief. † Each entry below represents the response of a single animal; the amount of histamine released from the lung of that animal appears in the corresponding position in the last column.

(31.2 ± 15.8 μg/g). The amount in the lungs of strain 13 guinea pigs was intermediate; these animals displayed a susceptibility lying somewhere between the susceptibility of the other two strains. There appears to be more histamine in the livers of Hartley guinea pigs than in the livers of strain 2; intestinal amounts also were slightly greater for the Hartley strain.

In preliminary experiments with the lung-slice method of Mongar and Schild (*9*), the amounts of histamine liberated from lung tissues of strain 2 and Hartley guinea pigs differed by 5- to 20-fold. We then employed the method of bronchospasm in vitro (*10*). Isolated lung preparations from guinea pigs sensitized 19 to 24 hours previously with I^{131}-labeled rabbit antibody globulin to egg albumin (*10*) were challenged with 2 mg of egg albumin which was added to the perfusion fluid.

Releases during bronchospasm in vitro of histamine from the lungs of strain 2 and Hartley guinea pigs are compared in Table 2. The release, which is presumably due to an enzymic mechanism (*10, 11*), is much less from the lungs of strain 2 guinea pigs. On the basis of the molecular ratios between tissue-sequestered antibody and released histamine in the Hartley guinea pig, it is estimated that for each molecule sequestered in the lung 1000 to 100,000 molecules of histamine are found in the perfusion outflow during bronchospasm (*10*). Hartley pigs receiving antibody (20 μg of nitrogen or more) to ovalbumin released up to 86 μg of histamine. These results provide some evidence that with doses of passively administered antibody (from 20 μg to 500 μg of nitrogen) all the *libérable* ("liberable," available for release) histamine in the lungs is discharged. Under these circumstances, strain 2 guinea pigs released only 0.5 to 2 μg of histamine, so that the molecular ratios of histamine to antibody (roughly 750 to 1500) were lower in this strain. The threshold dose of antibody was slightly higher (33 μg of nitrogen) in strain 2. The lungs of Hartley guinea pigs receiving 20 μg of nitrogen or more of the antibody to ovalbumin invariably showed complete bronchospasm, whereas under the same conditions lungs of strain 2 had either incomplete bronchospasms or complete bronchospasm of limited duration.

Thus, the amounts of liberable histamine in lung tissues are casually related to the susceptibility of Hartley guinea pigs and to the resistance of the strain 2 guinea pigs to acute anaphylactic shock. Since there is apparently a rather constant amount of non-liberable histamine (roughly 10 μg/g) in guinea pig lungs (*12*), the threefold difference in total lung content of histamine between strain 2 and Hartley guinea pigs is only a background to much greater difference in the amounts of histamine actually released during bronchospasm in these strains. Strain 2 animals with 15 μg/g in their lungs have 5 μg/g available for release, while Hartley guinea pigs, with 47 μg/g, have 37 μg/g available for reactivity. These differences may also explain the failure of strain 2 guinea pigs to manifest their resistance when challenged intravenously; even the small amounts of liberable histamine in this strain are sufficient for lethal bronchospasm if discharged suddenly. On the other hand, the large amounts of histamine in the lungs of Hartley guinea pigs can produce a lethal outcome even under the conditions of slow absorption of antigen from the subcutaneous tissues where enough histamine may be released over a short enough period to cause death by bronchospasm (*3*). This and previous work (*10*), make it evident that there is considerable individual variation within the random-bred Hartley strain.

The existence of "allergic types" among guinea pigs should promote research in the genetics of hypersensitivity. It seems to us that the susceptibility associated with large amounts of liberable histamine is probably a function of the presence of larger numbers of mast cells in the lung.

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