pht I (from 70 samle means)

	pn	mn	pp	pe	uhl	pnl
uh	0.2636	0.2359	0.1056	0.0283	0.4299	−0.1951
pn	–	0.3980	0.3707	0.0840	0.1053	0.2627
mn			0.3798	0.2046	−0.2317	−0.1043
pp				0.3805	0.2203	0.1737
pe					0.0995	0.1661
uhl						0.4510

mean 0.1825 ± 0.1949 (n = 21)

pht R (from 195 sample means)

	pn	mn	pp	pe	uhl	pnl
uh	0.6089	0.5250	0.3764	0.3260	0.5251	0.5412
pn	–	0.7555	0.7022	0.5597	0.4776	0.6121
mn		–	0.6660	0.5321	0.3997	0.4318
pp			–	0.6841	0.3424	0.4322
pe				–	0.2762	0.3783
uhl					–	0.5870

mean 0.5114 ± 0.1341 (n = 21)

The correlations printed in heavy type are significant at the $p < 0.01$ level. We observe a highly significant breakdown of correlations in **pht I** compared to **pht P** and **pht R.** This is an exciting phenomenon which can not be explained as an artefact of erroneous phenotype determination: a false allocation of **pht P** or **pht R** samples to **pht I** would in both cases raise the correlations in the **pht I** matrix and, on the other hand, a false allocation of **pht I** samples to **pht P** or **pht R** would not reduce the correlations in the **pht P** and **pht R** matrices.

A biological explanation of this phenomenon could be that **pht R** and **pht P** have lived in *de facto* reproductive isolation for longer periods in the past when there was a natural, compact woodland structure in Central Europe which reduced the chance of encounters of the two phenotypes (see sections 5.2, 6 and 10). This lead to genetical divergence. With the drastic change of woodland distribution and structure after the large clearfellings in the beginning of this millenium, the reproductive isolation was broken and hybrids still fertile and with sufficient fitness occurred. However, a reduced fitting-together between certain gene products seems possible. Such an affection of harmonizzing within multiple gene systems should be tolerated as far as only peripheral phenes which do not have a notable influence on fitness and no fundamental functional systems are affected. Thus a possible hybrid origin could explain the breakdown of correlations between pilosity characters in **pht I.**

Intranidal variability of pilosity data could indicate genetic heterogenity which should be larger in a hybrid population. In analysing intranidal variability we encounter several problems which make comparisons between the phenotypes very difficult:

(i) the frequency of polygyneous nests differs (97.6 % in **pht P,** 85.5 % in **pht I,** 24.1 % in **pht R**),

(ii) concluded from nest size differences, the average queen numbers in polygyneous nests differ (**pht P^P > pht I^P > pht R^P**),

(iii) the pilosity data are not normal distributed but positively skewed in case of very low nest means,

(iv) within a phenotype, the standard deviation of pilosity data increases more slowly than the nest mean (see Fig. 12) – i. e. the ratio SD/mean decreases with increasing means.

These problems could be avoided or diminished if the comparisons are restricted to monogyneous samples which have similar nest means of pilosity. For a test between **pht I** and **pht R** and the characters **pn** and **pp** I have considered only samples in the interval **pn** [9.7, 17.4] and **pp** [7.2, 12.3] where problem (iii) is absent and problem (iv) has no importance. Seven monogyneous samples of **pht I** with **pn** [9.7, 14.6] had standard deviations of **pn** (SD_{pn}) of 3.5, 3.7, 4.1, 6.2, 6.4, 6.8, 11.6 and eight monogyneous samples of **pht R** with **pn** [11.3, 17.4] had SD_{pn} of 1.9, 2.0, 2.3, 2.7, 2.7, 3.9, 3.9, 6.3. Seven samples of **pht I** with **pp** [7.2, 11.9] had SD_{pp} of 2.8, 3.0, 3.0, 3.85, 5.5, 5.64, 6.0 and 17 samples of **pht R** had SD_{pp} of 1.1, 1.4, 1.8, 2.2, 2.23, 2.3, 2.3, 2.4, 2.5, 2.5, 2.83, 3.55, 3.6, 3.6, 3.95, 4.1. According