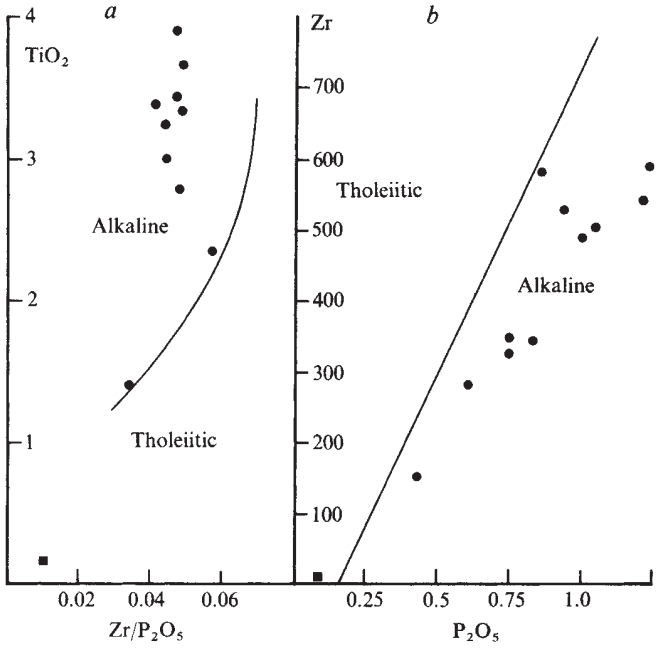

Fig. 3 Kopi boninite (■) and coeval alkaline basaltic rocks (●) plotted using immobile elements to discriminate between alkaline and theoleiitic fields (after ref. 14). *a*, TiO_2(wt%) against the ratio Zr/P_2O_5 (p.p.m.). *b*, Zr (p.p.m.) against P_2O_5 (%).

($Q = 6.0\%$), basic composition places the lava clearly within a spectrum of basaltic to andesitic rocks that are otherwise known only from modern oceanic areas, or ancient ophiolites that were segments of oceanic lithosphere.

The early Cretaceous sediments of eastern Wairarapa are thought to overlie or to represent the youngest part of a sequence of several kilometres of marine sediment of continental provenance, deposited more or less continuously throughout the Mesozoic. An Albian tectonic event deformed the soft sediments and initiated open-shelf deposition[8] which continued till late Tertiary time, when the present uplift and deformation began. Minor intraformational slumping may have occurred during deposition of Mangapokia sediments, but there is no evidence to suggest allochthonous emplacement of the lavas. Thus there is no indication that the area was anything other than a faulted, continent margin at the time the lava was erupted.

In the discussion of genesis of low-Ti magmas, there is general agreement that multistage melting is necessary to give extreme depletion of incompatible elements[1,4–6]. Olivine tholeiite and derivatives, produced by voluminous first-stage melting, are the habitual associates of most low-Ti lavas. However, lavas coeval with the Kopi boninite are alkalic basalts and derivatives, with marked enrichment in P, Ti, Y, Zr and Nb (Fig. 3), presumably generated by deep, low-volume, first-stage melting of upper mantle. Eruption of one or more such magma batches would have efficiently depleted a portion of mantle which then could have risen as a small diapir to melt at shallower depths and produce boninite.

If such a process did occur it is perhaps unusual that the association of boninite with alkalic basalt is so rare. The closest comparison seems to be in the Othris Mountains of Greece, where Cameron *et al.*[1] reported boninite in the Agrilia Formation. There, the associated volcanics are mildly undersaturated, alkaline types[12] erupted at a continental margin during a period of slow splitting, possibly caused either by continental rifting, or the initiation of a marginal ocean basin and nearby subduction zone[13]. It is probable that the eastern margin of the early Cretaceous, New Zealand continent was deeply fractured enough to allow boninite magma to erupt, but even if continental splitting had started it clearly failed to develop. It is certain that a spreading centre never formed at the site of deposition, and though there is a modern subduction zone to the east of northern New Zealand, the existence of one in early Cretaceous times is a moot point.

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Coefficients of relatedness in sociobiology

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A much-discussed, quantitative criterion for the spread of an altruistic gene is Hamilton's rule[1,2]

$$\frac{c}{b} < R \qquad (1)$$

where *c* and *b* are additive decrement and increment to fitness of altruist and recipient, respectively, and *R* is a measure of genetic relatedness between the two individuals. When rearranged as $-c.1 + b.R > 0$, the rule can be interpreted as requiring that the gene-caused action increase the 'inclusive fitness' of the actor. Since its introduction, Hamilton's rule and the attendant concept of inclusive fitness have gained increasing acceptance and use among biologists and have become integral in the field now named sociobiology. However, the essentially heuristic reasoning used in deriving these concepts, along with the lack of a complete specification even in the original outbred model[2], have led to many investigations into the population genetical underpinnings of Hamilton's rule[3–18]. On the basis of these considerations, several reports[3,10,12,13,18] have proposed new formulae for *R*. These formulae have no obvious relation to each other or to the coefficients originally suggested by Hamilton. This proliferation of coefficients is undoubtedly confusing to many and the net effect may be to generate distrust both of the rule and of the notion of inclusive fitness. Our purpose here is to show that these various formulae for *R* (refs 3, 10, 12, 13, 18), although independently derived, are actually the same.

What is *R* in equation (1)? On the basis of a particular outbred model, Hamilton[1,2] claimed that Wright's[19] 'coefficient of relationship' was the required *R*. However, later, giving a heuristic development but no further model, Hamilton[20,21] modified his identification of *R* with Wright's coefficient by claiming that equation (1) requires, in principle, a regression coefficient of

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genotype of recipient on genotype of altruist, whereas Wright's coefficient is the corresponding correlation coefficient. Such a correlation coefficient will often be the same as the regression coefficient but differs when the interactants are inbred to different extents[18]. These discussions[1,2,20,21] implied that R was independent of gene frequency and selection, and that equation (1) held for inbred populations (with the regression coefficient as R).

However, in recent models[3–17], R is taken to be the threshold value of c/b, below which the cost–benefit ratio must be for the gene to increase. The R formulated in this operational way need not necessarily correspond to any simple measure of genetic relationship. Some of these models[3–11,14,15,17] specify the whole mating process with respect to interactions within families. Other models[12,13,16] maintain the generality of Hamilton's[2] original approach, and apply to interactants of arbitrary relationship, but fail to specify the population and mating system processes which give rise to R. If Hamilton's rule is to be useful, it must turn out that R, as formulated by these various approaches, must not vary widely as gene frequency or the parameters of selection change, except as implied by the simple proportionality to c/b. In many cases of interest, R is constant[4,7–9,15], but in others[3,4,7,10–13,16] dependencies on gene frequency and dominance enter into calculations of R.

Our analysis here will not consist of any derivations of the coefficients from the gene frequency dynamics, as that is done from different points of view in the various works to be considered[3,10,12,13]. Instead, we will simply convert these various coefficients into a common symbolism and show that they are, in fact, identical. However, before doing so, we should acknowledge an important aspect of the derivations: either selection must be weak so that standard identity coefficients can be used as measures of genetic relationship, or rather special conditions must be imposed on the models so that there is no selection at internal points of the pedigree patterns studied.

The genetic relationship between two diploid individuals, X and Y, at a single locus can be summarized by the following nine 'condensed' identity coefficients $\Delta_1, \Delta_2, \ldots, \Delta_9$, where Δ_i is the probability of genetic identity event i obtaining (Fig. 1 upper left-hand corner)[22]. These coefficients consider identity states among all four alleles present in X and Y. The traditional two-allele coefficients, interpreted as probabilities, are

$$f_X = \Delta_1 + \Delta_2 + \Delta_3 + \Delta_4 \qquad f_Y = \Delta_1 + \Delta_2 + \Delta_5 + \Delta_6 \tag{2a}$$

$$f_{XY} = \Delta_1 + \tfrac{1}{2}(\Delta_3 + \Delta_5 + \Delta_7) + \tfrac{1}{4}\Delta_8 \tag{2b}$$

where f_X and f_Y are the inbreeding coefficients of X and Y, respectively, and f_{XY} is the 'coefficient of consanquinity' between X and Y, or the probability that a random allele from X is identical by descent with a random allele from Y at the locus of interest. Letting X denote the altruist and Y the recipient, the coefficient which we will use to relate the others may now be defined as

$$R = \frac{f_{XY}\alpha + (\Delta_1 + \frac{1}{2}\Delta_3)(1-\alpha)}{\frac{1}{2}\alpha + f_X(1-\frac{1}{2}\alpha)} \tag{3}$$

where $\alpha = 2q + 2h - 4qh$, p is the frequency of the non-altruist allele A with $q = (1-p)$ the frequency of the altruistic allele a, and h is the probability that a heterozygote performs an altruistic act. If we express the phenotypes as the probabilities of behaving altruistically (0, h, 1 corresponding to AA, Aa, aa),

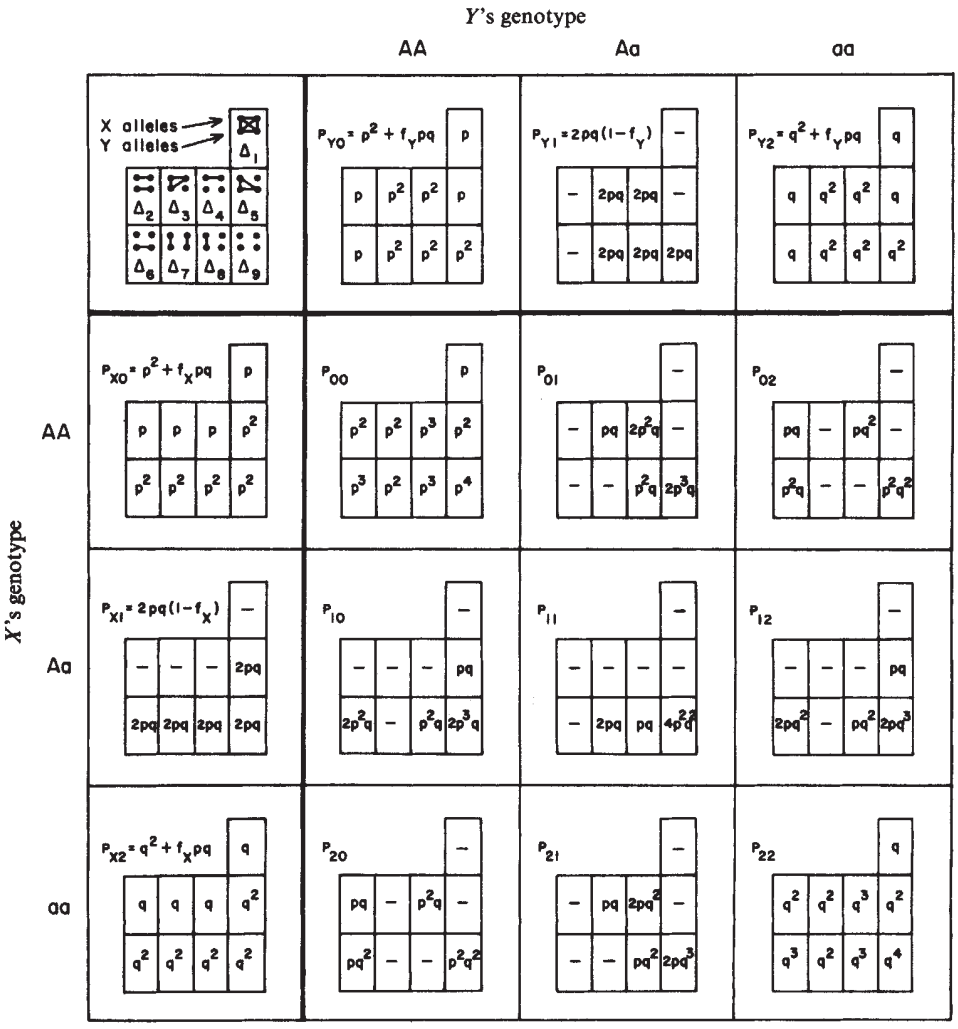

Fig. 1 Joint and marginal distribution of genetic identity states with genotypes. The nine 'condensed' identity states are given in the upper left-hand corner with probabilities Δ_i $(i = 1, 2, \ldots, 9)$, $\Sigma_i \Delta_i = 1$. The alleles of X are on top and the alleles of Y on the bottom. A line connecting alleles indicates identity by descent. Given the occurrence of each identity state, the distribution of the genotypes of X and Y are given within the large side and top three boxes, respectively. For example, if identity state 5 obtains, then X is AA, Aa, aa with probabilities p^2, $2pq$, q^2, respectively; the like probabilities for Y are p, 0, q, respectively. Given the distribution of identity states, the marginal distribution of genotypes is given in the top left corner of each of these six large boxes. The joint distribution of genotypes are given in the nine large boxes towards the bottom right of the figure. For example, if identity state 3 obtains, then

$$P_{00} = p^2, \quad P_{01} = pq,$$
$$P_{03} = P_{10} = P_{11} = P_{12} = P_{20} = 0,$$
$$P_{21} = pq \quad \text{and} \quad P_{22} = q^2.$$

Table 1 Joint and marginal distributions of X_g and Y_g and X_p and Y_p

X \ Y	Y_g	0	$\frac{1}{2}$	1	
	Y_p	0	h	1	
X_g	X_p				
0	0	P_{00}	P_{01}	P_{02}	$P_{X0}=p^2+f_Xpq$
$\frac{1}{2}$	h	P_{10}	P_{11}	P_{12}	$P_{X1}=2pq(1-f_X)$
1	1	P_{20}	P_{21}	P_{22}	$P_{X2}=q^2+f_Xpq$
		$P_{Y0}=p^2+f_Ypq$	$P_{Y1}=2_{pq}(1-f_Y)$	$P_{Y2}=q^2f_Ypq$	1

P_{ij} is the joint distribution of genotypes i and j and P_{Xi}, P_{Yi} are the marginal distributions of the genotypes of X and Y, respectively ($i, j = 0, 1, 2$). From the table, the following expressions can be calculated for use in calculations concerning equations (5): $E(X_p)=hP_{X1}+P_{X2}$, $E(Y_p)=hP_{Y1}+P_{Y2}$, $E(Y_g)=\frac{1}{2}P_{Y1}+P_{Y2}=q$, $E(X_g)=\frac{1}{2}P_{X1}+P_{X2}=q$, $E(Y_gX_p)=\frac{1}{2}hP_{11}+hP_{12}+\frac{1}{2}P_{21}+P_{22}$, $E(X_pX_g)=\frac{1}{2}hP_{X1}+P_{X2}$, $E(X_gY_g)P_{11}+\frac{1}{2}P_{12}+\frac{1}{2}P_{21}+P_{22}$, $E(X_g^2)=\frac{1}{4}P_{X1}+P_{X2}$.

and the genotypes as the fractions of altruistic alleles (0, $\frac{1}{2}$, 1 corresponding to AA, Aa, aa), then α is simply the regression of phenotype on genotype in a randomly mating population. This convention regarding the genotypes and phenotypes will be used throughout. With a change in notation, R as given by equation (3) is identical to the coefficient derived by Flesness and Holtzman[13] (equation (10), where $P(H)=\Delta_1+\frac{1}{2}\Delta_3$ and their $h=\frac{1}{2}\alpha$). It is important to note that if the gene effects are additive, if there is no inbreeding or if $p=q=0.5$, then equation (3) reduces to the coefficient proposed by Hamilton[20,21]:

$$R=\frac{2f_{XY}}{1+f_X} \tag{4}$$

Consequently, the regression coefficient (equation (4)) will be a good estimate of R in many cases of interest. We now show that the coefficients derived elsewhere in the literature[3,10,12,18] are identical to equation (3).

Below, we index genotypes with a single subscript i, with $i=0$, 1, 2 corresponding to genotypes AA, Aa, aa, respectively. Let P_{ij} be the joint distribution of genotypes i and j in the population with i being the genotype of X and j the genotype of Y (Fig. 1, Table 1). Let X_g, X_p and Y_g, Y_p denote the genotype and phenotype of X and Y, respectively.

Orlove[3] and Orlove and Wood[10] derive $R=\mathrm{Cov}\,(X_g, Y_p)/\mathrm{Cov}\,(X_g, X_p)$ in an outbred model of kin selection in haplodiploid families. Their coefficient[3,10] equals

$$R=\frac{\mathrm{Cov}\,(Y_g, X_p)}{\mathrm{Cov}\,(X_g, X_p)} \tag{5}$$

if there is no inbreeding. However, as we now show, equation (5) is more generally correct, as well as being the most concise and intuitive representation of equations (3) or (17).

To relate equation (5) to equation (3), we must first calculate the required covariances. The information needed to do so is given in Table 1, in which the genotypes and phenotypes of X and Y are given along with the joint and marginal probabilities. Using this information to evaluate the covariances in equation (5), we obtain directly

$$\frac{\mathrm{Cov}\,(Y_g, X_p)}{\mathrm{Cov}\,(X_g, X_p)}=\frac{\frac{1}{2}P_{11}h+hP_{12}+\frac{1}{2}P_{21}+P_{22}-(\frac{1}{2}P_{Y1}+P_{Y2})(hP_{X1}+P_{X2})}{\frac{1}{2}hP_{X1}+P_{X2}-(hP_{X1}+P_{X2})(\frac{1}{2}P_{X1}+P_{X2})} \tag{6}$$

From Table 1, we can also obtain for future use

$$\mathrm{Cov}\,(Y_g, X_g)=\tfrac{1}{4}P_{11}+\tfrac{1}{2}P_{12}+\tfrac{1}{2}P_{21}+P_{22}-(\tfrac{1}{2}P_{X1}+P_{X2})(\tfrac{1}{2}P_{Y1}+P_{Y2}) \tag{7}$$

$$\mathrm{Var}\,(Y_g)=\tfrac{1}{2}qp(1+f_Y) \tag{8a}$$

and

$$\mathrm{Var}\,(X_g)=\tfrac{1}{2}qp(1+f_X) \tag{8b}$$

Substituting equation (7) into the numerator of equation (6)

$$\mathrm{Cov}\,(Y_g, X_p)=\mathrm{Cov}\,(Y_g, X_g)+(h-\tfrac{1}{2})(\tfrac{1}{2}P_{11}+P_{12}-qP_{X1}) \tag{9}$$

The denominator of equation (6) can be simplified directly to

$$\mathrm{Cov}\,(X_p, X_g)=pq[\tfrac{1}{2}\alpha+f_X(1-\tfrac{1}{2}\alpha)] \tag{10}$$

As shown in Fig. 1 (see also refs 12, 22, 23), it is possible to express the joint probabilities of the interactions, P_{ij}, in terms of the condensed identity coefficients and the gene frequency. In particular,

$$\begin{aligned}P_{11}&=2pq(\Delta_7+\tfrac{1}{2}\Delta_8+2pq\Delta_9)\\P_{12}&=pq(\Delta_5+2q\Delta_6+q\Delta_8+2q^2\Delta_9)\end{aligned} \tag{11}$$

These formulae (equation (11) and Fig. 1) are only strictly correct for neutral genes; however, it is hoped that they are approximately correct if selection is weak. Substituting equation (11) into equation (9) and recalling

$$P_{X1}=(1-f_X)2pq$$

we obtain after some algebra

$$\mathrm{Cov}\,(Y_g, X_p)=\mathrm{Cov}\,(Y_g, X_g)+\tfrac{1}{2}pq(\alpha-1)[\Delta_5+\Delta_7+\tfrac{1}{2}\Delta_8] \tag{12}$$

Letting r_{XY} be the correlation between the genotypes of X and Y, we have

$$\mathrm{Cov}\,(Y_g, X_g)=r_{XY}[\mathrm{Var}\,(X_g)\,\mathrm{Var}\,(Y_g)]^{1/2} \tag{13}$$

However[19],

$$r_{XY}=\frac{2f_{XY}}{[(1+f_X)(1+f_Y)]^{1/2}} \tag{14}$$

On substituting equations (8) and (14) into equation (13), we obtain

$$\mathrm{Cov}\,(Y_g, X_g)=f_{XY}pq$$

and so equation (12) becomes

$$\mathrm{Cov}\,(Y_g, X_p)=f_{XY}pq+\tfrac{1}{2}pq(\alpha-1)(\Delta_5+\Delta_7+\tfrac{1}{2}\Delta_8) \tag{15}$$

After rearranging equation (15) and using equation (2b)

$$\mathrm{Cov}\,(Y_g, X_p)=pq[f_{XY}\alpha+(1-\alpha)(\Delta_1+\tfrac{1}{2}\Delta_3)] \tag{16}$$

On substituting equations (16) and (10) into equation (5), we obtain equation (3) as was to be shown.

Michod[12] (equation (3)) derived the following coefficient for use in Hamilton's rule

$$R=\frac{2xp\rho+(1-x)(1-2q)\rho'}{1+x-2q} \tag{17}$$

$$\rho=\frac{(\Delta_1+\frac{1}{2}\Delta_3)}{q+pf_X}+\frac{q(\Delta_5+\Delta_7+\frac{1}{2}\Delta_8)}{q+pf_X} \tag{18a}$$

and

$$\rho'=\frac{\Delta_5+\Delta_7+\frac{1}{2}\Delta_8}{1-f_X} \tag{18b}$$

which are Jacquard's conditional genic structures[12,22,23]. These conditional coefficients in equation (18) give the probability with which X can predict, on the basis of pedigree ties, the allelic distribution in gametes of Y. This probability depends on

whether X is homozygous (equation (18a)) or heterozygous (equation (18b)). In addition, the variable x appearing in equation (17) is the frequency of altruistic homozygotes among the total class of altruists and is

$$x = \frac{q^2(1-f_X)+f_Xq}{q^2(1-f_X)+qf_X+(1-f_X)2pqh} \quad (19)$$

Equation (19) corrects a mistake in Table 1 of Michod[12] where it was thought that the average inbreeding coefficient (averaged over the frequencies of X and Y in the population) should be used in equation (19). This is incorrect due to the conditional nature of the altruism. As the gene is only expressed in X, when X is in a certain relationship to Y (the relationship being summmarized by the Δ values), it is only the frequency of homozygous altruists among all X individuals which matter in equation (19). This fact alone accounts for the incorrect impression, given in Fig. 2b of that paper[12], that dominant genes could never increase in frequency. (Fig. 2a of ref. 12 is unaffected by these considerations.) After substituting equations (19) and (18) into equation (17) and collecting the Δ values in terms of f_{XY}, equation (3) is obtained after some rearrangement.

In conclusion, the various coefficients derived in the literature[3,10,12,13,18] for use as R in Hamilton's rule are equivalent. These coefficients were originally derived from very different points of view. Given the results here, these various approaches support each other and suggest that the one pleomorphic coefficient [equations (3), (5) or (17)] is, indeed, correct.

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Proline and valine—cues which stimulate grasshopper herbivory during drought stress?

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Insect herbivores, including members of Orthoptera and Lepidoptera, benefit from increased nitrogen in their food, particularly if it is in the form of easily digested amino acids[1,2]. I report here experimental evidence that grasshoppers detect and preferentially feed on grasses treated with the amino acids proline and valine, which commonly increase in plants under drought stress[3]. This ability may lead to insect concentrations on drought-stressed and nitrogen-enriched plants and thus exacerbate acridid population outbreaks through enhanced growth and survival.

Ecological studies of grasshoppers and locusts have often considered weather factors to be important determinants of population size or growth rate[4–9]. Dry soil conditions in particular seem to favour population increases of many economically important Orthopteran species[10]. It has been hypothesized that grasshopper growth rates, and consequent survival, increase sharply in drier conditions when perennial grasses have elevated nitrogen (N) levels in parts above ground[1]. Insect herbivores are generally likely to benefit from increased dietary N (ref. 2).

Such elevated nitrogen levels may be the result of increased concentrations of proline[3]. A highly soluble amino acid, proline does not hamper the activities of many photosynthetic enzymes and is able to provide osmotic adjustment in conditions of drought stress or salt accumulation[11–17]. Glycine and valine may function similarly[18]. Leaf laminae accumulate the largest quantities of the newly synthesized proline although it is also found in most other above-ground parts[19]. Genetic varieties of plants which accumulate larger concentrations of free proline tend to survive water stress better and grow faster after stress is relieved[20].

Mobile herbivores which can detect and concentrate their feeding on N-enriched plants will be favoured relative to conspecifics lacking these abilities. These abilities will be particularly important in environments with food patches of varying N quality. For example, in the northern Great Plains of North America, soil moisture is usually limiting to plant growth in summer. Here forage quality—normally influenced by rainfall—is patchy because thundershowers are local and infrequent.

I conducted a field experiment in a native Montana grassland to test the hypothesis that chewing insects can detect plants with elevated amino acid nitrogen levels within the range of levels that occur naturally. A completely randomized two-factor design was replicated three times. Plots of 2 m^2 separated by 4 m buffers were treated. Treatment levels by factor were Factor A (irrigation): none and 25 mm soil surface irrigation; Factor B (foliar sprays): none, 1.6 litre distilled H_2O, Pro + Val in equal amounts, mixed amino acids in equal amounts (Ala, Arg, Gly, His, Lys, Met, Phe), Gly and gelatin. For the last four treatment levels, amino acids or protein were dissolved in 1.6 litre distilled H_2O with a concentration equal to 0.7534 g N (except Gly which equalled 7.534 g N). The treatment level was chosen to lie within the range of plant water stress responses and raise the estimated N standing crop by 10%, based on 84 g m^{-2} standing crop plant biomass at an estimated 2% total N.

The irrigation treatments were carried out on 9 July 1979 between 0700 and 1000 h; foliar spraying was done by hand on 10 July 1979 from 1230 to 1700 h. Except for the Gly treatment, which was 10 times more concentrated, the maximum possible individual amino acid concentration was 0.93% of plant dry matter compared with measured proline levels of 1% or greater for barley[14], Bermuda grass[15], tobacco[16], clover[21] and several halophyte species[11,17] under water stress. Proline can accumulate to as much as 10–20% of shoot dry weight in *Triglochin maritima*[17]. I estimated that less than one quarter of the spray from any of the treatments contacted the leaves due to low plant density at the site.

The dependent variable measured was percentage of leaves chewed on stems of *Agropyron smithii* with four intact leaves, a C_3 grass, and *Bouteloua gracilis* with five intact leaves, a C_4 grass, stratified by leaf position enumerated from youngest to oldest. Virtually complete collections of stems of these two species were gathered by hand on 6 September 1979. The total number of stems sampled for each of the foliar spray treatments is shown in Fig. 1. Only chewed leaves were counted—broken leaf tips were not recorded.

Chewing was affected by two amino acid treatments but was unaffected by irrigation or the remaining amino acid sprays (χ^2 test, $P<0.001$). Because irrigation treatments showed no influence on chewing ($P>0.3$) data were pooled across water levels in the subsequent analysis. Paired t-test analysis of percentage of leaves chewed by leaf position indicated

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