are widespread in Lepidoptera and perhaps other insect orders as well. The development of such an effective means of circumventing a rather formidable host plant resistance mechanism no doubt provides the black swallowtail with a distinct competitive advantage over other psoralen-susceptible insect herbivores.

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8. M. Berenbaum [*Science* **201**, 532 (1978)] has shown that the closely related southern armyworm (*S. eridania*) is highly susceptible to the phototoxic effects of linear furocoumarins and that this species does not thrive (for one reason or another) on plants that contain appreciable amounts of psoralens.
9. [^{14}C]Xanthotoxin was prepared by demethylation of xanthotoxin to xanthotoxol, and subsequent remethylation with [^{14}C]methyl iodide. The resulting preparation (9.86 mCi/mmole) was purified on TLC to > 99 percent radiochemical purity.
10. Most *S. frugiperda* larvae were reluctant to consume xanthotoxin-treated parsley, but this refusal was overcome when the insects were starved for 2 hours before treatment.
11. Samples were diluted with water, acidified with HCl, and extracted at least three times with ethyl acetate. The ^{14}C in all phases (including extracted tissue and excreta slurries) was quantified by liquid scintillation counting (LSC). The organic extracts were concentrated and applied to silica TLC plates that were developed in a solvent system of ethyl acetate, methanol, and glacial acetic acid (150:50:2). Radioactive components were visualized by autoradiography (x-ray film) and quantified by LSC.
12. Gut contents from individual, freshly dissected *P. polyxenes* larvae were incubated with 5 μg of [^{14}C]xanthotoxin in 2.0 ml of phosphate buffer (*p*H 7.8) at 32°C under either nitrogen or carbon dioxide gas.
13. Thin-layer chromatography R_f values in the ethyl acetate, methanol, and glacial acetic acid solvent system were as follows: xanthotoxin (0.67), metabolite **1** (0.46), metabolite **2** (0.12), minor metabolites (0.51, 0.26).
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17. Some insect species may use behavioral responses to circumvent the toxic effects of plant photosensitizers. Certain leaf rolling microlepidopteran larvae that feed on photosensitizer-containing hosts are thought to utilize leaf rolling behavior primarily to avoid light.
18. We thank Jean Person for technical assistance.

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Magnesium Deficiency–Induced Spasms of Umbilical Vessels: Relation to Preeclampsia, Hypertension, Growth Retardation

Abstract. *Isolated umbilical arteries and veins, obtained from normal women at the end of pregnancy, were incubated in Krebs-Ringer bicarbonate solution and exposed to magnesium at concentrations ranging from 0 to 9.6 millimoles per liter. The basal tension of the vessels increased when magnesium was withdrawn and decreased when the concentration of magnesium was raised. Absence of magnesium in the medium significantly potentiated the contractile response of the vessels to bradykinin, angiotensin II, serotonin, and prostaglandin $F_{2\alpha}$. It appears that magnesium deficiency may be responsible for spasms of umbilical and placental vasculature. Our findings may provide a rationale for why magnesium sulfate is an effective therapy in preeclamptic syndromes in pregnant women.*

The symptoms of preeclampsia in pregnant women include hypertension, edema, increased vascular reactivity to pressor substances, uteroplacental changes (ischemia, infarctions), cerebral and visual disturbances, and coagulation defects (*1, 2*). In addition, almost half of all fetuses delivered from preeclamptic mothers exhibit growth retardation. It is estimated that, worldwide, the syndrome kills 5 million pregnant women and fetuses annually (*1, 3, 4*). Although preeclampsia has been known for centuries (*5*), its pathophysiology remains unresolved (*1–4, 6*). Its incidence is highest among indigent populations, perhaps because of malnutrition (*1, 5, 7, 8*). The preferred treatment for severe cases is parenteral administration of $MgSO_4$, which somehow restores normal blood pressure and vascular reactivity (*1, 2, 4, 6*).

Hypomagnesemia has been seen with preeclampsia (*9–12*). Normal pregnant women often show progressive hypomagnesemia during the last 2 months of pregnancy (*11–13*). According to recent surveys (*11, 14*), the dietary intake of Mg^{2+} among pregnant women worldwide has been steadily declining since around 1900, to the point where the Mg^{2+} balance of many is negative (*11, 12, 14*). Acute hypomagnesemia in animals and humans is often associated with increases in blood pressure and in peripheral vascular resistance (*11, 15*). Artificial lowering of the Mg^{2+} content of isolated peripheral, coronary, and cerebral vessels from rats, rabbits, dogs, and piglets induces rapid contractile responses (*15–17*). Acute hypermagnesemia reduces both spontaneous and drug-induced tone in these peripheral arteries and veins (*15–17*). Such evidence suggests that extracellular Mg^{2+} plays a role in regulating vasomotor tone.

There are reports that placentas from women with preeclampsia or eclampsia exhibit decreased Mg^{2+} and increased Ca^{2+} (*11, 18*). A higher than normal ratio of Ca^{2+} to Mg^{2+} has been shown to provoke vasospasm in coronary, cerebral, and certain peripheral blood vessels in animals (*15, 16*). It was once suggested that peripheral vasospasm might play a role in preeclampsia (*19*). To investigate the possibility that vasospasm of the umbilical arteries and veins can be produced by Mg^{2+} deficiency, we determined the influence of sudden Mg^{2+} withdrawal and hypermagnesemia (2.4 to 9.6 m*M*) on vascular tone and on vasoactive drug–induced responses in human umbilical arteries and veins.

Arteries and veins, obtained from the normal umbilical cords of 34 pregnant women at full-term spontaneous delivery, were helically cut and set up isometrically (*20*). The tissues were equilibrated in normal Krebs-Ringer bicarbonate solution (118 m*M* NaCl, 4.7 m*M* KCl, 2.5 m*M* $CaCl_2$, 1.2 m*M* KH_2PO_4, 1.2 m*M* $MgCl_2$, 10 m*M* glucose, and 25 m*M* $NaHCO_3$) and aerated with 95 percent O_2 and 5 percent CO_2 at 37°C. After 3 hours of incubation under tension, the vessels were exposed randomly to normal (1.2 m*M*), zero, and high (2.4, 4.8, and 9.6 m*M*) concentrations of Mg^{2+} and their contractile responses were measured with Grass FT-03 force-displace-

Table 1. Influence of extracellular Mg^{2+} on the basal tone of human umbilical arteries and veins. The number of donors represented in each group is given in parentheses. Tissues were first incubated in Krebs-Ringer bicarbonate solution containing 1.2 m*M* Mg^{2+}. All the values (means ± standard errors) differ significantly from those for the 1.2 m*M* Mg^{2+} solution ($P < 0.01$, paired *t*-test). Minus signs signify relaxation.

Mg^{2+} (m*M*)	Tension (mg)	
	Arteries	Veins
0	1436.8 ± 295.2 (34)	2157.6 ± 395.0 (33)
2.4	−110.2 ± 28.2 (6)	−250.0 ± 54.2 (6)
4.8	−250.0 ± 56.2 (6)	−516.7 ± 124.8 (6)
9.6	−576.3 ± 61.5 (19)	−655.2 ± 62.1 (19)

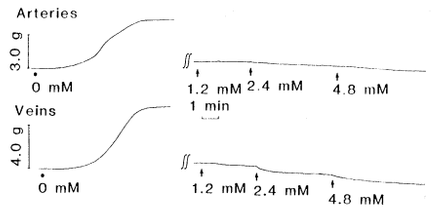

Fig. 1. Effects of extracellular concentration of Mg^{2+} on the basal tone of isolated umbilical arteries and veins. Dots at left indicate the point at which Krebs-Ringer bicarbonate solution containing Mg^{2+} was replaced by Mg^{2+}-free solution; the traces on the right show the results obtained as progressively higher concentrations of Mg^{2+} were added (at arrows) to vessels that had been incubated in Krebs-Ringer bicarbonate containing 1.2 m*M* Mg^{2+}. Vertical bars represent tension. The contractile responses were maintained for 45 to 60 minutes.

ment transducers and recorded on a Grass model 7 polygraph. The effects of several vasoactive drugs on these responses were then determined.

Sudden withdrawal of extracellular Mg^{2+} resulted in a rapid increase of tension in the umbilical arteries and veins of 30 of the 34 women (Fig. 1 and Table 1). In contrast, sudden increases in Mg^{2+} resulted in a concentration-dependent relaxation in all vessels tested (Fig. 1 and Table 1). A similar direct influence of extracellular Mg^{2+} on vasomotor tone was previously demonstrated in isolated arteries, arterioles, and portal veins from various mammalian species (*15, 16*). These effects cannot be attributed to osmolarity differences, inhibition of Na^+- and K^+-dependent adenosine triphosphatase activity, or release of endogenous neurohumoral agents from the arterial or venous walls (*16, 21*).

Withdrawal of Mg^{2+} potentiated the constrictor actions of bradykinin, serotonin, angiotensin II, and prostaglandin $F_{2\alpha}$ (Fig. 2 and Table 2). Most of these enhanced responses in the absence of extracellular Mg^{2+} were much greater in magnitude than those observed previously in rat, rabbit, and dog blood vessels exposed to Mg^{2+}-free medium (*16*). In contrast, elevated Mg^{2+} obliterated the constrictor action of bradykinin, prostaglandin $F_{2\alpha}$, and angiotensin II and decreased the action of serotonin (Fig. 2 and Table 2). All these agents are circulating vasoconstrictor substances in the umbilical and placental vasculature (*20*) and often demonstrate unexplained increased pressor responses in preeclamptic women. It is clear that, although these agents may demonstrate hardly any contractile effects in the presence of Mg^{2+}, they all have potent spasmogenic actions in the absence of Mg^{2+}.

There is extensive evidence that the effects of the concentration of extracellular Mg^{2+} on vascular tone are reflections of this metal's influence on membrane permeability to Ca^{2+}, binding, and translocation and on membrane stability (*15–17*). Studies of animal blood vessels show that Mg^{2+} sites in the membrane can act physiologically to regulate entry and exit of Ca^{2+} (*16, 22*). Lowering the concentration of extracellular Mg^{2+} increases total exchangeable and intracellular Ca^{2+} fractions in blood vessels (*16, 22*). Such findings indicate that when extracellular Mg^{2+} is lowered Ca^{2+} influx is enhanced, causing contraction (*16, 22*). The potentiated contractile responses to vasoactive agents in the absence of Mg^{2+} are probably also due to enhanced influx and translocation of Ca^{2+} into the vascular muscle cells. This is consistent with (i) the decreased ratio of Mg^{2+} to Ca^{2+} in the placentas of women with preeclampsia and eclampsia (*11, 18*), (ii) the progressive decline in dietary intake of Mg^{2+} in pregnant women since around 1900 (*11, 14*), (iii) the frequent association of hypomagnesemia with preeclampsia and eclampsia (*9–13*), (iv) the increased vascular reactivity to pressor substances (*1, 2*), and (v) the occurrence of uteroplacental ischemia in preeclampsia (*1, 2*).

Table 2. Influence of extracellular Mg^{2+} on vasoactive drug–induced contractions of human umbilical arteries and veins. Values are means ± standard errors. The number of donors represented in each group is given in parentheses. N.D., not done.

Mg^{2+} (m*M*)	Tension (mg)	
	Arteries	Veins
	Bradykinin (4.7×10^{-12}M)	
0	798.8 ± 186.9* (8)	3729 ± 649.6* (8)
1.2	18.8 ± 13.1 (8)	2275 ± 419.4 (8)
4.8	0* (8)	0* (8)
	Serotonin (2.5×10^{-9}M)	
0	1375 ± 128.8* (6)	4855 ± 978.4* (6)
1.2	650.0 ± 63.6 (6)	675 ± 78.6 (6)
4.8	150.6 ± 42.4* (6)	65.6 ± 14.6* (6)
	Prostaglandin $F_{2\alpha}$ (2.8×10^{-7}M)	
0	375.0 ± 41.6 (4)	N.D.
1.2	50.0 ± 28.4 (4)	N.D.
4.8	0 (4)	N.D.
	Angiotensin II (2.5×10^{-8}M)	
0	585.6 ± 114.4* (5)	196.5 ± 68.5* (7)
1.2	175.8 ± 58.6 (5)	28.8 ± 9.6 (7)
4.8	0* (5)	0* (7)

*$P < 0.01$, paired *t*-test.

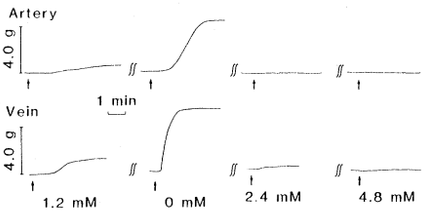

Fig. 2. Responses of an umbilical artery and a vein to stimulation with bradykinin ($4.7 \times 10^{-12}M$), added (at arrows) to Krebs-Ringer bicarbonate containing various concentrations of Mg^{2+}.

A progressive hypomagnesemia throughout pregnancy (due to deficits in dietary intake of this mineral, alterations in Mg^{2+} and Ca^{2+} exchange sites at membranes of umbilical and placental vessels, or defects in Mg^{2+} metabolism) could produce progressive vasoconstriction, resulting in spasms of umbilical and placental arteries and veins and of other peripheral vessels in the pregnant mother and fetus. Adequate Mg^{2+} intake or metabolism would prevent these vascular events. Such a rationale would explain why $MgSO_4$ effectively lowers arterial blood pressure and returns vascular reactivity to normal in preeclamptics and why timely Mg^{2+} therapy greatly reduces the likelihood of fetal growth retardation (*1, 11*). Our findings also may explain the high incidence of infant mortality and congenital malformations in geographic regions with soft drinking water and Mg^{2+}-poor soil (*23*). Lastly, the familial tendency to develop preeclampsia and eclampsia is consonant with our findings, since the level of Mg^{2+} in certain tissues is under genetic control (*24*).

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Clustered Third-Base Substitutions Among Wild Strains of *Escherichia coli*

Abstract. *Nucleotide sequences of translated regions of the* trp *operon in 12 wild strains of* Escherichia coli *reveal striking uniformity among eight strains (suggesting recent common ancestry and supporting the importance of periodic selection in natural populations) and clustered substitutions in four strains (implicating events affecting runs of nucleotides).*

Nucleotide sequences of homologous regions in different genera of bacteria (or in any long-separated genomes) may be expected to reveal adaptive differences, adaptive uniformity, and neutral variation. The neutral variation may represent both single and successive events at a single nucleotide site, given sufficient time of divergence from a common ancestor. In contrast, neutral differences observed between DNA sequences of independent isolates of a given species are likely in general to represent single events only. With this distinction in mind, we present some recent observations.

Nucleotide sequencing of the coding regions of the entire *trp* operon in *Escherichia coli* and *Salmonella typhimurium* has revealed third-base differences in 40 to 50 percent of all codons, but far fewer first- and second-base differences (*1*). These third-base differences appear to be distributed randomly, with the exception of one 21-codon stretch (as discussed below).

We have obtained from each of 12 wild *E. coli* strains a fragment of DNA extending at least from nucleotide 4343 in *trpC* through nucleotide 5990 in *trpA* (*2*). This 1648-bp segment, which includes all of *trpB*, corresponds to a deletion in the *trpCBA* region borne on a plasmid and filled by transduction (*3*). These fragments have been sequenced in large part and compared with *E. coli* strain K12. In contrast with the intergeneric (*Escherichia-Salmonella*) differences just mentioned, the nucleotide differences among the wild *E. coli* strains are highly clustered (Tables 1 and 2). Eight strains are remarkably similar: each differs from strain K12 by no more than one base in a thousand. Three other strains, identical in sequence so far, differ from K12 at ten sites. A final strain, 202I, differs from K12 at 44 sites. Thus the differences are not randomly distributed among these strains. Some base substitutions are clustered spatially also. For example, strain 45E has a set of four substitutions (4963 to 4975) in five consecutive codons in *trpB*. Strain 202I has the same set, as well as three more in a four-codon stretch 24 bases further along in *trpB*. In addition to these clusters there are other scattered substitutions, some shared between 45E and 202I and some not. Of the ten nucleotides in 45E that differ from K12, seven are shared by 202I. At the

Table 1. Characteristics of the wild *E. coli* strains tested. Bases were sequenced from positions 4321 through 5989.

Item	K12-like								45E-like			202I
Strain*	39A	66B	191F	200L	200M	201C	210J	217T	45E	70B	224H	202I
Source†	H, I	H, I	L, Z	P_1, N_1	P_1, N_1	P_2, N_1	A, Z	G, N_2	A, Z	R, Z	F, Z	A, Z
Number of bases sequenced	1300	1300	1200	1300	1400	1300	1100	1300	1300	950	1400	1300
Base change versus *E. coli* K12‡	4779 C → T	None	None	5922 C → A	None	5266 C → T	5114 T → G	5719 G → A	(10)	(10)	(10)	(44)
Electromorph§												
ADH	F	F	+	+	F	F	F	+	F	+	+	0
MDH	+	+	+	+	+	+	+	+	+	+	+	+
G6PD	+	+	+	+	+	+	F	+	+	+	+	+
6PGD	R	S	F	+	F	+	C	S	T	+	+	+
Thermostability class, 6PGD¶	S_4	S_3	S_3	+	S_4	+	S_2	+	+	N.T.	N.T.	N.T.

*Strain numbers follow (*2*). †Source symbols: H, human; L, leopard; P_1, domestic pig 1; P_2, domestic pig 2; A, Celebes black ape; G, domestic goat; R, lowland gorilla; F, giraffe; I, Iowa (State Hygienic Laboratory); N_1, first Indonesian sample; Z, Woodland Park Zoo, Seattle; N_2, second Indonesian sample. ‡Position and type. §ADH, alcohol dehydrogenase; MDH, malate dehydrogenase G6PD, glucose-6-phosphate dehydrogenase; 6PGD, 6 phosphogluconate dehydrogenase; mobilities [see (*2*)] are 0, no activity; C, R, S, T, +, and F are listed in the order of increasing mobility. ¶Thermostability categories: +, most common class; S, temperature-sensitive class, with subscript number rising with increasing sensitivity. Class differences are roughly 1 kcal/mole in activation energy of catalytic inactivation (*7*). Strains marked N.T. were not tested, but 95 percent of all 6PGDH "+" electromorphs tested were in the "+" thermostability class (*7*).