ations that are uniquely detectable by RNase A cleavage, most changes that can be found by Southern or Northern blotting are easily identified, which brings the total fraction of LN mutations that can be diagnosed at the nucleic acid level to 50% (5/14 plus 15%).

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Isotopic labeling was achieved with 100 μCi of [^{32}P]guanosine triphosphate (650 Ci/mmol, diluted from 3000 Ci/mmol) (Amersham). Total cellular RNA (200 μg) [J. M. Chirgwin, A. E. Przybyla, R. J. MacDonald, W. J. Rutter, *Biochemistry* **18**, 5294 (1979)] from HeLa or lymphoblast cell lines transformed with Epstein-Barr virus was mixed with 5 × 10^6 cpm of probe in 200 μl of 80% formamide, 400 m*M* NaCl, 40 m*M* Pipes, *p*H 6.7, heated to 85°C for 5 minutes, and hybridized overnight at 65°C. The mixture was cooled to 25°C, diluted with 100 μl of 0.5*M* NaCl, and slowly applied to a 0.4-cm^2 piece of mAP that had been soaked in 0.5*M* NaCl for 5 minutes. The paper was washed four times for 15 minutes each in 0.5*M* NaCl at 25°C and heated to 65°C for 10 minutes in 180 μl of water to elute the HPRT mRNA:probe hybrid. The mAP was removed, the eluate chilled on ice, and 20 μl of 2.5*M* NaCl added. 100 μl of RNase A was added in 200 m*M* NaCl, 100 m*M* LiCl, 30 m*M* tris, *p*H 7.5, and 3 m*M* EDTA, and the samples incubated for 30 minutes at 25°C. Pretreatment of RNase A and the subsequent analysis of the RNase A cleavage products were according to Winter *et al.* (*4*). One half of the original reaction mixture (equivalent to 100 μg of total cellular RNA) was loaded in each lane. Autoradiographic exposures range from 12 to 16 hours, with one intensifying screen (Cronex) and Kodak X-AR5 x-ray film at −70°C. ^{32}P–end labeled Hae III-digested ΦX174 DNA fragments were employed as molecular size markers on the polyacrylamide gels.

10. R.A.G. is a research fellow and C.T.C. is an investigator of the Howard Hughes Medical Institute. We also thank J. T. Stout and P. I. Patel for valuable discussions and M. F. Browner for bringing mAP to our attention.

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Expression and Processing of the AIDS Virus Reverse Transcriptase in *Escherichia coli*

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The ability to express the genes of pathogenic human viruses, such as the acquired immune deficiency syndrome (AIDS) virus (also called human immunodeficiency virus) in bacterial cells affords the opportunity to study proteins that are ordinarily difficult or inconvenient to obtain in amounts sufficient for detailed analysis. A segment of the AIDS virus *pol* gene was expressed in *Escherichia coli*. Expression resulted in the appearance of reverse transcriptase activity in the bacterial cell extracts. The extracts contained two virus-related polypeptides that have the same apparent molecular weights as the two processed forms of virion-derived reverse transcriptase (p66 and p51). The formation of these two polypeptides depended on the co-expression of sequences located near the 5′ end of the *pol* gene, a region that is thought to encode a viral protease. This bacterial system appears to generate mature forms of the AIDS virus reverse transcriptase by a proteolytic pathway equivalent to that which occurs during virus infection of human cells.

THE STRUCTURE OF THE HUMAN immunodeficiency virus (HIV) genome (*1*) is similar to that of most retroviruses with respect to the placement of the *gag*, *pol*, and *env* genes (Fig. 1A). The HIV genome encodes several additional genes including *tat* and *art/trs* (*2*) as well as two open reading frames, A and B, which encode proteins of unknown function. By analogy to other retroviruses, expression of the HIV *pol* gene probably occurs by the occasional suppression of the *gag* translation termination codon thereby fusing the *pol* reading frame with the upstream *gag* reading frame (*3*).

The mammalian retrovirus *pol* gene typically encodes three protein products that express a total of four catalytic activities (*4*): a protease that cleaves the viral *gag* and *gag/pol* precursor polyproteins; the viral DNA polymerase [reverse transcriptase (RT)] with an associated ribonuclease H activity; and an integrase/endonuclease that functions in the integration of viral DNA into the host cell genome. The HIV *pol* gene is atypical with respect to other mammalian retrovirus *pol* genes in that it appears to encode four polypeptide products. Three of these *pol*-encoded polypeptides have been identified in HIV virions: two closely related forms of RT, p66 and p51, that differ in size (66 kD and 51 kD) but share a common amino terminus (*5, 6*); and a 34-kD protein (p34) that is the presumed integrase/endonuclease (*6*). The amino terminal sequences of these three polypeptides have been determined, thus permitting their assignment to specific regions of the *pol* gene open reading frame (*5, 6*). Like other mammalian retroviruses, the RT domain is encoded in the central portion, and the integrase/endonuclease is encoded in the 3′ portion of the *pol* gene open reading frame (Fig. 1B). Sequence comparisons (*1*) show that the remaining 5′ portion of the HIV *pol* gene encodes a polypeptide that shares homology with the protease domain of other avian and mammalian retroviruses.

Two plasmids were constructed containing HIV *pol* gene sequences under the control of the inducible *lac* promoter. The plasmids differ in the presence or absence of the presumed protease domain (Fig. 1C). The first plasmid, pBRT1prt$^+$, contains the protease and RT coding domains, starting with a Bgl II site located at the fifth codon of the *pol* open reading frame and terminating at an Eco RI site located in the middle of the integrase coding domain. The second plasmid, pBRT3prt$^-$, contains only the RT domain, starting with a Hinc II site located nine codons upstream of the position corresponding to the amino terminus of p66/p51 (*5, 6*) and terminating at the Eco RI site in the middle of the integrase coding domain.

Extracts were prepared from uninduced and induced bacterial cultures containing either the parental plasmid without a viral insert (pIBI20) or one of the pBRT plasmids. The presence of RT activity was detected by assaying bacterial cell extracts for RNA-dependent DNA polymerase activity with a homopolymeric polyribonucleotide template with a complementary oligodeoxy-

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ribonucleotide primer, poly(rC) · oligo(dG) (Table 1). Induction of the *lac* promoter resulted in the appearance of elevated levels of RT activity when the cultures contained plasmids encoding the enzyme either with (pBRT1prt$^+$) or without (pBRT3prt$^-$) the protease coding domain. Extracts made from cultures containing the pBRT1prt$^+$ plasmid had four- to fivefold higher levels of RT activity than did extracts from cultures with the pBRT3prt$^-$ plasmid (Table 1). Little RT activity was seen in cultures containing the parental plasmid without an insert.

To determine the size of the *pol* gene products in the induced bacterial cultures, we subjected the bacterial cell extracts to Western blot analysis. Equivalent amounts of protein from different extracts were fractionated in a polyacrylamide gel under denaturing conditions. The proteins were then transferred from the gel to a nitrocellulose filter, and the presence of *pol* gene–specific products was detected by using human serum that contained antibodies to HIV proteins (Fig. 2). Extracts made from bacterial cultures induced with IPTG and containing either of the pBRT plasmids showed evidence of protein that reacted with the human serum (Fig. 2, lanes 1, 4, and 8). Extracts made from the induced cells containing the pIBI20 plasmid and extracts from uninduced cells did not show evidence of any virus-related protein (Fig. 2, lanes 2, 5, and 6). In the case of pBRT3prt$^-$, the plasmid without protease sequences, the predicted product encoded by the plasmid has a molecular size of 84 kD. A product of this size was seen in extracts of induced pBRT3prt$^-$ cultures (Fig. 2, lane 1). However, most of the immunoreactive protein in this extract was present as a series of smaller sized species, suggesting that the primary translation product was unstable in *Escherichia coli* and subject to degradation by

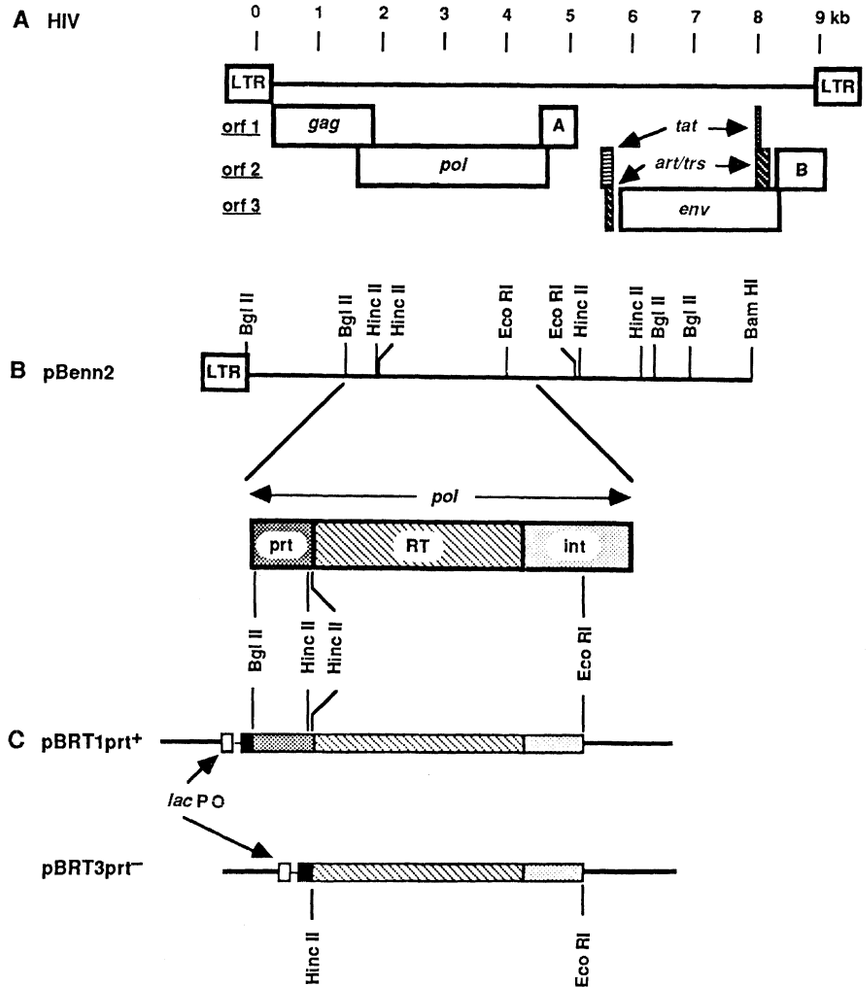

Fig. 1. (**A**) Schematic diagram of the AIDS virus genome. Boxes indicate the relative locations of the LTRs and the open reading frames (ORF) of the *gag*, *pol*, *env*, *tat*, and *art/trs* genes. Two additional ORFs are shown, A and B, which encode proteins of unknown function. (**B**) Physical map of the HIV sequences in the plasmid pBenn2 (*16*). This plasmid contains a partial copy of the HIV genome that begins with the left-hand LTR, includes all of *gag* and *pol*, and terminates at a Bam HI site in *env*. Most of the restriction endonuclease cleavage sites shown were determined experimentally; the rest are predicted from the published LAV DNA sequence (*1*). The *pol* ORF is expanded showing the relative locations of the domains encoding the protease (prt), reverse transcriptase (RT), and integrase/endonuclease (int). (**C**) Structure of *pol* gene segments inserted into the expression vector plasmid. The plasmid pBRT1prt$^+$ contains a Bgl II to Eco RI fragment of pBenn2 inserted between the Bam HI and Eco RI sites in the pIBI21 (International Biotechnologies Inc.) polylinker region. This plasmid expresses all of the prt and RT domains and a portion of the int domain. The plasmid pBRT3prt$^-$ contains a Hinc II to Eco RI fragment of pBenn2 inserted between the Hinc II and Eco RI sites in the pIBI21 polylinker region. This plasmid expresses all of the RT domain and a portion of the int domain. The shading of the prt, RT, and int regions are as in (B). The lactose operon promoter and operator signals (*lac* PO) direct the transcription of the adjacent *pol* gene sequences. The filled box to the right of *lac* PO indicates the location of sequences encoding a leader polypeptide derived from the vector polylinker DNA sequence. This leader polypeptide adds 29 amino acids and 32 amino acids, respectively, to the pBRT1prt$^+$ and pBRT3prt$^-$ *pol* gene translation products. An oligonucleotide encoding a translation termination codon was inserted at the Eco RI site in each of the pBRT plasmids.

Table 1. Measurement of RNA-dependent DNA polymerase activity in bacterial extracts. Bacterial cultures were grown at 37°C to a density of 0.3 to 0.4 (absorption at 600 nm) in M9 medium supplemented with 0.5% Casamino acids, 10 μg/ml of thiamine, and 50 μg/ml of ampicillin. Uninduced cultures were grown in M9 medium as above with the addition of 0.2% glucose. Cultures were induced by the addition of isopropylthio-β-galactoside (IPTG) to 5 m*M*, and the cultures were grown for an additional hour at 37°C. Bacterial cell extracts were prepared as described by Tanese *et al.* (*10*). Bacterial cells are collected by centrifugation, resuspended in 0.05*M* tris-HCl, *p*H 7.4, 0.6 m*M* EDTA, 0.375*M* NaCl, and then digested with lysozyme (1 mg/ml, 30 minutes at 0°C) and lysed by the addition of 0.2% NP-40. After the addition of 1*M* NaCl, the extracts are clarified by centrifugation and the supernatant is assayed for RT activity. The final extract volume was one-eighth of the culture volume. Reverse transcriptase activity was measured in 50-μl reactions containing 10 m*M* tris-HCl (*p*H 7.5), 8 m*M* $MgCl_2$, 10 m*M* dithiothreitol, 40 m*M* KCl, 20 m*M* NaCl, 0.16 μ*M* labeled deoxyguanosine triphosphate (1×10^6 cpm/pmol), 50 μg/ml of poly(rC) · oligo(dG), and 0.1 to 1.7 μg of extract protein. Samples (10 μl) were withdrawn at various times and the incorporation of [^{32}P]dGMP into DNA was assayed by the DEAE filter–binding technique and liquid scintillation spectrophotometry. The radioactivity incorporated was measured as 10^{-3} picomoles per minute per microgram of protein. Each experiment was repeated at least twice and the results of a typical experiment are shown.

Plasmid	[^{32}P]dGMP incorporated
pBRT1prt$^+$	
Uninduced	0.29
Induced	7.8
pBRT3prt$^-$	
Uninduced	0.07
Induced	1.7
pIBI20	
Induced	0.12

bacterial proteases. The predicted primary translation product of the pBRT1prt$^+$ *pol* gene fragment, which includes the presumed protease domain, is a 101-kD protein. Extracts of cells with this plasmid showed primarily two protein species of 66 kD and 51 kD (Fig. 2, lanes 4 and 8). None of the predicted 101-kD species and only trace amounts of species smaller than 51 kD were detected. The two pBRT1prt$^+$-specific polypeptides comigrated with two HIV virion polypeptides (Fig. 2, compare lane 3 with lane 4, and compare lane 7 with lane 8), and they also comigrated with the two related forms of RT that were purified from HIV particles (Fig. 2, compare lane 8 with lanes 9 to 11). To examine the smaller protein species present in HIV virions and in induced pBRT1prt$^+$ extracts, we fractionated the proteins in Fig. 2B in a gel with a higher percentage of polyacrylamide than that used for the proteins in Fig. 2A. The results suggested that the primary translation product encoded by pBRT1prt$^+$ was processed by the viral protease to give the same two species of RT that are present in mature HIV particles. We cannot exclude the possibility that the inclusion of the protease domain at the amino terminus of the *pol* gene product creates a more favorable substrate for a bacterial protease which is responsible for generating the 66-kD and 51-kD polypeptides; however, we consider this possibility unlikely.

The two related forms of HIV reverse transcriptase in mature virions (*5, 6*) could arise by any of several mechanisms, including differential protein modifications [for example, glycosylation such as occurs with the HIV *env* gene product gp120 (*7*)], differential messenger RNA (mRNA) splicing [as in the expression of the SV40 early gene region (*8*)], or differential proteolytic processing [as in the formation of the α and β forms of the Rous sarcoma virus RT (*9*)]. Our results suggest that the two virion forms of HIV reverse transcriptase (p66 and p51) arise as a consequence of differential proteolytic processing of a single precursor polypeptide since neither mRNA splicing nor the appropriate modification of the mature protein are likely to occur in bacterial cells.

Several attempts have been made previously to express retrovirus RNA–dependent DNA polymerases in bacteria. Enzymatically active forms of the viral DNA polymerase from both HIV and murine leukemia virus (MuLV) have been expressed in *E. coli* (*10–12*), and in the case of the MuLV protein the activity has been purified and characterized (*13*). Although the expression of a stable form of RT was achieved in the MuLV system, construction of a clone that directed the synthesis of a stable protein required progressive deletion of MuLV *pol* gene sequences from both the amino and carboxyl termini (*13*). The instability of the pBRT3prt$^-$-encoded 84-kD protein is typical of many eukaryotic proteins expressed in *E. coli*. In contrast, the p66 and p51 generated in cells expressing pBRT1prt$^+$ appears to be strikingly refractory to nonspecific proteolytic degradation. Perhaps the maturation of p66/p51 from a larger precursor protein by the normal proteolytic pathway allows the protein products to assume a conformation that is resistant to nonspecific proteolysis.

Proteolytic processing of the HIV *gag* gene product expressed in yeast has been detected previously (*14*). In this system, processing was blocked by a mutation within the 5′ terminus of the *pol* gene. These results showed that at least a portion of the viral protease is encoded within the *pol* gene region. Our results indicate that the entire catalytic domain of the protease is encoded within *pol*.

There are few examples of bacterially expressed eukaryotic proteins that undergo proper proteolytic processing. One example is the poliovirus protease P3-7c, which is normally generated by autocatalytic processing of a polyprotein precursor. When sequences encoding the poliovirus protease and adjacent sequences were expressed in bacteria, the mature protease was formed by autocatalytic cleavage of the precursor protein at flanking protease recognition signals (*15*). By comparison, the primary pBRT1prt$^+$-encoded polypeptide undergoes several cleavage events to give rise to at least two independent products. Both the poliovirus protease and the HIV protease function in the absence of any eukaryotic factors, indicating that sufficient information is encoded in the primary protein sequence of the precursor polypeptide to permit accurate folding of both the protease and its target substrate.

The expression and apparent processing of the HIV *pol* gene products in bacteria provides a convenient system for the analysis of both the RT and protease activities. In addition, the stable forms of RT generated in this system are a potential source for large-scale purification of these proteins. Both the RT and the protease are targets for inhibitors of virus replication. It is likely that a detailed understanding of each of the catalytic activities encoded by the HIV *pol* gene will provide new opportunities for therapeutic intervention.

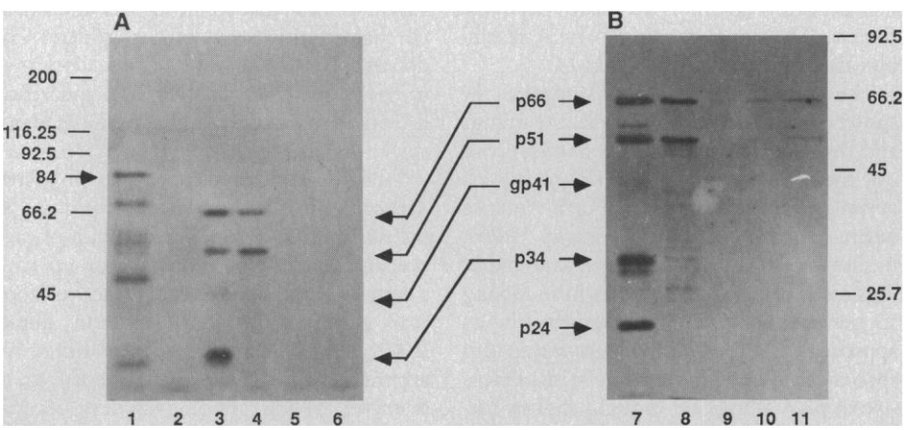

Fig. 2. Western blot analysis of HIV *pol* gene polypeptides synthesized in bacteria. Bacterial protein extracts were prepared as described in Table 1. The proteins in the extracts were denatured and fractionated by sodium dodecyl sulfate (SDS)–polyacrylamide gel electrophoresis. The fractionated proteins were electrophoretically transferred to nitrocellulose membranes and reacted with serum obtained from an AIDS patient. Identification of bands reacting with the human serum was accomplished by reaction with a second antibody conjugated with alkaline phosphatase (Promega Biotec). The arrows between (A) and (B) identify HIV virion proteins (lanes 3 and 7). Mobilities of size standards (in kilodaltons) are indicated to the left and right of (A) and (B), respectively. (**A**) Protein extracts were from cells containing pBRT3prt$^-$ and induced with IPTG, lane 1 (the arrow to the left identifies the 84-kD primary translation product); uninduced cells containing pBRT3prt$^-$, lane 2; purified HIV virions, lane 3; cells containing pBRT1prt$^+$ and induced with IPTG, lane 4; uninduced cells containing pBRT1prt$^+$, lane 5; and cells containing pIBI21 and induced with IPTG, lane 6. Proteins were fractionated in an 8% SDS-polyacrylamide gel. (**B**) Protein extracts were from purified HIV virions, lane 7; cells containing pBRT1prt$^+$ and induced with IPTG, lane 8; and increasing amounts of partially purified HIV reverse transcriptase (*17*), lanes 9 to 11. Proteins were fractionated in a 10% SDS-polyacrylamide gel.

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A Sheep in Wolf's Clothing: Tephritid Flies Mimic Spider Predators

MONICA H. MATHER AND BERNARD D. ROITBERG

Mimicry where prey resemble predators to avoid predation is unusual. Snowberry flies, *Rhagoletis zephyria* Snow, possess striped wing patterns that resemble the legs of jumping spiders. Observations and comparisons of responses of the jumping spider *Salticus scenicus* (Clerck) to conspecifics, snowberry flies, and other prey flies showed that snowberry flies can avoid predation by jumping spiders through spider mimicry. The mimicry effect was decreased by obliterating snowberry fly wing stripes.

AGGRESSIVE MIMICRY, WHEREIN predators mimic their prey to facilitate prey capture, and Batesian mimicry, wherein harmless species copy coloration patterns of a protected (for example, toxic) species, have been well demonstrated for several biological interactions (*1, 2*). However, the case in which prey mimic their predators to avoid predation is rarely reported. We investigated the possibility that tephritid fruit flies mimic one of their predators, jumping spiders. Since jumping spiders are territorial and tend to avoid conspecifics (*3*), flies mimicking them might avoid predation. The obvious similarity between these flies and jumping spiders arises from wing banding patterns and wing waving behavior common to many tephritid species. The wing banding pattern resembles that of spider legs (Fig. 1) (*4*).

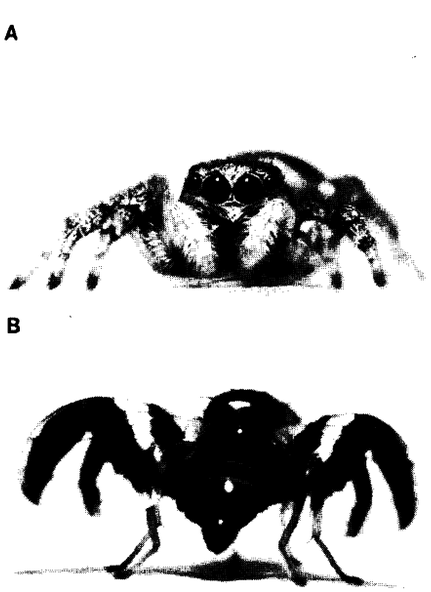

Fig. 1. (**A**) Front view of zebra spider *Salticus scenicus* and (**B**) posterior view of snowberry fly *Rhagoletis zephyria*. Note similarity between spider legs and fly wing markings.

The principal species of our study was the snowberry fly *Rhagoletis zephyria*, the mimic, and the common zebra spider *Salticus scenicus*, the model and predator. *Rhagoletis zephyria* is found throughout North America where snowberry, *Symphoricarpos albus*, bushes grow. The characteristic wing markings are common to all species in its sibling species complex (*5*). When disturbed by an approaching object, individuals within this species complex adopt a characteristic defensive display. Wings are brought slightly forward and a jerky side-to-side dance, similar in appearance to the gait of a jumping spider, is performed. Generally, the display will proceed until the approaching object withdraws or the fly leaves its position. The display dance is also employed in courtship and agonistic encounters between conspecific flies (*6*).

Zebra spiders are common throughout North America, where they inhabit walls, fences, bushes, and trees and pursue prey that include flies, moths, stinkbugs, and aphids; these can vary in size from a fraction of to more than twice a spider's size (*7*). Jumping spiders capture their prey by first stalking those that they encounter within their visual field (*8*). Stalking spiders crouch and crawl "cat-like" toward their prey and, once within striking range, pounce upon and immobilize them.

To conduct our studies, snowberry flies were obtained from naturally infested snowberry bushes during 1985 (*9*), and zebra spiders were collected from building walls, wooden railings, and fence posts around the Simon Fraser University campus from May to August 1986. After capture, each spider was fed a single onion fly, *Hylemya antigua*, housed in a glass jar and then starved for 2 days before testing.

To test whether or not snowberry flies mimic jumping spiders we performed a series of observational experiments within plexiglas arenas fitted with Plexiglas "observation domes" that confined prey and isolated them within view of the spiders. The domes were of sufficient size to allow prey to move freely within them and apparently provided a clear view of prey to the predator spiders (Fig. 2).

Individual spiders were released in arenas harboring single prey that were held within the observation dome. The behavior of each predator spider was recorded for up to 5 minutes or until it vacated a "reactive" zone that surrounded the observation dome. Based upon preliminary observations, we arbitrarily defined a reactive zone of 7 cm in diameter. When jumping spiders become aware of potential prey or conspecifics, they generally cease movement and then orient toward the object. Following this, stalking behavior begins (*8*) or the spiders either turn away and leave, or flee in an attempt to escape (*7*). The latter response is common during encounters with conspecifics, particularly since first inhabitants of territories, regardless of size, are dominant (*3*). Occasionally, when close encounters do occur,

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