type T cells was comparable to that observed on day 5 (Fig. 4B). The differential effects on effector and naïve T cells by PEP deficiency may reflect the coregulated expression of PEP and the closely related PTP-PEST during T cell differentiation (*14*, *21*). Additional studies of mice deficient in both PTPases will help to test this hypothesis.

The enhanced immune functions observed in $pep^{-/-}$ T cells were also associated with spontaneous development of germinal centers (GCs) in the spleens and Peyer's patches of these animals (Fig. 4E). Wild-type mice generally demonstrate small numbers of immature GCs (*22*). In contrast, $pep^{-/-}$ animals demonstrated increased numbers of large, well-formed GCs, with a corresponding increase in the numbers of GL-7^+ B cells, immunoglobulin $IgG1^+$ foci, and intermediate CD21 ($CD21^{int}$) $CD23^+$ follicular B cells (*14*). Analysis of signaling mediated by the B cell–antigen receptor (BCR), however, revealed minimal intrinsic biochemical and functional alterations in splenic B cells derived from $pep^{-/-}$ mice (fig. S4, A to C). Complementation studies with adoptive transfer experiments using $pep^{-/-}$ and wild-type T and B cells may reveal additional functions of PEP within the B cell compartment. Nonetheless, GC formation in $pep^{-/-}$ mice depended on cooperation between T and B cells, as administration of a blocking CD40L-specific mAb disrupted GC formation (*14*), demonstrating that the B cell effects were, in part, secondary to enhanced $pep^{-/-}$ T cell functions.

Germinal centers serve as important sites for immune dysregulation in the pathogenesis of autoimmune disorders (*22*), and spontaneous GC formation has been observed in a number of mouse strains that develop autoimmune disorders or have altered B cell function (*23*–*26*). Although $pep^{-/-}$ mice demonstrated increased serum levels of IgG1, IgG2a, and IgE, neither young nor old $pep^{-/-}$ mice demonstrated any increased incidence of autoantibodies or evidence of autoimmune-mediated organ damage, as compared with wild-type littermates (Fig. 4, F and G) (*14*). This uncoupling of spontaneous GC formation from autoimmune stigmata indicates a stepwise requirement for immune cell dysregulation for the development of clinical manifestations of autoimmune disorders. Because PEP deficiency does not appear to affect the deletion of autoreactive T cells, additional defects in central or peripheral tolerance are likely required as initiators of some autoimmune disorders. In the context of these initiating immune defects, enhanced expansion of autoreactive effector/memory T cells, as manifested by PEP deficiency, could culminate in clinical autoimmunity.

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A Predator Unmasked: Life Cycle of *Bdellovibrio bacteriovorus* from a Genomic Perspective

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Predatory bacteria remain molecularly enigmatic, despite their presence in many microbial communities. Here we report the complete genome of *Bdellovibrio bacteriovorus* HD100, a predatory Gram-negative bacterium that invades and consumes other Gram-negative bacteria. Its surprisingly large genome shows no evidence of recent gene transfer from its prey. A plethora of paralogous gene families coding for enzymes, such as hydrolases and transporters, are used throughout the life cycle of *B. bacteriovorus* for prey entry, prey killing, and the uptake of complex molecules.

Bdellovibrio bacteriovorus is a highly motile delta-proteobacterium that preys on other Gram-negative bacteria (*1*). *Bdellovibrio* are ubiquitous in nature, having been discovered in a wide variety of environments that include both aquatic and terrestrial habitats as well as mammalian intestines (*2*). Their prey includes plant, animal, and human pathogens (*3*). Thus, studying the molecular mechanisms of *Bdellovibrio*'s large complement of degradative enzymes gives insight into those targets in prey cells that have proven to be evolutionarily successful points of attack and offers cues for the design of antimicrobial agents.

Here we report the complete genome sequence of *B. bacteriovorus* strain HD100, which can only grow in the presence of prey. Despite the small dimensions of *Bdellovibrio* cells (0.2 to 0.5 μm wide and 0.5 to 2.5 μm long), its genome consists of 3,782,950 base pairs (bp) on a single circular chromosome and is predicted to code for 3584 proteins (fig. S1). No evidence was found for additional episomes, with the exception of a single copy of the novel IS

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element ISBba777 and a previously unknown *Bdellovibrio* prophage (Table 1). Despite having ready access to the prey's genetic information, no recent prey-derived horizontal gene transfer can be observed. Such a transfer from predator to prey should have resulted in regions in the *B. bacteriovorus* genome with GC content higher than the average, because the range of soil prey bacteria that *Bdellovibrio* was originally isolated from all have a higher than average GC content. The four regions of the genome that do deviate from the average GC content are high in AT and code for ribosomal, lipopolysaccharide (LPS) synthesis, prophage, and restriction modification genes, which in general are higher in AT in any bacterial genome (Table 1).

Of the predicted coding sequences, 55% can be assigned to a putative function by their homology to entries in the public database without suggesting a particular phylogenetic relationship to any of the completed microbial genomes. Eleven percent of the predicted open reading frames (ORFs) have homology to proteins of unknown function, whereas 34% cannot be assigned any function (Table 1, fig. S2, and table S1). In addition to the protein-coding genes, 36 transfer RNA (tRNA) genes were found, together with two ribosomal RNA (rRNA) clusters, each consisting of one copy of 5*S*, 16*S*, and 23*S* genes (Table 1).

Published experimental evidence [(*3*) and references therein] and the predicted gene content model the predatory life cycle of *Bdellovibrio* in eight stages (designated I through VIII) (fig. S3).

In attack phase (stage I), *B. bacteriovorus* swims at high speed using a single sheathed polar flagellum with a characteristic dampened filament waveform (*4*). Six clusters of motility and flagellar synthesis genes were found together with six copies of flagellin genes at four independent loci (supporting text). The detection of prey does not seem to depend on genes for the reception of homoserine-lactones generated by dense prey populations, nor does *Bdellovibrio* seem to be capable of producing known quorum-sensing compounds by itself.

Once *B. bacteriovorus* has collided with a prey cell, it remains reversibly attached to it for a short "recognition" period (*5*), after which it becomes irreversibly anchored via the pole opposite the flagellum (stage II). Multiple candidate genes for adhesion are found in the genome. Besides passive protein-protein and LPS-LPS interactions between outer membrane components, we suggest that active adhesion also occurs, based on the presence of multiple pili genes in *Bdellovibrio*. This mechanism combines adhesion and prey invasion. At least four clusters of *pil* genes were found on the chromosome (table S2), as were numerous dispersed *pil* genes coding for type IV pili (TFPs); this is the fourth highest number reported for any bacterial genome to date. TFPs or fimbriae perform multiple functions in many bacterial cells, such as protein secretion, adherence, DNA uptake, and twitching motility, depending on the composition of their central pilin fiber (*6*). Pilus tips are known to hold diverse sets of adhesive biopolymers that are specific to different surfaces (*6*). *Bdellovibrio* pili can be discerned at the prey interaction pole in electron micrographs (*7*). We think that a set of twitching-type pili pulls *B. bacteriovorus* through a previously generated penetration pore (see below) in the outer membrane, while attached to the inner side of the prey peptidoglycan. Squeezing of bacteria through a pore narrower than a bacterial cell has recently been shown to occur with TFPs in an in vitro assay (*8*).

A previously identified prey interaction (*hit*) locus (*9*) lies in a section of a *pil* and adherence gene cluster (*6*) (Fig. 1). Mutations in this locus were shown to be characteristic of prey independence; however, no function could be assigned to any ORF at this locus. Our analysis shows that three of the four formerly predicted *hit* locus ORFs (*9*) are inaccurate, with only one of the ORFs corresponding to a predicted gene from this analysis (Bd0108, Fig. 1). No putative function can be assigned to the previously reported hypothetical protein encoded by *hit*; however, *hit* is clearly part of a transcriptional unit together with a gene coding for a cell wall–associated protein with a cellulose-binding domain (*wapA*, Bd0109), the flagellar pilus assembly genes *tadA* (Bd0111) and *tadB* (Bd0110), and additional *pil* genes that encode structural elements of a type IV pilus. *Tad* and *pil* genes have been shown to be essential for tight adherence and colonization (*10*).

Before invasion of the prey cell can be achieved, *B. bacteriovorus* generates a small opening in the prey cell's outer membrane and peptidoglycan layer (stage III), which is ultimately resealed. A mixture of hydrolytic enzymes is applied in a locally targeted manner that prevents excessive damage to the prey and counters diffusion. Candidate genes for this function include serine (74), cysteine (8), and aspartate (1), as well as metal-dependent (53), proteases (table S3). There is also a burst of glycanase activity [previously reported (*11*)], which solubilizes prey peptidoglycan early in invasion and is sustained throughout the predatory cycle.

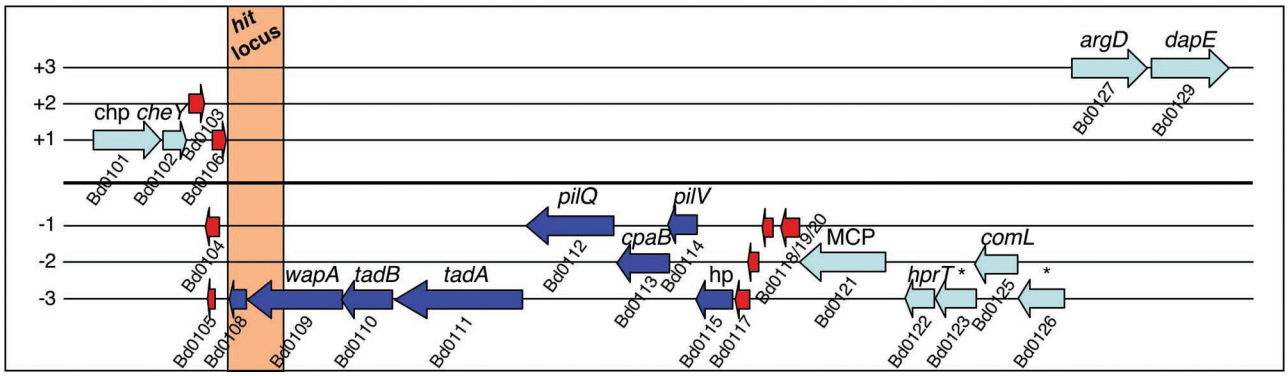

Fig. 1. The host interaction locus (*hit*) of *B. bacteriovorus*. The previously described *hit* locus (host interaction locus, orange bar) (*9*), consisting of a 950-bp sequence, is congruent with a section of the *B. bacteriovorus* genome that is predicted to code for pilus and adherence genes. The genes in this cluster are likely to functionally interact as structural elements of a pilus and are predicted to be transcribed as one transcriptional unit. The previously predicted gene *hit* corresponds to the ORF Bd0108 in the *B. bacteriovorus* HD100 genome and is part of the pilus and adherence transcriptional unit, together with a gene *wapA* coding for a cell wall–associated protein with a cellulose-binding domain (Bd0109), the flagellar pilus assembly genes *tadA* (Bd0111) and *tadB* (Bd0110), and additional *pil* genes of the type IV pilus. The *hit* locus containing the *tad* and *pil* gene cluster seems to have been inserted in between two chemotaxis genes (*cheY*, Bd0102; and *mcp*, Bd0121), because they are flanked on both sides by large potentially noncoding regions (red arrows) for which only short artificial or hypothetical ORFs could be found. The predicted ORFs of this region were assigned the following putative functions: chp, (conserved) hypothetical protein; WapA, cell wall–associated protein with a cellulose-binding domain; tad, tight adherence Flp pilus-assembly proteins TadB and TadA; pil, pilus assembly; cpaB, Flp pilus-assembly protein CpaB; MCP, methyl-accepting chemotaxis protein; hprT, hypoxanthine-guanine phosphoribosyltransferase; *, tetratrico peptide repeat domain–containing protein; comL, competence lipoprotein; argD, acetylornithine/succinyl-diaminopimelate aminotransferase; dapE, succinyl-diaminopimelate desuccinylase.

Upon entry into the prey periplasm, *B. bacteriovorus* sheds its flagellum (stage III). Soon afterward it starts cellular events such as DNA replication and the synthesis of cellular biopolymers (stage IV) (*12*).

Once *B. bacteriovorus* has entered the periplasm, it alters the cell shape of its prey, forming a joint structure termed the bdelloplast (stage V). The previously rod-shaped prey becomes rounded by a process believed to involve the hydrolysis of peptide crosslinks and glycanase activity (*13*). Nevertheless, it has been shown that the bdelloplast is osmotically stable (*11*), which suggests that the structural integrity of the prey's peptidoglycan is maintained. Several genes for glycan-modifying enzymes, such as soluble and membrane-bound lytic murein transglycosylases (8 and 1) and a membrane-bound effector of murein hydrolases, are found in the genome (table S3).

At this stage, *B. bacteriovorus* begins to extract and ingest solutes from the prey's cytosol via a broad range of transport systems from both the adenosine triphosphate (ATP)–binding cassette (ABC, 147) and major facilitator superfamily (MFS, 97) classes of transporters (figs. S4 and S5 and table S4). Only 7 out of 127 bacterial genomes have been reported so far to code for even higher numbers of ABC-type transporter genes. *B. bacteriovorus* has to transport charged organic substances not only across the prey's cytoplasmic membrane but also across its own outer and inner membranes. The genomic data illuminate the potential diversity of substances that could be taken up, including transporters for multidrug resistance, organic solvent resistance, amino acids and peptides, and phosphate and nitrate transporters (figs. S4 and S5 and table S4). It remains to be seen whether the prey's membrane transporter systems are used in reverse to translocate cytoplasmic solutes to *Bdellovibrio* [as suggested in (*14*)] or whether the predator actively inserts its own gene products for this purpose into the prey's cytoplasmic membrane (*14*). In this regard, at least 15 of the hydrolytic enzymes are predicted to be extracellularly localized, suggesting a *B. bacteriovorus*–driven process (table S3).

It is of particular interest to determine which prey components are used as nutrients and which are used as direct source material for the de novo synthesis of biopolymers by *Bdellovibrio*. Apparently, *B. bacteriovorus* HD100 is capable of synthesizing only 11 of the amino acids needed for protein synthesis from intermediates of energy metabolism. Moreover, 10 amino acid degradation pathways are missing (table S5). However, all the enzymes are present that are required for the formation of the full range of activated tRNAs (fig. S1 and Table 1). These findings suggest that *B. bacteriovorus* is capable of protein biosynthesis only while it has access to amino acids from its prey. The two- to threefold increase in cell size and the flagellum elongation observed after *B. bacteriovorus*'s release from prey (*2*) presumably depend on prey-derived amino acid pools that were stored during the bdelloplast stage.

Previous biochemical experiments (*15*) indicate that ATP is directly obtained from the prey's cytosol; however, no homologs of known ATP transport proteins were seen in the *Bdellovibrio* genome like those described for the endoparasitic bacteria *Chlamydia* and *Rickettsia* (*16*). The mechanism for ATP transport remains unknown. *B. bacteriovorus* does, however, have a full complement of metabolic enzymes needed to generate ATP via glycolysis, the tricarboxylic acid cycle, and fatty acid degradation, as well as the ability to carry out oxidative respiration. This observation and the large number of predicted hydrolytic enzymes indicate that *Bdellovibrio* degrade prey molecules and resynthesize their own from the constituent bases, sugars, or acids rather than directly transporting and using complex metabolic intermediates of the prey.

The filamentous *B. bacteriovorus* cell that has grown to several multiples of its normal size begins to septate (stage VI). Although the gene products for chromosome partitioning and septation are homologous to those encoded by known genes (*mreB*, *mbl*, *ftsZ*, and *smc*), the mechanism itself is likely to be different, because one long filamentous cell divides into many progeny cells of equal size, and odd numbers of progeny are common (*7*). Because the amount of replicated chromosomal DNA of the *Bdellovibrio* cells adds up to a multiple of the available nucleotides generated from the prey's hydrolyzed chromosome, it is evident that *Bdellovibrio* has to synthesize large amounts of nucleotides from scratch. The organism has been found to contain a complete set of genes for purine and pyrimidine metabolism. There also is a complete LPS biosynthesis pathway, which gives rise to a unique lipid A molecule that has been described to be less antigenic than those of enteric bacteria (*17*).

Within the exhausted prey protoplast, the *B. bacteriovorus* progeny develop into flagellated cells that are ready for further attack (stage VII). At this stage, *B. bacteriovorus* produces hydrolytic enzymes (*18*) to dissolve the remaining peptidoglycan layer and the outer membrane of the prey cell (stage VIII) to release the progeny.

Bdellovibrio's hydrolytic arsenal is used in at least three distinct stages of its life cycle: prey entry (III), degradation of biopolymers (V), and the exit from the bdelloplast (VIII) (fig. S3). The largest group of paralogous hydrolytic enzymes, by far, is found in the family of proteases and peptidases (150), glycanases (10), deoxyribonucleases (DNases) (20), ribonucleases (RNases) (9), and lipases (15) (Table 1 and table S3). The significance of the proteases and peptidases becomes apparent when considering their density in the genome, which is the highest reported so far, with the exception of the minimalist genome of *Buchnera aphidicola*. The enzymatic activities of these gene products must be contained and organized in a timely fashion. The

Table 1. General features of the *B. bacteriovorus* HD100 genome (European Molecular Biology Laboratory accession no. BX842601).

Species	*B. bacteriovorus*
Strain	HD100 (Deutsche Sammlung Mikroorganismen DSM50701)
Size	3,782,950 bp
GC content	50.7%
GC in coding areas	50.4%
Predicted number of ORFs	3584
Coding sequences (CDS)	1995
similar to known proteins	
Conserved hypothetical proteins	382
Hypothetical proteins	1207
Coding potential	93%
Average CDS length	982 bp
rRNA operons	2
tRNA genes	36
Mobile genetic elements	1 IS element
	4 transposases
Four regions of deviating GC content	LPS synthesis
	Prophage insertion in $tRNA^{met}$
	Ribosomal gene cluster
	Restriction modification system
CDS coding for hydrolytic enzymes	150 proteases/peptidases
	20 DNases
	9 RNases
	10 glycanases
	15 lipases
	89 other

transcriptional control of these genes is likely to be carried out by numerous sigma factors and interacting proteins (21), as well as numerous transcription factors regulated by sensor histidine kinases (40) (table S1).

The ability of *B. bacteriovorus* to conduct a highly orchestrated and focused hydrolytic assault on bacterial prey depends on its type I and type II secretion systems, as well as on the twin-arginine translocation system (*19*). The absence of type III and type IV secretion systems, together with the fact that there are no reports of *Bdellovibrio* invading mammalian cells (*20*), is encouraging for their possible development as living antibiotics in future pharmacological applications. In addition, *Bdellovibrio*'s large complement of proteases and other hydrolases, revealed by the genome sequence, may provide a valuable reservoir of enzyme-based antimicrobial substances. Understanding their modes of operation and targets, as well as the mechanisms of predator resistance to these compounds, will aid the hunt for new antibacterial strategies.

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