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OPINION

Do bacteria have sex?

Rosemary J. Redfield

Do bacteria have genes for genetic exchange? The idea that the bacterial processes that cause genetic exchange exist because of natural selection for this process is shared by almost all microbiologists and population geneticists. However, this assumption has been perpetuated by generations of biology, microbiology and genetics textbooks without ever being critically examined.

Terms such as sex and recombination have different meanings in different contexts. Here, I use recombination to mean the breaking and joining of DNA strands; genetic exchange, gene transfer or HORIZONTAL TRANSFER to refer to processes that produce new genetic combinations; and meiotic sex to mean the cyclical alternation between haploid and diploid stages in eukaryotes. Sex refers to any process selected by the benefits of genetic exchange.

Understanding the evolutionary causes of genetic exchange in bacteria has important implications for our understanding of the evolution of meiotic sex in eukaryotes. The primary function of meiotic sex seems to be to produce new combinations of chromosomal genes, but extensive work by population geneticists has been unable to show why this would be beneficial[1–3]. If bacteria do have genes that have evolved for genetic exchange, then they provide much-needed independent systems in which to study how sex can evolve. If they do not, then meiotic sex must have evolved to provide eukaryotes with benefits that are not needed by bacteria and its evolutionary causes must be sought among eukaryote-specific phenomena.

Bacteria have several well-studied processes that can transfer genes, and the analysis of genome sequences has revealed that these processes have made important contributions to bacterial evolution. DNA can be transferred between cells by TRANSDUCTION, CONJUGATION or TRANSFORMATION, and can be physically recombined into their chromosomes by various cytoplasmic proteins. The many sequenced bacterial genomes contain abundant examples of genes that were unambiguously acquired by horizontal transfer. For example, most of the physiologically important differences between *Escherichia coli* and *Salmonella typhimurium* result from recombination: genes for lactose, citrate and propanediol use, and indole production, have all been acquired in this way[4].

How not to study selection for sex

Because the ability to create new genetic combinations affects FITNESS only indirectly and because the outcomes are intrinsically unpredictable, selection for the creation of new genetic combinations is much harder to investigate than selection on processes that contribute directly to survival or reproduction. One reason why so much misunderstanding surrounds the evolution of sex is that the least rigorous and most misleading evidence has had the greatest influence, whereas the strongest has been mostly overlooked.

The large number of transferred genes we find in modern bacterial genomes has misled many researchers about the benefits of genetic exchange. Many of the transferred genes are obviously beneficial to their new hosts and this is frequently interpreted as conclusive evidence that gene transfer must be adaptive. The foreign origin of many of these genes is firmly established, but the bacterial genomes that they are found in are unfortunately a very biased record of evolutionary processes. The problem, of course, is natural selection. Because natural selection eliminates almost all deleterious changes, the genomes of modern organisms are the result of several billion years of evolutionary success stories, with not a single failure represented. In a way, the sequences we see are a type of anecdotal evidence — each represents a unique event that has, against the odds, survived. Even if harmful exchange events were 100-fold more common than beneficial ones, we would only see the latter in genomes today. So, finding transferred genes in modern genomes shows that some transfers, like some mutations, are adaptive, but this finding does not address the larger issue of the average costs and benefits of exchange.

Filtering by natural selection is like the filtering of lottery outcomes by the media. On the basis of what we read in the newspapers, we would expect everyone who buys a lottery ticket to be a winner. Of course, most scientists know better than to buy lottery tickets, but many have failed to apply the same logic to the processes that generate genetic diversity. Research papers do not explicitly claim that genetic exchange must be adaptive because we see its benefits and not its harmful consequences — if this were done, the error would be obvious. Nevertheless, the error is probably responsible for much of the complacency with which most biologists view the evolution of genetic exchange.

Rigorous approaches to sex

Until recently, scientific approaches to the problem of the evolution of sex have almost exclusively been the domain of theoretical population genetics. The formulation of explicit mathematical statements is a rigorous tool for evolutionary analysis, but this rigour often demands a corresponding sacrifice of relevance. Mathematical modelling can show how selection on the genes that cause genetic exchange might act, but only under hypothetical and unrealistic assumptions about the processes and their consequences, which are required if the equations are to be solvable. Computer simulations are more versatile, as equations need not be solved but only applied repeatedly, but the assumptions that underlie the programming must still be simple. For example, both theoretical and computer models of the evolution of meiotic sex often assume that all mutations have identical effects on fitness. As a consequence, although both mathematical and computer modelling have been useful for showing that some explanations for meiotic sex are possible whereas others are not, they have failed to produce solid answers[2,3,5,6].

The power of long-term selection experiments on microbial cultures ('experimental evolution') has only recently been appreciated[7]. These experiments tell us how selection can act under laboratory conditions, by testing experimentally whether a given set of conditions leads to a change in the frequencies of certain genotypes in a population. Because many thousands of generations can be followed, the

experiments can detect relatively weak selective processes. However, a considerable limitation to laboratory selection experiments is that culture conditions inevitably fail to reflect the evolutionarily relevant conditions that are experienced by bacteria in their natural environments. The problem is not that experimenters do not wish to use natural conditions, but that it is usually impossible to determine which features of the natural environments of microorganisms are most important.

Neither theoretical nor experimental models of evolution have shed much light on genetic exchange in bacteria. Two models found that exchange could be beneficial only under conditions that were generally more restrictive than for sexual recombination in eukaryotes[8,9]. Another found that the genes that cause genetic exchange interfere with selection on the genes that affect mutation rates[10]. One well-controlled selection experiment that has addressed this problem found that introducing genetic exchange into laboratory populations of *E. coli* did increase their genetic variation, but that there was no concomitant increase in the rate or extent of adaptation[11].

Fortunately, the most powerful way to investigate the evolution of genetic exchange does not depend on mathematical tractability, nor on assumptions about selectively important components of the environment. Instead, the nature of the genes and the processes responsible for genetic exchange can reveal how selection has acted in shaping them. Because such genes and regulatory mechanisms evolved in the natural world over evolutionary time, they are more sensitive and accurate indicators of selection than laboratory evolution experiments or evolutionary theory can ever be. This kind of analysis does not depend on, or suffer from, preconceptions about the evolutionary function of the process being studied. In fact, most of the evidence has been produced by molecular biologists and bacterial geneticists, who had little concern for the evolutionary issues that their results are helping to clarify. Below, I consider the processes that contribute to genetic exchange, and what our current understanding of their mechanisms and regulation reveals about their evolution.

"The large number of transferred genes we find in modern bacterial genomes has misled many researchers about the benefits of genetic exchange."

What causes genetic exchange?

Bacterial genetic exchange is not like meiotic sex. Whereas meiotic sex regularly mixes two complete sets of genes and randomly reassorts the alleles into new individuals, bacterial recombinants form by processes that are non-reciprocal and fragmentary, and that are not regular components of bacterial life cycles. Any one recombination event transfers a single fragment of the chromosome from one cell, called the 'donor', to another, called the 'recipient'. Three well-studied processes are responsible for most naturally occurring DNA transfer: transduction by bacterial viruses, conjugation by bacterial plasmids and DNA uptake by naturally competent bacteria (transformation) (FIG. 1). Once in the cytoplasm of the recipient, transferred DNA fragments escape degradation only if they physically recombine with the chromosome. This usually occurs by replacing a recipient sequence with a very similar sequence from the donor, although unrelated donor sequences can sometimes be added to the recipient chromosome.

Recombination. Without the breaking and joining of DNA strands, DNA transfer could never lead to new genetic combinations. So, to understand the causes of genetic exchange we need to find out why cells have proteins that cause recombination. The strongest evidence comes from the phenotypes of mutants that lack these proteins and from molecular analyses of the protein activities. This evidence has shown that these proteins exist to promote DNA replication and repair, not genetic exchange.

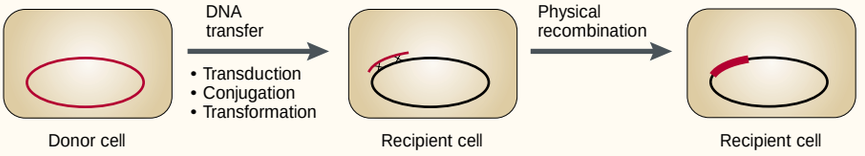

Figure 1 | **Genetic exchange in bacteria.** Transduction, conjugation and transformation can transfer a DNA fragment from a chromosome of a donor cell to a recipient cell. Physical recombination can then integrate this DNA into the recipient chromosome.

Most bacterial recombination is 'homologous'; that is, the two recombining segments have identical or near-identical sequences and base pairing between their strands replaces one with the other. The REC PROTEINS RecA and RecBCD have key roles in this process, along with other proteins (RecE, RecF, RecG, RecJ, RecN, RecO, RecQ, RuvABC, Ssb, PolA, DNA ligase and DNA gyrase A and B). Because mutations in the genes that specify these proteins disrupt recombination, the genes were often given '*rec*' names when first discovered. They were thought to function mainly in the 'recombination pathways' that were believed to have evolved to promote genetic exchange. Effects on DNA repair and overall viability were noted, but were usually considered to be secondary. Decades of genetic and functional analysis have shown that DNA replication and repair are, in fact, the primary functions of these proteins, and that these functions are achieved by mechanisms that also increase recombination[12,13]. For example, RecBCD, RecG and the RUV PROTEINS all contribute to restarting stalled replication forks[14–16], and RecA carries out a process called RECOMBINATIONAL REPAIR and also regulates repair by sensing DNA damage[12].

A less-common process is non-homologous or illegitimate recombination, in which two unrelated sequences become connected either by incorrect rejoining of broken ends or by insertion of one DNA segment into another. The former is mediated by DNA ligase, which is essential for DNA replication and repair, and the latter by TRANSPOSASES, which are encoded by, and essential for, the replication of transposable genetic elements.

So, both homologous and non-homologous recombination are carried out by proteins that have other important cellular functions. But how do we know that the recombination activities of these proteins have not also been selected? We can never prove that they have not, but there is no justification for invoking such selection, first because selection for each primary function is so strong that it dwarfs any possible selection for genetic exchange, and second because each protein seems to promote exchange only as a side effect of its other activity. For example, mutations in any of the *ruvA*, *ruvB* or *ruvC* genes cause a 50% reduction in cell viability, measured under conditions in which no genetic recombination is possible[17]. This extreme decrease in viability would certainly preclude success in the natural environment and is more than sufficient to account completely for the evolution of these proteins. Furthermore, their

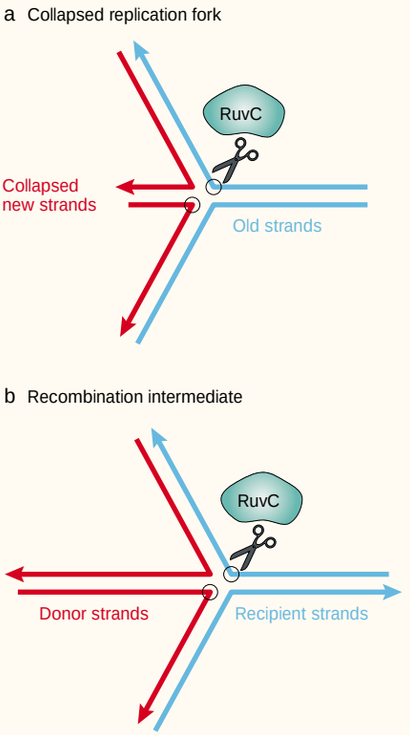

Figure 2 | **The role of RuvC in DNA replication and recombination.** RuvC is a crossover junction endodeoxyribonuclease, which cuts DNA at the circled positions to resolve the topologically identical Holliday junctions that arise during **a** | DNA replication and **b** | recombination.

mode of action in replication fully accounts for their mode of action in recombination. The Ruv proteins contribute to viability by resolving four-stranded DNA structures called HOLLIDAY JUNCTIONS, which arise when replication forks are stalled, and which prevent further DNA replication and thus kill the cell if unresolved. The role of RuvC in recombination is the same as its role in replication (FIG. 2). When DNA recombines it forms Holliday junctions that are topologically identical to those at stalled replication forks and viable recombinants are only produced if the junctions are resolved. There is no indication that the activity of RuvC has been in any way modified by selection for genetic exchange. Similar analyses can be done for other 'recombination' proteins — for example, RecA has repair and recombination activities that are essentially identical[12]. The presence of a damaged base in the substrate for repair, but not for recombination, is the only difference between these events. So, the genetic exchange produced by the various DNA-transfer processes seems to depend on recombination that occurs as a side effect of DNA repair and replication.

Transduction and conjugation. Two of the well-studied DNA-transfer processes — transduction and conjugation — depend on infectious agents that move DNA from cell to cell. In both, gene transfer seems to be a simple side effect of the infectious activities of these agents (FIG. 3). The strongest evidence of how natural selection acts on these processes is the location and action of the genes responsible for them. Transduction is the most common process[18] (FIG. 3a). Transduction is also used for strain construction in the laboratory[19]. It is caused by the many bacterial phages (viruses) that occasionally package host DNA instead of phage DNA into viral particles and then inject this DNA into new cells. All the genes involved in transduction are on phage genomes, not host chromosomes, which indicates that there is selection for transfer of phage DNA but not host DNA. By promoting production of infectious phages, these genes strongly enhance their own evolutionary success. No host genes promote the packaging of DNA (host or phage), which indicates that this packaging probably has no significant benefit to the host. In many phages, the gene product that is responsible for initiating DNA packaging recognizes a sequence in the phage genome and transduction depends on this protein mistaking a host sequence for the phage sequence[20]. There is no evidence that such host sequences have been modified to promote packaging in phage particles.

The infectious agents responsible for conjugation are mostly plasmids — small, circular DNA molecules that replicate independently of the host chromosome (FIG. 3b); some transposons can also cause conjugation. Both types of conjugative element cause their host cells to form a connection to cells that lack the element and to pass a copy of the DNA of the element to the new host cell. If chromosomal DNA is connected to the element it too will be transferred[21]. Again, we can infer selection for transfer of the conjugative element but not for the host genes because all the genes specific to conjugation are on the element and there are no host genes that specifically promote conjugal DNA transfer. Some host proteins are required for the conjugation process, such as DNA polymerase, but these also make direct contributions to host fitness, which fully explain their roles in conjugation; they make no distinction between host and conjugative-element substrates. Nor are there sequences or genes that physically connect host DNA to conjugative plasmids and cause their transfer. Instead, these connections usually arise as side effects of the activities of other genetic parasites, most commonly the short transposable elements called insertion sequences. So, conjugation, like transduction, seems to transfer host genes by accident.

Competence and transformation. Our improved understanding of conjugation and transduction, and of the enzymes that cause physical recombination of the transferred DNA, consistently supports the hypothesis that transfer and recombination of chromosomal genes are unselected side effects of processes that have evolved for other functions. However, the evolutionary function of a third DNA-transfer process remains controversial. This third process is the development of a state called competence, in which bacteria can take up DNA fragments from their environment. A cell the chromosome of which recombines with such a fragment might change its genotype and thus become 'transformed'. Some bacteria cannot become naturally competent, at least under laboratory conditions (in *E. coli*, 'competence' refers to artificially permeabilized cells), but many can[22].

Unlike conjugation and transduction, competence is not caused by infectious agents. The genes required are all chromosomal, which indicates that the benefit of DNA uptake is to the recipient, not to another genetic element or parasite[23]. (DNA donors cannot benefit as they are already dead.) And, unlike mutations in recombination genes, mutations in competence genes do not have marked effects on viability; mutants that are unable to take up DNA usually grow well under standard culture conditions.

Although competence has usually been thought to exist to favour genetic exchange[23–25], homologous DNA can, in principle, also be used as a template to repair otherwise-lethal DNA damage[26]. Such a repair function could be much more important to the cell than genetic exchange, because DNA damage is more harmful and more common than deleterious mutation or an unreliable environment. (Theory predicts that both of these factors can select for genetic exchange under some circumstances.) Experiments that tested whether competent *Bacillus subtilis* cells could use externally supplied DNA for repair were inconclusive[26–28] and had two serious weaknesses. First, this type of experiment was not sufficiently sensitive to detect modest but potentially significant differences in survival. Second, the cells needed special treatment to become competent, because they would not take up DNA under standard culture conditions.

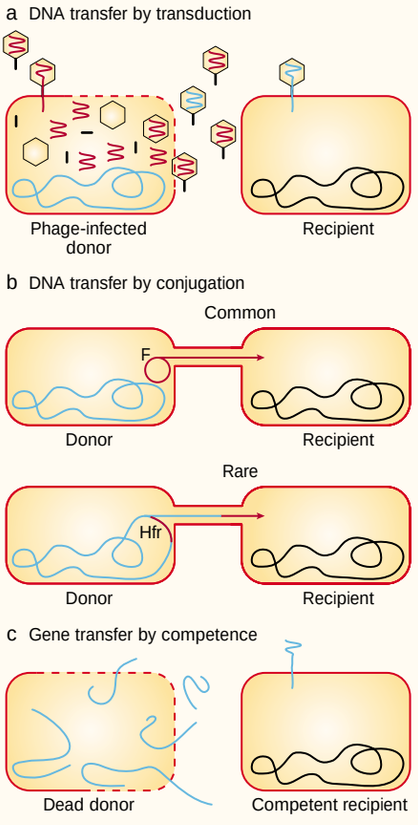

Figure 3 | **Methods of DNA transfer.** **a** | Transduction is the phage-mediated transfer of host genetic information. In a phage-infected bacterial cell, fragments of the host DNA are occasionally packaged into phage particles and can then be transferred to a recipient cell. **b** | Conjugation is the transfer of DNA from a donor cell to a recipient that requires cell-to-cell contact. Genes on conjugative plasmids, such as the F plasmid, encode products that are necessary for this contact, and replication and transfer of the plasmid to the recipient. When, on rare occasions, the F plasmid becomes integrated into the host chromosome (Hfr), conjugation results in a partial transfer of the donor chromosome. **c** | Cells that are competent can take up free DNA from their environment. For all three methods of DNA transfer, the donor chromosomal DNA will only be permanently maintained and expressed in the recipient cell if it is integrated into the recipient genome by physical recombination.

Natural selection for competence

The ability to take up DNA is tightly regulated in most naturally competent bacteria. Although this regulation is a disadvantage for those doing selection experiments in the laboratory, it is an advantage for those wishing to understand selection in the natural environment. If competence evolved to provide templates for DNA repair, the most effective form of regulation should induce competence when DNA is damaged. However, experiments in both *B. subtilis* and *Haemophilus influenzae* have shown no connection between the cellular machinery for sensing DNA damage and that for inducing competence[29]. Because all known DNA-repair mechanisms are induced by the presence of DNA damage, the failure of DNA damage to contribute to the regulation of competence strongly indicates that DNA repair is not the main function of competence.

Surprisingly, the most obvious, immediate and inevitable benefit of DNA uptake has generally been overlooked or, more recently, discounted[23,24]. Like other molecules that are taken up by bacterial cells, DNA can be used as a nutrient[30]. Some bacteria might break it down as a source of carbon and nitrogen, but its primary use is likely to be as a source of nucleotides for DNA and RNA synthesis. This spares resources that would otherwise be needed for nucleotide synthesis, a very 'expensive' cellular process[31]. Furthermore, many competent bacteria live in very DNA-rich environments, so this uptake might make a substantial contribution to the energy budget of the cell. For example, *H. influenzae*, *Streptococcus pneumoniae* and *Neisseria meningitidis* live in respiratory tract mucus (~300 μg DNA per ml of mucus[32]); *Helicobacter pylori* and *Campylobacter jejuni* live in gastrointestinal mucus (~200–400 μg DNA secreted into the gastric lumen every 10 min (REF. 33)); and *B. subtilis* lives in soil (>10 μg DNA per g of soil[34]). In laboratory cultures, competent bacteria degrade most of the DNA they take up and use the released nucleotides mainly for DNA synthesis[35]. Although Gram-positive bacteria directly internalize only one DNA strand[23], in nature, the nucleotides released by hydrolysis of the other strand will also be efficiently taken up and used. DNA is likely to be of more value as a source of nucleotides than for DNA repair, because cells continuously need nucleotides for DNA and RNA synthesis even in the absence of damage.

The nutrient hypothesis, like the DNA-repair hypothesis, can best be tested by looking at its regulation. If DNA is mainly a source of nutrients, competence should be induced by nutritional signals. The most important signals are likely to be the depletion of nucleic acid pools and of the energy resources needed for nucleotide synthesis. Most research into the regulation of competence has not been motivated by an interest in its function but, nevertheless, evidence of nutritional regulation is accumulating.

The clearest evidence for nutritional regulation of competence comes from *H. influenzae*, in which competence has traditionally been induced by transferring cells to a 'starvation' medium. Induction of competence genes absolutely requires an increase in cyclic AMP, a signal produced when preferred energy sources are depleted[36–38]. A more recent finding is that an essential feature of the *H. influenzae* starvation medium is its lack of purine nucleotides and nucleosides, the presence of which prevents the transcription of competence genes[39]. So, for *H. influenzae*, the regulation of competence fits the predictions of the nutrient hypothesis.

"Surprisingly, the most obvious, immediate and inevitable benefit of DNA uptake has generally been overlooked or, more recently, discounted. Like other molecules that are taken up by bacterial cells, DNA can be used as a nutrient."

Nutritional signals also have roles in regulating competence in other bacteria, although interpretations have been hampered by the common assumption that genetic exchange must be more important than food. In *B. subtilis*, competence is normally induced by transfer to a nutrient-limited liquid medium or to solid media that lack a required amino acid or base[40]. Many of the factors that regulate competence in *B. subtilis* reflect nutrient availability; CodY senses nitrogen levels[41] and PtsG controls CATABOLITE REPRESSION[42]. Regulatory mechanisms are also shared with known nutrient-acquisition processes, such as secretion of degradative enzymes[43]. Furthermore, although the *comEA* and *comEC* genes in *B. subtilis* are essential for DNA uptake, the *comEB* gene in the same competence-regulated OPERON encodes dCMP deaminase, an enzyme that is required for salvage of dCMP[44]. This protein has no role in DNA uptake, but its presence in this operon might reflect a role in processing the deoxynucleotides that DNA uptake provides. In *Acinetobacter*, competence genes are maximally expressed when nutrients become depleted and when growth ceases in late stationary phase, although DNA cannot be

taken up until the cells are transferred to fresh medium[45]. The involvement of QUORUM-SENSING PEPTIDES in competence regulation in various bacteria has been interpreted as an adaptation for genetic exchange by inducing DNA uptake when DNA from CONSPECIFICS is likely to be available[46,47]. However quorum-sensing mechanisms often have well-established roles in nutrient acquisition; many control secretion of degradative enzymes that release nutrients for the cell to take up[48]. Other quorum-sensing functions might act as early warning signals of the nutrient shortages that are likely to result from a high population density.

Of course, many aspects of competence are not yet understood. *N. meningitidis* and *H. influenzae* have sequence-biased DNA-uptake systems that cause them to preferentially take up DNA from their own or closely related species[23]. In both *H. influenzae* and *S. pneumoniae*, competence-regulating proteins seem to control the expression of genes that have no obvious connection to competence[49,50]. Only a small fraction of *B. subtilis* cells become competent in laboratory cultures, which indicate that an important component of regulation has been overlooked. The induction of DNA-repair enzymes, such as RecA, in some competent cells has been interpreted as an adaptation for recombination. Instead, it could mean either that DNA uptake causes DNA damage or that incoming single-stranded DNA sends a false signal of DNA damage.

"Why have no genes been selected to cause genetic exchange, given that beneficial recombinants have made so many contributions to modern bacterial genomes? The explanation … new genetic combinations are, like mutations, more often harmful than beneficial …"

However, these and similar puzzles might disappear once their causes are better understood. For example, the 'gene-transfer agent' (GTA) of *Rhodobacter capsulatus* was originally thought to have evolved for genetic exchange. It packages 3–4-kb fragments of chromosomal DNA into protein particles that can inject the DNA into new cells. However, we now know that GTA is encoded by a defective PROPHAGE, so the transfer that it causes might simply be a form of transduction by a phage that can no longer specify its own replication[51]. Cellular regulation of GTA-encoded genes could therefore reflect selection to reduce harmful effects on its host, rather than to optimize genetic exchange[52]. The short, repeated sequences called Chi are another example, the true functions of which were not originally appreciated. Chi sequences were once thought to be abundant in the *E. coli* genome because they are needed to produce genetic exchange by homologous recombination, but are now known to orientate RecBCD-mediated repair at DNA replication forks[14,53].

Conclusions

The analysis presented here avoids speculation about hypothetical conditions and constraints, by drawing conclusions from genes and mechanisms that have been produced by natural selection. In effect, investigations into regulatory systems ask the bacteria which factors have been important to them in their natural environment over evolutionary time. For conjugation, transduction and the enzymes that cause physical recombination, the evidence is robust: genetic exchange occurs as an unselected side effect of processes that evolved for more immediate functions. Although questions remain to be answered about competence, the accumulating evidence for its nutritional regulation is shifting the burden of proof onto those who favour a genetic exchange function.

Why have no genes been selected to cause genetic exchange, given that beneficial recombinants have made so many contributions to modern bacterial genomes? The explanation is probably the same as for the processes that create mutations: new genetic combinations are, like mutations, more often harmful than beneficial, and although the rare beneficial outcomes have been preserved, the processes themselves have been selected against because of their usually harmful outcomes.

Many factors have contributed to misconceptions about bacterial sex. One is terminology. Molecular biologists and population geneticists both use the term 'recombination', but the first group means the machinery that breaks and joins DNA, whereas the second means the new genetic combinations that this machinery can produce. Gene names can also be misleading to non-specialists; the names of '*rec*' genes reflect their laboratory discovery, not their primary function. Another very misleading factor is the bias introduced by natural selection, which sweeps all the deleterious out-

Glossary

CATABOLITE REPRESSION
Transcriptional repression of a prokaryotic operon by the metabolic products of the enzymes that are encoded by the operon.

CONJUGATION
In prokaryotes, transfer of DNA from a donor cell to a recipient cell is mediated by direct cell–cell contact.

CONSPECIFICS
Members of the same species.

FITNESS
A measure of the capacity of an organism to survive and reproduce.

HOLLIDAY JUNCTIONS
Cross-shaped junctions at which four strands of DNA meet and exchange partners, an important intermediate of recombination.

HORIZONTAL TRANSFER
Acquisition of genetic information from another cell.

OPERON
A genetic unit or cluster that consists of one or more genes that are transcribed as a unit and are expressed in a coordinated manner.

PROPHAGE
An inactive bacteriophage genome integrated into the host genome.

PROTISTS
Single-celled eukaryotes.

QUORUM-SENSING PEPTIDES
Peptides secreted and detected by cells. Cells respond to extracellular peptide only when cell densities are sufficiently high (the 'quorum state') that the extracellular concentration of the peptide exceeds a threshold.

REC PROTEINS
A general class of protein that participates in recombination.

RECOMBINATIONAL REPAIR
DNA repair made possible when a damaged DNA strand base-pairs with a complementary undamaged strand from a different molecule.

RUV PROTEINS
Proteins that translocate and resolve Holliday junctions.

TRANSDUCTION
Virus- or phage-mediated introduction into a cell of a DNA fragment derived from a different cell.

TRANSFORMATION
Change of the genotype of a cell brought about by uptake of free DNA.

TRANSPOSASE
An enzyme that carries out the site-specific DNA recombination required for transposition.

comes under the carpet, giving the false impression that most exchange is adaptive. Yet another is the erroneous belief that selection for 'evolvability' will override selection for viability. Although it is true that evolvability — the ability to generate adaptive genetic variation — will be indirectly favoured by selection, this is usually much too weak to counteract direct selection on the maladaptive variation that is also generated. This error underlies Weismann's widely accepted hypothesis that sexual reproduction exists to prevent extinction by creating genetic differences[54].

What does the study of bacterial genetic exchange processes indicate about the evolution of meiotic sex? Mutation and accidental genetic exchange provide bacteria with all the genetic variation they need, so perhaps eukaryotes evolved sexual reproduction because they get much less accidental exchange than bacteria do, or because they need much more. Neither seems especially likely. Meiotic sex arose in PROTISTS, not plants or animals[55]. Both viral infections and phagocytosis are likely to cause genetic exchange in protists, and this exchange might easily be as common in protists as exchange is in bacteria. Conversely, there is no obvious reason why protists would need more genetic exchange than bacteria: the two groups have similar mutation rates and overlapping genome sizes[56]. We can only hope that research into the molecular mechanisms and regulation that underlie meiotic sex will provide new insights.

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Links

DATABASE LINKS RecA | Rec | RecE | RecF | RecG | RecJ | RecN | RecO | RecQ | RuvABC | SSB | PolA | DNA ligase | DNA gyrase | CodY | PtsG | *comEA* | *comEC* | *comEB*
FURTHER INFORMATION *Salmonella* | *Escherichia coli* | Redfield lab

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