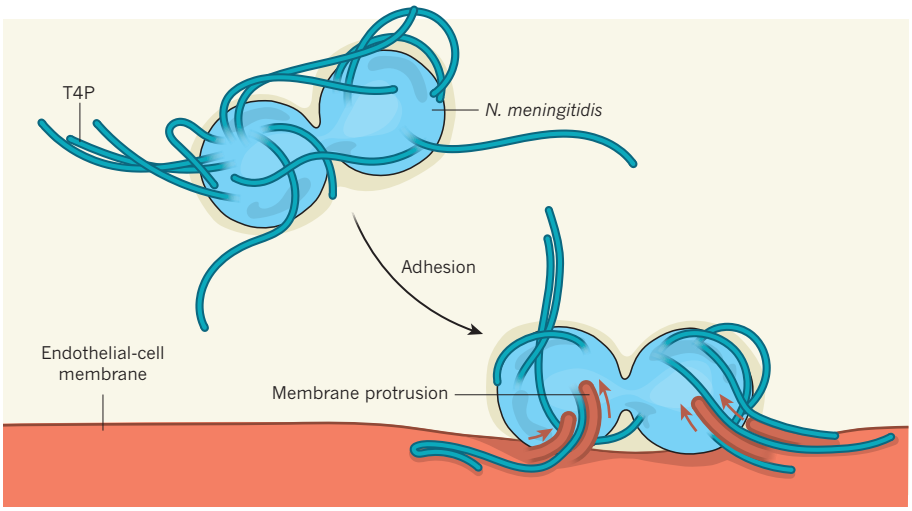

Figure 1 | Enhancing adhesion between a bacterium and an endothelial cell. The bacterium *Neisseria meningitidis* attaches itself to the endothelial cells that line blood vessels in host organisms. The bacterium uses fibres known as type IV pili (T4P) to induce the formation of protrusions from endothelial-cell membranes. These protrusions strengthen the bacterium's hold on the membrane, helping it to colonize cells without being swept away by the surrounding blood flow. Charles-Orszag *et al.*[2] propose that the adhesion of T4P to the membrane drives a process called one-dimensional wetting, in which the protrusions are drawn along the T4P fibres (red arrows). (Adapted from Fig. 5 of ref. 2.)

for forming membrane protrusions, which the authors call one-dimensional wetting, is driven by adhesion between the membrane and the fibre. The membrane protrusions could help to anchor a bacterium to a host cell as its T4P extend and retract, without breaking the adhesive interactions between the T4P and the membrane — thus maintaining the dynamic nature of the fibres.

Because the remodelling of endothelial-cell membranes by *N. meningitidis* had previously been observed only for cultured cells, the researchers studied blood vessels in human skin grafted onto mice to confirm that remodelling also occurs *in vivo*. They then complemented those experiments with *in vitro* studies to explore the mechanism involved. Unfortunately, the *in vitro* experiments did not examine the interaction of isolated T4P with model membranes, because this would have required the appropriate receptor proteins to be introduced into the membranes. Instead, Charles-Orszag *et al.* studied two model systems: artificial cells (known as giant unilamellar vesicles) interacting with filaments of a protein called actin through adhesion between the filaments and molecules attached to the cells; and endothelial-cell membranes interacting with mimics of the fibres found in the extracellular matrix around cells.

The authors show that 1D wetting does indeed occur in these systems, and that it can be understood quantitatively using their model. Their *in vitro* observations highlight the essential feature of this phenomenon: the presence of adhesion between a deformable membrane and a nanoscale fibre. Their observations also suggest that 1D wetting could occur more generally for physiologically important interactions of human cells with other biological nanofibres, and that it could have a major role in cell migration.

Further work is needed to understand 1D wetting in more detail. Systematic studies in which the fibre radius, strength of the adhesive interaction and surface tension of the membrane are varied would improve our understanding. In addition, further developments in microscopy will lead to better visualization of the structure and dynamics of the protrusions involved in 1D wetting.

Charles-Orszag and co-workers' results reveal opportunities for biomimetic strategies for wetting synthetic nanofibres and for producing strong adhesives, and new ways of moving nanoscale objects. Their findings also imply that reducing or disabling the 1D wetting of *N. meningitidis* T4P would limit the bacterium's ability to colonize and infect host cells, opening up a potential avenue for drug discovery. More generally, 1D wetting might enable cell function and health to be manipulated through interactions of cells with nanofibres to which biologically active molecules have been attached. ■

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GENETICS

A genomic approach to mosquito control

A high-quality genome sequence for the mosquito *Aedes aegypti* has now been assembled. The sequence will enable researchers to identify genes that could be targeted to keep mosquito populations at bay. SEE ARTICLE P.501

SUSAN E. CELNIKER

Every year, millions of people are bitten by the mosquito *Aedes aegypti*. Thousands die as a result of infection by the viruses the mosquito carries[1], which can cause diseases such as yellow fever, dengue fever and Zika. Current mosquito-suppression methods typically involve pesticides. However, mosquitoes quickly develop resistance to these chemicals[2], and pesticides can accumulate in the food chain, with adverse effects on beneficial insects, other wildlife and humans. New control methods are therefore needed. On page 501, Matthews *et al.*[3] describe a high-quality genome sequence for *A. aegypti* (Fig. 1). This exemplary work could be a major step towards addressing our current inability to manage expanding mosquito populations.

Arguably the most promising alternatives to pesticide-based mosquito control are targeted molecular strategies based on genetics. The first requirement for the success of such strategies is high-quality sequencing of the mosquito genome. This would enable researchers to identify gene targets that could be manipulated to achieve a range of effects: to disrupt the mosquito's host-targeting systems; to make sterile males; to convert females into harmless males; or to render the insect incapable of harbouring viruses.

The repetitive nature of the 1.3-gigabase-long

JOAO PAULO BURINI/GETTY

Figure 1 | The mosquito *Aedes aegypti*. Matthews *et al.*[3] describe a high-quality genome sequence for this mosquito species.

A. aegypti genome has severely hampered efforts to generate a high-quality sequence. Previous attempts[4,5] resulted in patchy genomes that were assembled using short sequence reads. To overcome these challenges, Matthews *et al.* used next-generation sequencing to generate 166 Gb of long sequence reads with an average length of 17 kilobases. The authors used sophisticated mapping and gap-filling techniques to determine the positions of 94% of their sequence reads on the mosquito's three chromosomes, successfully assembling 1.28 Gb of the genome. The assembly has many fewer gaps than previous assemblies, and is a 100-fold improvement in terms of its N50 — a statistical measure based on the median assembled DNA-sequence length.

With this assembly in hand, Matthews and colleagues were able to improve our knowledge of the sequences of thousands of genes, and to discover new members of existing gene families. For example, the researchers identified more than 300 genes that encode ligand-gated ion channels, which allow ions to pass through membranes. These genes fall into three classes of receptor: odorant, gustatory and ionotropic. Together, they sense a wide range of chemicals, including carbon dioxide and chemicals that emanate from humans. Matthews *et al.* identified 54 previously unknown genes encoding ionotropic receptors — almost doubling the number known before. These genes are ideal candidates to target for disruption, because they confer the mosquito's ability to detect odours that indicate the presence of a host.

Of note, the authors identified 14 members of the best-studied subgroup of ionotropic receptors, nicotinic acetylcholine receptors, which act in the insect nervous system[6]. These receptors are the targets of insecticides called neonicotinoids, which have gained much attention owing to their adverse effects on beneficial insects such as bees. Knowing the sequences of the genes that encode these receptors should enable researchers to design insecticides that specifically target mosquitoes, sparing beneficial species.

Gene duplication is one mechanism by which insects can develop resistance to pesticides. Matthews *et al.* used their assembly to resolve a complicated gene-repeat region involved in one such resistance event. The region contains a cluster of three *Glutathione S-transferase (GST)* genes, which the authors found had been duplicated four times. These genes are important for metabolizing toxins, with one gene, *GSTe2*, capable of metabolizing the insecticide DDT. Increased expression of *GSTe2* has been associated with DDT resistance in a laboratory-colonized *A. aegypti* strain[7], supporting the idea that the gene duplication identified by the authors is involved in pesticide resistance. These data provide a proof of principle that the new genome will be an invaluable resource for researchers looking to analyse any gene family implicated in pesticide resistance.

Sex determination in *A. aegypti* is controlled by a sex-specific region called the M locus that is located on chromosome 1 in males only. It was known that the region contained the male-specific genes *myo-sex* and *Nix*, but they were absent from previous genome assemblies. This gap has been filled in the new genome. The authors estimate the M locus to be 1.5 megabases long (0.1% of chromosome 1), and show that it contains a much more repetitive sequence than does the rest of the genome — 73.7% compared with 11.7% genome-wide. The high repeat density is similar to that found in the Y chromosome of other animals[8].

Apart from the M locus, the sequence of chromosome 1 is very similar in males and females. This type of chromosome structure is known as homomorphic. Matthews and colleagues' genome will provide researchers with the opportunity to examine how the homomorphic sex chromosomes of *A. aegypti* are maintained, rather than evolving into heteromorphic chromosomes that are broadly different between the sexes — a better-understood phenomenon that is exemplified by the human X and Y chromosomes.

Finally, the authors used genetic-mapping techniques to identify regions of the genome that are associated both with the ability of mosquitoes to act as vectors for dengue virus and with resistance to the pesticide deltamethrin. The latter analysis highlighted candidate genes not previously known to be involved in pesticide resistance.

Even though Matthews and co-workers' genome is a radical improvement on previous assemblies, important genes might still be missing, because there are a few thousand gaps in the main chromosomes, and large gaps spanning specialized structures called centromeres, to which proteins bind during cell division. Nonetheless, the authors' sophisticated genome-sequencing strategy should act as a template for future efforts to assemble complex genomes. The genome and the gene sets themselves are publicly available for others to use (see go.nature.com/2dc6kxp), and, thanks to genome-editing technologies such as CRISPR–Cas9, researchers will easily be able to explore the effects of disrupting each gene identified as a candidate for targeting.

The use of tools rooted in genomic analysis and manipulation is a key step towards a pesticide-free world. Matthews and colleagues' work makes a major contribution to this goal. ■

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CORRECTION

The News and Views article 'Beating the quantum limits (cont'd)' (*Nature* **331**, 559; 1988) gave the wrong citation for Masanao Ozawa's paper. It should have referred to M. Ozawa *Phys. Rev. Lett.* **60**, 385 (1988).