Recently, I sat down with a local molecular life scientist at Oklahoma State University to ask her about a recent scientific achievement she considers to be radically exciting for her field. Dr. Erika Lutter, a native of Canada and now microbiologist in Stillwater, Oklahoma, told me she recently discovered a phenomenon in the bacteria responsible for the nation’s most common sexually transmitted infection, *Chlamydia trachomatis*, which revered a decade old dogma about the way this microbe leaves host cells to infect others.

In her paper, Comparison of Murine Cervicovaginal Infection by Chlamydial Strains: Identification of Extrusions Shed *In vivo*, Lutter and her team of researchers infected the cervicovaginal cavities of mice models with multiple strains of the STI bacteria in order to observe, for the first time, in a living host organism what is called extrusion shed. Observed before *in vitro*, or in a non-living, petri dish environment, this is the process by which the chlamydia bacteria exit the cell, not by host cell lysis (destruction of host cell), but by the covert hijacking of the host cell’s membrane bound vacuoles, thus the bacteria is able to travel through host tissue, as though it had an invisibility cloak created from the host cell's membrane. When asked about just how these bacteria are able to accomplish this rare form of intracellular travel, Dr. Lutter, with visible excitement, told me the bacteria performs what is effectively “reverse endocytosis,” whereby the germ uses a myelin phosphatase, as opposed to the more usual actin phosphatase, to manipulate the host cell vacuole and escape the cell while invisible to host immune system defenses. This is a hugely important finding, she explains, because the potential to pharmacologically target this myelin phosphatase for inhibition could mean an effective method to prevent the spread of chlamydia infections through a patient’s body.

The significance of this finding can be understood in Dr. Lutter’s explanation of the study’s publication: “In the following weeks after publishing our findings, I received dozens of calls from researchers who were just sure that I had messed up– that I had forgotten to account for various variables. This study disproved the dogma of intracellular bacteria’s sole exit strategy being cell lysis, and many scientists took this novel finding with an extreme, yet understandable, skepticism.” One of the major limitations of her study is the use of a murine (mouse) model. Skeptics of her findings say the study, while is indeed significant in demonstrating the first *in vivo* extrusion shed of Chlamydia, it does not go far enough because we cannot definitively extrapolate the mouse-based findings to actual chlamydial events in humans. And this is critical, Dr. Lutter told me, as humans are the natural host species for *Chlamydia trachomatis*, and as such it is essential to study this phenomenon directly in Chlamydia’s natural habitat because the foreign environment of the mouse model could, although unlikely, be responsible for the extrusion shed that would not otherwise happen if occurring within a human. Accordingly, Dr. Lutter is currently planning on conducting a similar study with human cervicovaginal samples, so that she may strengthen her evidence and further our progress to a better understanding of *Chlamydia* *trachomatis* and possible vaccines targeting this cellular process.

References

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