modulate and overcome bacterial features that normally interfere with antibacterial treatment. But another interesting strategy of improving infection treatment of antibiotic-resistant organisms is to strengthen the host response. This can be done in several ways, such as using antibodies to target bacterial resistance mechanisms, stimulating bacterial eradication inside macrophages, enhancing bacterial killing by neutrophils, or reducing the immune response to minimize tissue damage.

TLR5 agonists together with antibiotics in a mouse pneumococcal infection model resulted in less risk of dissemination, increased number of early neutrophils, and improved antibiotic efficacy (Porte et al. 2015). The advantage of this strategy is an increased clearance of bacteria and the potential decreased dosing of antibiotics that may decrease the emergence of antibiotic resistance and dysbiosis of the microflora, normally adverse effects from antibiotic treatment. In a separate study screening for immunostimulatory molecules, streptazolin was identified and shown to activate phospho-inositol-3 kinases that resulted in activation of NF-κB, which increased bacterial killing by macrophages (Perry et al. 2015). There are additional studies that have been performed to investigate immunomodulation as a strategy to improve antibacterial treatment (Hancock et al. 2012). Many of those rely on molecules that activate toll-like receptors or NODs. Although promising as sensitizing options to eradicate antibiotic-resistant bacteria, immunostimulation needs to be tightly regulated not to result in immune dysregulation with adverse effects.

Specific antibodies have become a staple of anticancer treatment, and in similar ways antibodies are being investigated for their ability to attack antimicrobial resistance mechanisms. Antibody treatment is not a new concept and was explored already in the 1930s by Avery and co-workers, who showed that anti-capsular antibodies could eradicate pneumococci from the bloodstream of mice (Avery and Dubos 1931). As mentioned above, antibodies to IHF eradicated biofilms and synergized with antibiotics to eradicate antibiotic-resistant bacteria (Brandstetter et al. 2013). In a study by Barekzi et al., injection of mice with human pooled sera into the site of infection with *E. coli* or *K. pneumoniae* produced synergistic and protective effects with antibiotics in lethal wound infections (Barekzi et al. 2002). This may be a strategy that can be used more widely (Casadevall et al. 2004).

A study by Lo et al., used a different approach and showed that AR12, a compound able to help macrophages eradicate bacteria by forcing lysosome fusion, had synergistic effects with aminoglycosides on eradicating intracellular infection with *Salmonella typhi* both *in vitro* and *in vivo* (Lo et al. 2014). Intracellular bacteria are often more difficult to treat because many antibiotics have poor penetrance into eukaryotic cells. For example, GAS infections tend to recur in patients after antibiotic treatment (Cue et al. 2000). This is not because *Streptococcus pyogenes* has acquired intrinsic antibiotic resistance but because the bacteria hide inside the cell where they stay protected