Argument 1:
P1: People use antibiotics because they want to quickly eliminate the effects of bacterial infection.
P2: Bacterial infection is caused by the production of toxins that reach an unsafe level as bacterial populations increase.
C1: Using an antibiotic that disables a bacterium's toxicity will quickly eliminate the harmful effects associated with bacterial infection.

Argument 2:
P3: However, bacteria resistant to the anti-toxicity antibiotic survive the selective pressure of antibiotic treatment and are able to pass their resistance genes to other bacteria.
P4: The passing of these resistance genes between members of different bacterial species can give widespread antibiotic resistance among many different species of bacteria.
P5: This antibiotic resistance causes the anti-toxicity antibiotic to become ineffective against those bacteria.
P6: Renewed bacterial infection occurs that responds favorably to the selective pressure, and therefore resists the anti-toxicity antibiotic treatment.
C2: An anti-toxicity antibiotic is only a short-term solution to eliminating bacterial infection. A longer-term solution would be one that prevented bacteria from responding favorably to the selective pressure of antibiotic treatment.

Argument 3:
P7: Antibiotic resistance is the result of the selective pressure imposed on a bacterial population by antibiotic treatment.
P8: A bacterial population responds to this selective pressure when its resistant individuals outnumber and reproduce more than its non-resistant individuals.
P9: Resistant bacterial populations grow because resistance genes have been shared between members of that population.
C3: A gene transfer-disabling antibiotic would prevent bacteria from responding to the selective pressure, thereby preventing the growth of resistant bacterial populations.

Argument 4:
P10: People want to take antibiotics only as long as it will help their infection symptoms go away.
P11: An anti-toxicity antibiotic will help a person's infection symptoms go away.
P12: It is difficult to make sure that people take their antibiotics as prescribed, throughout the entire recommended time period.
P13: If people do not take their antibiotics as prescribed, then there is a greater chance that resistant bacteria will develop through the transfer of resistance genes.
P14: We do not want any more resistant bacteria to develop.
P15: A gene transfer-disabling antibiotic would prevent the development of more resistant bacteria.
C4: A gene transfer-disabling antibiotic should be used in conjunction with an anti-toxicity antibiotic for best results.

Argument 5:
P16: There is a probability that a bacterial strain will survive antibiotic treatment by developing resistance to that antibiotic.
P17: There are only a finite number of ways that a bacterial strain can become resistant to any given antibiotic (vs. an infinite number of ways).
P18: If the above statement is true, then the probability that a bacterial strain will survive an antibiotic treatment is inversely proportional to the number of different antibiotics used. That is, if 5 different antibiotics are used, the chances of any single bacterium surviving will be much less than if only one antibiotic is used.
C5: When treating a patient with antibiotics, the best results can be obtained by using not just one antibiotic, but many antibiotics.

Argument 6:
P19: There are good bacteria in the body that help to suppress the growth of harmful bacteria.
P20: Antibiotics with low selective toxicity will kill off a number of the good bacteria as well as the harmful ones.
P21: Using multiple low selective toxicity antibiotics may have a harmful effect on a patient by killing off most of the good bacteria and creating an environment where harmful bacteria may flourish.
C6: It is necessary for all antibiotics used in multiple-antibiotic treatment to have a very high selective toxicity.

Argument 7:
P22: Recently, the genomes of many types of bacteria have become fully sequenced. The genetic information which governs the synthesis of proteins involved in bacterial toxin formation, binary fission, conjugation, and the bacterial capability to mutate readily is now readily available for study.
P23: Instruments are available that will allow the surface of cells to be imaged, and for the molecular structure of individual proteins and other surface structures to be determined.
P24: Software is available that have allowed pharmacologists to create designer drugs that will bind to select structures on the surface of specific types of cells.
C7: If these current scientific and technological resources were effectively utilized together, we should be able to create anti-toxicity and gene transfer-disabling antibiotics with species-specific selective toxicity.

Listing of conclusions of the above sub-arguments:
C1: Using an antibiotic that disables a bacterium's toxicity will quickly eliminate the harmful effects associated with bacterial infection. C2: An anti-toxicity antibiotic is only a short-term solution to eliminating bacterial infection. A longer-term solution would be one that prevented bacteria from responding favorably to the selective pressure of antibiotic treatment.
C3: A gene transfer-disabling antibiotic would prevent bacteria from responding to the selective pressure, thereby preventing the growth of resistant bacterial populations.
C4: A gene transfer-disabling antibiotic should be used in conjunction with an anti-toxicity antibiotic for best results.
C5: When treating a patient with antibiotics, the best results can be obtained by using not just one antibiotic, but many antibiotics. C6: It is necessary for all antibiotics used in multiple-antibiotic treatment to have a very high selective toxicity.
C7: If certain current scientific and technological resources were effectively utilized together, we should be able to create anti-toxicity and gene transfer-disabling antibiotics with species-specific selective toxicity.

M1: (the main point of the paper, which I derived from my listing of conclusions above. This is also my abstract.)
A drug therapy consisting of anti-toxicity and gene transfer-disabling antibiotics would bring not only the relief of symptoms caused by bacterial toxins, but would also put an effective block against the further spread of antibiotic-resistance genes amongst bacteria. If multiple anti-toxicity and gene transfer-disabling antibiotics designed for the same infection were created with a very high selective toxicity, the probability of any bacteria surviving will be pushed closer to zero than other drug therapies are currently capable of doing. The promise of this new drug therapy

lies in two areas. First, the therapy circumvents the central problem of antibiotic resistance by keeping resistant bacteria from responding favorably to artificial selection by antibiotic treatment. Second, the therapy will allow the alleviation of particular infection symptoms caused by bacterial toxins without the risk of having resistant toxin-producing bacteria propagate. These two areas represent things that current antibiotics are not yet able to do. Although the realization of this new drug therapy may seem out of reach, the genomes of many strains of infectious bacteria have already been fully sequenced. Along with this have come many other advances in technological and computing areas, including pharmacologists' recent ability to use computer software to visualize and design molecules that will bind to a variety of chemical components within or on the surface of a cell. With more research, it should also become possible to design and piece together antibiotics that fall under the anti-toxicity and gene transfer-disabling categories.