Risk homeostasis hypothesis: a rebuttal

Brian O’Neill, Allan Williams

In his latest overview published as an “Opinion”, Wilde asserts that “The theory of risk homeostasis (also known as ‘risk compensation’) was primarily developed and validated in the area of road safety”. Nothing could be further from the truth. These so-called theories that purport to explain human behavior in the face of risk are nothing more than hypotheses with a large body of empirical evidence refuting the studies that allegedly validate them.

Risk homeostasis proponents start with the plausible notion that individuals’ perceptions of risk can influence behavior, but then the proponents implausibly extend this notion to develop a theory of universal behavior. Thus, according to the “theory”, if you acquire a brand new car with airbags, which reduces the risk of a life threatening head or chest injury in a serious frontal crash, you will decide to drive your new car with more reckless abandon than your old one because your risk of a serious injury is now lower. You will be unconcerned that your more reckless driving behavior will increase the chances of crashing and damaging your new car because returning to your previous level of injury risk is what you really crave!

Only abstract theoreticians could believe people actually behave this way, and one wonders whether some advocates of risk homeostasis have even thought about their own behavior when they get a new “safer” car.

What about studies cited by proponents that allegedly prove this is how people do behave? After all, Wilde cites an article claiming to show that “airbag equipped cars tend to be driven more aggressively and that aggressiveness appears to offset the effect of the airbag for the driver”. He chooses not to cite the many studies that show significant reductions in driver deaths in airbag equipped cars, thus refuting the work he cites.

The basic postulate of the homeostasis hypothesis is that accident or injury rates remain relatively constant regardless of interventions intended to make a system safer. Thus, when components of the system become safer—for example, safer cars, better highways, etc.—somehow the system users collectively change their behavior so that the risk as measured by a rate stays constant. According to Wilde, this happens through a feedback mechanism “similar to a thermostat”. But unlike a thermostat with a feedback mechanism that is clear and understandable—thermostats send signals to furnaces or air conditioners to produce more heat or cooling—there is no plausible feedback mechanism that can signal individual drivers to take more or less risk to ensure that the rate remains constant.

This hypothesis is so bizarre that a feedback mechanism postulated by Wilde takes the following form: “Each action carries a certain level of injury likelihood such that the sum total of all actions taken by people over one year explains the accident rate for that year. This rate, in turn, has an effect on the level of risk that people perceive and thus upon their subsequent decisions, and so forth". This is how it is supposed to work: we all wait for information on the “accident rate” and then adjust our behavior through some mysterious collective process so that the rate remains constant!

According to Wilde, “the injury rate per head of population is the most relevant”, and he claims that between 1927 and 1987 “the motor vehicle death rate per 100 000 inhabitants showed no clear secular trend, neither upward nor downward”. Presumably Wilde believes a constant per capita rate supports the homeostasis hypothesis. However, he also points out that during this same 60 year period “the death rate per km driven fell by a factor of about 9”. Think about the remarkable feedback mechanism that must have occurred to produce a collective behavior change that kept the per capita rate constant while average annual mileage and total mileage was increasing. How could it happen? Did enough motorists study Accident Facts each year to see if the per capita rate had changed? Or did they get enough information from the “mass media”, as Wilde suggests? Incidentally, the motor vehicle crash death rate per capita in the United States dropped 26% between 1966 and 1987, and has dropped another 18% since then. I wonder how Wilde explains that?

It is not surprising that many other researchers who have studied this issue have concluded that risk homeostasis has no credence as a theory and is almost entirely lacking in empirical support. Haight noted in 1986, “In my view, a sufficient argument against the validity of risk homeostasis is provided by the incoherence of its ‘theoretical’ formulation” (p 364). Evans in 1986 concluded that “there is no convincing evidence supporting it and much evidence refuting it” (p 81), a view Haight considered “generous”. In 1991 Evans further noted that “the tone of advocacy for the claim
Risk homeostasis hypothesis

has been largely philosophical, metaphysical, and theological in nature, unencumbered by the standards, methods, or norms of science, and at times happily abandoning the rigors of Aristotelian logic and the multiplication table" (p 299). 10

Despite such criticism, a few people continue to promote risk homeostasis, and the “debate” about its merits continues. As Evans noted, when the journal Ergonomics devoted most of a 1988 issue to risk homeostasis on grounds there is somehow a legitimate debate, “To me this seems as plausible as devoting an issue to the proposition that the earth is flat simply because a few adherents, who conjure up ad hoc explanations for every piece of contrary evidence, still claim it is, and that the issue must not be considered settled until these believers concede” (p 299). 10

In his overview of risk homeostasis presented here, Wilde offers a few carefully selected examples he claims provide evidence for risk homeostasis. Numerous other studies not mentioned by Wilde come to opposite conclusions. For example, Wilde refers to a study reporting that childproof caps do not decrease poisonings. But convincing studies in the literature indicate they do, and the Consumer Product Safety Commission estimates that since passage of the Poison Prevention Packaging Act requiring child resistant packaging, more than 700 children’s lives have been saved from poisonings by prescription drugs and aspirin alone.11–14 There are many examples involving motorcycle helmets, vehicle design standards, seat belt laws, etc, where the findings run contrary to risk homeostasis theory. 15–23

It would be foolish to think people never adjust their behavior in response to perceived risks, but a more productive approach is to try to determine the conditions under which this occurs. For example, engineering features intended to reduce crash likelihood are often apparent to drivers and provide direct and immediate feedback about the driving task. Features such as improved braking, improved headlight, or vehicle handling characteristics may lead to changes in driving behavior, for example, faster speeds. But even in these circumstances it is inconceivable that the behavior changes, which must vary from driver to driver, will somehow result in a return to some previous injury or death rate. On the other hand, features such as airbags, high penetration resistant windshields, or breakaway signposts that reduce the likelihood of injury in a crash but do not affect the driving task, and of which the driver is in many cases unaware, would not be expected to alter driving behavior. The data are consistent with this hypothesis.

A relatively simple model of the driving task can account for instances in which driver behavior seems to compensate for changes in the risk of automobile travel without the need to hypothesize a variable of perceived risk, or a motive to control risk, or the misinformed notion of risk homeostasis that all changes to vehicles or the traffic system stimulate users to reset safety to its prior level.

Risk homeostasis is not a theory. It is a hypothesis that repeatedly has been refuted by empirical studies. As Evans has noted, it commands about as much credence as the flat earth hypothesis.