The study of Jin et al. “High Dietary Inorganic Phosphate Increases Lung Tumorigenesis and Alters Akt Signaling” published in the American Journal of Respiratory and Critical Care Medicine, January 2009 reports that high consumption of dietary inorganic phosphate increased lung tumorigenesis in lung cancer susceptible mice. Researchers stated that they fed a “normal” amount of inorganic phosphate to one group and a “high” amount of inorganic phosphate to a second group of mice that were all genetically manipulated to be highly susceptible to developing lung cancer. Researchers found that both groups of mice developed lung cancer, but those fed high amounts of phosphate developed more tumors. One must be extremely careful about extrapolating the conclusions of this study to phosphates.

Inorganic phosphates have a long history of safe use in food. For decades numerous toxicology studies have examined the safety of phosphate based food additives. These toxicological studies have been reviewed by several panels of internationally recognized experts and form the science upon which worldwide regulatory approval has been granted to phosphate based food additives. The study by Jin et al is very limited and its results are contrary to numerous toxicological studies, using acute and chronic exposures, which clearly demonstrate the safety of phosphate based food additives. Further, it is not scientifically credible to think that a minor alteration of the diet alone, such as reducing phosphate consumption, would be effective in preventing such a profound disease as lung cancer, which is known to be multifactorial. Furthermore, the American Lung Association's 2008 review on lung cancer reports that smoking is the number one cause of human lung cancer (80-90%) with radon ranking second (9-14%).

Furthermore, the findings are not directly applicable to humans, as mice eat a greater amount of food everyday based on their body weight than humans do. Thus, feeding the same percentage of body weight of phosphates to a mouse would result in a much greater total amount consumed relative to its body weight than it would in a human. (The high dose used in this experimental study with mice would provide at commensurate level of phosphate 60-times higher than the maximum human dietary exposure estimate.)

Furthermore, the study does not specify the type of phosphate the mice were fed and the cation associated with the phosphate salt (e.g., calcium, sodium, potassium, etc.) could have independent effects on the mice.

Additionally, consumption of phosphates in food has not increased over the years, as the authors allege. Phosphate usage in food has remained steady over the past several decades.