

Type 2 Diabetes and Caffeine



There has been a large number of studies done that suggested coffee drinking may reduce the risk of developing type 2 diabetes. This information has been reported by the media which, unfortunately, has ignored the limitations of those studies. They have been based on correlational observations rather than on controlled experimental studies which are considerably more accurate. For comment on this issue see James. D. Lane comments below.

Five controlled experiments were done in 3 separate institutions in which caffeine was given by mouth to individuals and blood tests were subsequently drawn to test for glucose and insulin levels.

Two of the studies were done on people with type 2 diabetes. In both cases the glucose and insulin levels were increased by the caffeine.(1) (2)

Two were done in non-diabetic individuals who had not been coffee drinkers. In both of those situations there was a disruption in carbohydrate (glucose) control. (3) (4)

One study was done in non-diabetic coffee drinkers. It showed that daily caffeine intake reduced insulin sensitivity. (5)

All five of these controlled studies show that caffeine adversely affected the metabolism of sugar. That being the case they cast significant doubt on the claim that coffee drinking might reduce the risk of getting type 2 diabetes.

References

1. Lane JD Endocr Pract 2007 13: 239
2. Robinson LE J Nutr 2004 134: 2528
3. Graham TE 2001 79: 559
4. Dekker MJ 2007 98: 556
5. Mackenzie T 2007 56: 1694

Comments from James D. Lane, PhD

Email of 4-8-08: The epidemiological studies that suggest that coffee drinking may reduce the risk of developing type 2 diabetes share certain limitations that have been ignored by the press. Despite the large number of such studies that provide similar evidence, our conclusions should be guarded, due to these limitations.

1. Studies of this type can only provide evidence of association (correlation) between a “risk factor” and an outcome and can never prove causation. Unfortunately, causal effects are usually attributed, as in the suggestion that coffee prevents diabetes.
2. A critical bit of evidence to support a causal action is the identification of a plausible biological mechanism through which coffee drinking could act to prevent diabetes. Although much has been made of the possible benefits of antioxidant chemicals present in coffee, no one has shown that these antioxidants are actually absorbed by the body, or that they act to improve the body’s antioxidant status. This research remains to be done, and without the evidence this mechanism is only a possibility.
3. My major concern is related to a simple flaw that may be present in most (or all) of the studies. Most of the studies included only adults of 40+ years, with average ages over 50 years. Individuals who already had type 2 at the start of the study were always excluded from the sample, because the study focused only on “new” cases of diabetes.

However, a significant proportion of type 2 diabetes appears by age 40, so many diabetics may be excluded. If caffeine promotes the development of type 2 diabetes, as I have suggested, coffee drinkers would be likely to develop the disease sooner than those who abstain, perhaps before the minimum age of the study sample. Then all of the people who had developed diabetes as a result of coffee drinking would be excluded from the study at the start. During follow-up, the rate of new diabetes would be lower in coffee drinkers (because many of the coffee drinkers had already gotten the disease prior to the start of the study). This situation is a standard problem from epidemiological cohort studies of disease risk factors. Remember that none of the studies were designed to test the effects of coffee, the data collected for other purposes were simply reanalyzed. That means that many of the specific issues related to possible coffee effects have never been part of the study planning.

The main point is that caffeine (coffee) may have done its damage before the age when people would be eligible to enter these studies. If caffeine caused early type 2, these studies would not see its effect, and might see the reverse. This makes sense to me.

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