

**Denise Robertson, PhD, RNutr.**

Denise Robertson has a PhD in Clinical Nutrition from the University of Newcastle and is a registered Nutritionist. Her interest has always been in the complex interplay between the gastrointestinal tract and whole-body physiology; nutrition is implicated now as both the “cause” and “treatment” for many conditions such as obesity and type 2 diabetes. After working in Oxford for 7 years she now forms part of the Metabolic Research Team at Surrey University, working entirely in human models of human disease. In addition to nutritional research, she has active links with chronobiology researchers looking at the effects of sleep and clock genes on diabetes risk in addition to the role of gut microbiota in diabetes. She has won awards for her translational work; The Nutrition Society David Cuthbertson

Medal (2006), the Association for the Study of Obesity Young Achiever Award (2008) and the University of Surrey researcher of the year (2011).

**Clinical evidence – Findings from human trials**

Before the positive results from animal models can be fully utilised in terms of a public health strategy to reduce the incidence of obesity and/or type 2 diabetes, robust translational clinical evidence is required. Clinical studies in healthy individuals, those at increased risk of type 2 diabetes (T2D) and finally those with T2D have been undertaken using various doses of RS, and for variable supplementation periods. Accepting the caveat of increased heterogeneity within any human population, several key and important consistencies have been demonstrated with important ramifications for human health. RS intake in humans improves insulin sensitivity using gold-standard methodology, an effect which appears to be tissue specific; beneficial effects are noted on skeletal muscle, adipose tissue and pancreatic function, however there is limited evidence for an effect on hepatic metabolism in humans. Many mechanisms have been proposed following successful pre-clinical work, with evidence suggesting a metabolic link between the gut and periphery, originating from the fermentation of resistant starch by the microbial population.

**Learning objectives:**

1. To understand the evidence for a clinical benefit of RS intake in humans
2. To appreciate which effects noted in animals have not been translated into human
3. What mechanisms can we provide empirical evidence for?