Dietary bread crust advanced glycation end products bind to the receptor for AGEs in HEK-293 kidney cells but are rapidly excreted after oral administration to healthy and subtotally nephrectomized rats. 

In renal HEK-293 cells, the dietary Maillard reaction compounds casein-linked Nepsilon-carboxymethyllysine (CML), CML, bread crust (BC), and pronyl-glycine (a key compound formed in association with the process-induced heat impact applied to bread dough) all showed activation of p38-MAP kinase. Expression of the C-terminus truncated receptor for advanced glycation end products (RAGE) resulted in a reduction of HEK-293-MAP kinase activation. As these findings suggested a RAGE-mediated activating effect of CML, BC, and pronyl-glycine on kidney cellular signal transduction pathways, an in vivo study was performed. Male Wistar rats were subjected to a sham operation (CTRL, n = 20) or to 5/6 nephrectomy (NX, n = 20). Both groups were randomized into two subgroups and fed 20 g of a diet containing either 25(%) by weight BC or wheat starch (WS). GC-MS analyses of CML, carboxyethyllysine (CEL), and pentosidine revealed increased levels of CML and CEL in the liver but decreased levels of CML in the
kidneys of CTRL and NX rats fed the BC diet compared to those on the WS diet. However, urinary levels of CML were also elevated in the CTRL and NX rats on the BC diet, pointing to enhanced excretion of AGEs after BC administration. Although renal insufficiency in the NX rats was reflected by proteinuria, the renal handling of CML and, presumably, other AGEs was not impaired.