

Type 2 diabetes gene-environment interactions report examples

[Health & Medicine](#), [Diabetes](#)



Manifestations and Outcomes Type 2 Diabetes: Gene-Environment Interactions, Manifestations and Outcomes

Type 2 diabetes is a polygenic form of diabetes, so various environmental and genetic factors have been linked to its onset, further complications, and clinical outcomes. Scientific research estimates that the heritability of type 2 diabetes is 40 percent if one parent has type 2 diabetes and 70 percent if both parents have type 2 diabetes (as cited in Lin & Sun, 2010). The exact modes of inheritance in type 2 diabetes are currently unknown. Research shows that several ethnic groups have high prevalence of diabetes developed through genetic factors, but various genes have been linked to type 2 diabetes development, such as the TCF7L2, IGF2BP2, CDKAL1, or SLC30A8 gene, and it is not clear how they maintain homeostasis, interact, and contribute to diabetes development (Zeggini, 2007).

Manifestation

The main characteristic of type 2 diabetes is hyperglycemia. The most common physiological causes underlying hyperglycemia are impaired insulin secretion and peripheral insulin resistance (Crandall, 2010). Polyuria, excessive thirst, dehydration, weakness, hypotension, fatigue, polyphagia, weight loss, nausea, vision impairment, and opportunistic pathogens are the most common manifestations of hyperglycemia in diabetes patients (Crandall, 2010).

Physiological changes. Diabetes is caused by several metabolic changes that are often grouped under the common term “metabolic syndrome.”

Researchers argue that diabetes diagnosis should cover all aspects of the metabolic syndrome, including insulin resistance, hypertriglyceridemia, obesity, hypertension, central adiposity, and hypoalphalipoproteinemia, because all those pathophysiological changes can contribute to diabetes development (Hanson, Imperatore, Bennett, & Knowler, 2002). Overall, the exact pathogenesis of type 2 diabetes is complex and further research is required for clinicians to understand it completely.

Behavioral changes. Psychological disorders are often comorbid with type 2 diabetes, but it is not clear whether psychological disorders precede type 2 diabetes or develop after diabetes onset. Some researcher indicates that antipsychotic and antidepressant medication can be accountable for inducing the adverse health effects responsible for the metabolic syndrome development (Newcomer et al., 2002). Others indicate that type 2 diabetes patients have higher risks for developing depression disorders because approximately 50 percent of type 2 diabetes patients develop a major form of depression (Lustman, Griffith, Freedland, Kissel, & Clouse, 1998).

Prognosis and Outcomes

It is estimated that 30 percent of the population suffers from diabetes, and 90 percent of all diabetes cases are type 2 diabetes (Feinglos & Bethel, 2008). Possible complications of diabetes include retinopathy, nephropathy, and neuropathy; those complications are associated with mortality rates between 50 and 60 percent (Crandall, 2010). Gene manipulation therapy is currently investigated in type 2 diabetes, but the translation of clinical research into practical application is currently impossible because diabetes is

a heterogeneous disorder (Stumvoll et al., 2005). Further research is required to clarify the roles of various loci in homeostasis, their interactions, and contributions to diabetes development.

Environmental factors are often associated with encouraging type 2 diabetes onset in people with genetic susceptibility, and most treatment strategies rely on encouraging lifestyle changes to prevent complications and adverse health outcomes in diabetes patients. For example, medical nutrition therapy was associated with improving fasting plasma glucose levels and blood glucose levels (Henry, Scheaffer, & Olefsky, 1985); physical activity was associated with a decrease in diabetes incidence by 58 percent (Tuomilehto et al., 2001). Most importantly, patient education, active clinical interventions, and frequent blood glucose monitoring are the main preventive measures for decreasing adverse complications in diabetes patients (Zimmet et al., 2001).

Crandall, J. P. (April 2010). Diabetes mellitus. The Merck Manual. Retrieved from http://www.merckmanuals.com/professional/endocrine_and_metabolic_disorders/diabetes_mellitus_and_disorders_of_carbohydrate_metabolism/diabetes_mellitus_dm.html?qt=&sc=&alt=

com/professional/endocrine_and_metabolic_disorders/diabetes_mellitus_and_disorders_of_carbohydrate_metabolism/diabetes_mellitus_dm.html?qt=&sc=&alt=

Feinglos, M. N., & Bethel, A. (Eds.). (2008). Type 2 diabetes mellitus: An evidence-based approach to practical management. Totowa, NJ: Humana Press.

- Hanson, R. L., Imperatore, G., Bennett, P. H., & Knowler, W. C. (2002). Components of the "metabolic syndrome" and incidence of type 2 diabetes. *Diabetes*, 51(10), 3120-3127.
- Henry, R. R., Scheaffer, L., & Olefsky, J. M. (1985). Glycemic effects of intensive caloric restriction and isocaloric refeeding in noninsulin-dependent diabetes mellitus. *The Journal of Clinical Endocrinology & Metabolism*, 61(5), 917-925.
- Lustman, P. J., Griffith, L. S., Freedland, K. E., Kissel, S. S., & Clouse, R. E. (1998). Cognitive behavior therapy for depression in type 2 diabetes mellitus: A randomized, controlled trial. *Annals of Internal Medicine*, 129, 613-621.
- Newcomer, J. W., Haupt, D. W., Fucetola, R., Melson, A. K., Schweiger, J. A., Cooper, B. P., & Selke, G. (2002). Abnormalities in glucose regulation during antipsychotic treatment of schizophrenia. *Archives of General Psychiatry*, 59(4), 337-345.
- Stumvoll, M., Goldstein, B. J., & VanHaefen, T. W. (2005). Type 2 diabetes: Principles of pathogenesis and therapy. *Lancet*, 365, 1333-1346.
- Tuomilehto, J., Lindström, J., Eriksson, J. G., Valle, T. T., Hämäläinen, H., Ilanne-Parikka, P., Usitupa, M. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine*, 344(18), 1343-1350.
- Zeggini, E. (2007). The new era for type 2 diabetes genetics. *Diabetic Medicine*, 24(11), 1181-1186.

Zimmet, P., Alberti, K. G., & Shaw, J. (2001). Global and societal implications of the diabetes epidemic. *Nature*, 414, 782-787.