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Preventing Disease with Diet and Exercise

Khursheed N. Jeejeebhoy, MBBS, PhD, FRCPC Presented at the University of Toronto's Primary Care Today: Educational Conference and Medical Exposition, Toronto, Ontario, May 2007.

Over the last century, our lifespan has increased by 30 years from 50- to 80- years-of-age. However, this increase in lifespan has come at a cost which often means a long, but not necessarily healthy life. We have disabled seniors guzzling handfuls of pills each day with several plastic and metal prosthetic devices residing in their bodies. In contrast to this, we also have 90-year-olds who are active and free of disease. We refer to them as having "good genes." **The question is whether we are doomed to depend on our genes or can we change our environment; thus change the response of our genetic self to be healthy?** This article will show **evidence-based information** which indicates that we can live a long and healthy life **by modifying the action of "harmful genes."**

Environment

I was struck by the effect of our environment on individuals when visiting Australia. **The aboriginal population living in the bush were healthy and strong. Those living in an urban community, such as Alice Springs, were diabetic, hypertensive and had coronary artery disease (CAD).** The same genes but different environment made all the difference in their health.

Similarly, **persons living in remote villages in the Dominican Republic have longer lifespans than those living in the city.**

The level of exercise along with the type and amount of dietary intake in the two environments is what makes the difference. To illustrate this point, a randomized controlled trial in patients with stable angina[1] showed that **exercise training was better than angioplasty in terms of event-free survival, better ability to exercise and work and, more importantly, the diameter of the coronary artery became smaller with time in the angioplasty group as compared with the exercise group.** In these patients, lifestyle change was **better than a modern marvel of technology.**

In another context, the effect of diet components on all causes of death, cardiovascular (CV) and cancer deaths was studied in a Greek population of 22,043 adults who were followed for 44 months.[2] A 10-point score for adherence to a Mediterranean diet (Table 1) was used to determine the effect of diet on outcome. Over the 44 months, there were 275 deaths. **A two point increase in adherence to a Mediterranean diet was associated with a 25% reduction in overall mortality as well as a similar reduction in mortality due to CV disease and cancer.**

Table 1
Definition of a Mediterranean diet

- **High intake of:**
- Vegetables
- Legumes
- Fruits and nuts
- Cereal
- Fish
- **Low intake of:**
- Meat
- Poultry
- Dairy products
- A **high monounsaturated-to-saturated fat ratio**
- **Moderate alcohol intake**

A single-blind randomized, controlled trial of in 1,000 patients with angina pectoris, MI or surrogate risk factors for CAD was performed. Four-hundred and ninety-nine patients were allocated to **a diet rich in whole grains, fruits, vegetables, walnuts and almonds.** Five-hundred and one patients consumed a controlled local diet similar to the Step I *National Cholesterol Education Program* (NCEP) prudent diet. [3] The results showed that **total cardiac endpoints were significantly fewer in the intervention group than in the control group** (39 vs. 76 events, $p < 0.001$). **Sudden cardiac deaths were also reduced** (six vs. 16, $p = 0.015$), **as were nonfatal MIs** (21 vs. 43, $p < 0.001$).

The role of diet and exercise
It is obvious that diet and exercise can have profound effect on hard endpoints such as death. The question is how does it work?

Metabolic syndrome
The major cause of morbidity and mortality in Western countries and, increasingly with the rise of prosperity in countries such as India and China, is the Insulin Resistance Syndrome or Metabolic syndrome. This is defined as fasting hyperinsulinemia alone or with fasting blood glucose levels > 6.1 mmol/L with **at least two** of the following:

1. Abdominal obesity of waist-to-hip ratio > 0.9
2. Dyslipidemia defined as triglyceride levels > 1.7 mmol/L or HDL-C < 0.9 mmol/L
3. BP > 140/90 mmHg or on antihypertensive drugs

The major peripheral site of insulin resistance is skeletal muscle. Glucose disposal by muscle in insulin-resistant subjects is reduced due to reduced glucose transport-phosphorylation and reduced glycogen synthesis. [4] In contrast, **adipose tissue is not insulin resistant** and the high insulin-glucose levels promote fatty acid synthesis from glucose and deposition in adipose tissue.

Effect of exercise on insulin resistance
Aerobic exercise at 65% of maximum volume (V) O2 for **45 minutes three times a week for six weeks** increases glucose transport into muscle and glycogen synthesis to well within the normal range. **Aerobic exercise makes an individual with genetic insulin resistance insulin sensitive.**

Resistive or strength training exercise
The effect of aerobic exercise has been indicated above in reducing insulin resistance. The other form of exercise is resistive or musclebuilding. **This form of exercise involves movements against resistance or weights for different muscle groups.** It has been shown that a reduced insulin resistance was found in subjects 50- to 80-years-of-age who performed this type of exercise for **1.25 hours, three times a week.** [5] The exercise trained different muscle groups at **80% of maximum resistance tolerated for 24 repetitions.**

Another study found that resistive exercises have major benefits in musculoskeletal health. Subjects (87-years-of-age) **who could not walk** were randomized to placebo exercise or to resistive exercise of the extensor muscles for 45 minutes three times a week. **Resistive exercise significantly improved** stair climbing and self-mobility due to **improved strength and balance of wasted muscles** in these otherwise chair-bound persons. [6]

Effect of diet on insulin resistance
The effect of feeding a carbohydrate source on blood insulin and glucose levels varies depending on the chemical structure of the carbohydrate. **Soluble carbohydrates, such as sugar or glucose, are rapidly absorbed and result in marked increases in blood glucose and insulin levels.** The **same amount of carbohydrate fed a raw starch results in a small increase in blood glucose and insulin levels.** The ratio of the area under the curve of the blood glucose response over two hours, after feeding a given source of carbohydrate

(e.g., starch), divided by the area when fed glucose, is called glycemic index. [7] **The lower the glycemic index, the slower the release of carbohydrate from the given food with consequent lower blood glucose and insulin** levels. **Eating low glycemic index foods reduces the insulin response to a given carbohydrate and prevents hyperlipidemia.** [5]

Effect of weight loss on insulin resistance
In addition to the unique effects of dietary components and exercise, the third way of altering insulin resistance is weight reduction, which in this context means the **reduction of abdominal fat**. However, **being overweight is not the same as insulin resistance. Weight reduction only benefits patients who are not only overweight but also insulin-resistant.** This fact is illustrated by a study of 24 obese women who were tested for insulin resistance by infusing a standard amount of glucose and insulin and measuring their steady-state blood glucose levels. In the insulin-sensitive individuals, the mean steadystate blood glucose was 3.8 mmol/L, whereas in the insulin-resistant women of the same degree obesity and who were infused at the same rate with both glucose and insulin, their blood glucose was markedly elevated at 12.1 mmol/L. Both groups were given **a calorie-restricted diet** and lost about 8 kg of weight. On repeating the insulin clamp, the steady-state blood glucose in the sensitive group did not change but **the resistant group showed a significant fall in steady-state glucose levels** from 12.1 mmol/L to 8 mmol/L. [8]

Diet composition and weight loss
Diets cause weight loss by simple mass balance, namely if energy restriction, in relation to requirements, causes weight loss. **The main reasons why diets do not work is that reducing intake causes hunger and, ultimately, a desire to eat more. The successful diet among other factors should reduce intake when the person is allowed to eat the given diet ad libitum.** Two trials compared energy intake (with a fat restricted diet soluble carbohydrate [sugar and refined starches] diet) with a fat restricted **complex carbohydrate** and a control group (usual food choices). **All were permitted to be eaten ad libitum.** [9,10]

Complex carbohydrate diet
The complex carbohydrate diet resulted in a lower intake of calories as compared with the others by about 300 Kcal/day. These trials showed that a **low-fat, high-complex carbohydrate diet resulted in greater weight loss, lower cholesterol and a stable triglyceride level in blood;** whereas a **low-fat, high simple carbohydrate diet increased energy intake resulting in no change to weight or cholesterol and increased triglycerides in the blood.** From these trials it can be concluded that **for weight control**, it is necessary to **take a diet rich in complex carbohydrates and low in sugars or refined carbohydrates.**

Protein
Another dietary component which reduces intake of energy when allowed to be eaten ad libitum is protein. **Higher protein intakes, as given in the Atkins diet, clearly reduce intake and increase weight loss** in comparison to Zone and Learn diets which restrict fat intake and the Ornish diet which almost eliminates fat in the diet. [11] It was shown that **obese subjects following the Montignac diet (rich in protein with 30% energy intake) ad libitum consumed significantly less energy** than those randomized to a American Heart Association Step 1 diet, which is low in fat but has only 15% protein. [12]

Diet and hypertension
The Dietary Approaches to Stop Hypertension (DASH) study showed that **a diet rich in fruits, vegetables and milk (high in potassium and calcium) significantly reduced BP.** [13]
Furthermore, **even when this diet was eaten, an additional reduction in BP was achieved by reducing sodium intake to 50 mmol q.d.** [14]
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Do diet and exercise alter outcome?
While it is clear that diet, exercise and weight loss will reduce insulin resistance, will these measures alter morbidity and mortality? Bijnen, et al [15] followed a cohort of 472 men for five years. The effect of **walking or cycling for 20 minutes, three times per week resulted in a risk reduction for mortality** of 0.44 (CI 0.25 to 0.80). **Past activity was not protective**. In a 16 year cohort study, Sandvik, et al [16] showed a **graded reduction in mortality with increasing physical activity.** The effect of diet on mortality has been discussed earlier. [2,3]

Conclusion

The data presented above indicate that individuals with a personal or family history of diabetes, CAD and hypertension would benefit from **avoiding soluble carbohydrates**, eating a **diet high in proteins** mainly from milk and fish and **eating fruits and vegetables ad libitum** which **adds up to the Mediterranean diet**. In addition, **regular aerobic exercise, raising the pulse to 65% to 70% of the maximum, three times a week and resistive exercise to avoid muscle loss with ageing:**

- **improves function,**
- **reduces the risk of diabetes,**
- **reduces the risk of CAD and**
- **may prolong life.**

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Dr. Jeejeebhoy is an Emeritus Professor of Medicine, University of Toronto and a Physician, St. Michael's Hospital, Toronto, Ontario.