Global Change Needed to Curb Obesity Pandemic

BY SARA FREEMAN

FROM THE LANCET

LONDON – The global obesity pandemic needs addressing on a global level with governments around the world taking the lead, according to a series of papers published in the Lancet.

The series is published ahead of the United Nations High-Level Meeting on Noncommunicable Diseases being held in New York next month. This U.N. meeting is set to discuss what can be done to prevent and control four key disease areas – cardiovascular diseases, cancers, chronic respiratory diseases, and diabetes – for which obesity is a known contributing or exacerbating factor.

"In many ways, the holy grail for governments today, certainly in the United Kingdom, across Europe and the United States, is improving health care while at the same time reducing costs; that's the paradox of modern health care today," said Dr. Richard Horton, editor of The Lancet, during a press briefing to highlight the Lancet Obesity Series.

Dr. Horton added: "So far, obesity had been stubbornly resistant to traditional public health approaches, and what this series tries to do is to look at the reasons why that might be and how we might get out of that public health 'cul-de-sac' that we are in today."

The first article in the series (Lancet 2011;378:804-14) looks at the factors that might have contributed to the rising rates of obesity seen around the globe in the past 4 decades. As rates of obesity have risen steadily and simultaneously in many countries, "that points to global drivers," said the paper's lead author Dr. Boyd Swinburn, professor of population health and the director of the World Health Organization Collaborating Centre for Obesity Prevention at Deakin University, Melbourne.

"There is quite a lot of evidence coming out that this is largely driven be changes in the food system," which produces a food supply comprising "increasingly processed, available, affordable, and highly promoted, 'tasty' food," Dr. Swinburn observed.

With both high- and low-income countries affected, the solution appears to lie more with policy changes to try to improve individuals' local environments rather than try to change the public's behavior.

"Support for individuals to counteract obesogenic environments will continue to be important, but the priority should be for policies to reverse the obesogenic nature," Dr. Swinburn and his colleagues reported.

The lead author of the second article (Lancet 2011;378:815-25), Dr. Y. Claire Wang, noted that if historical trends continue, there could be 65 million more obese adults in the United States alone by 2030, with 11 million more obese adults projected at the same time point in the United Kingdom.

This would bring the total of obese individuals to be 164 million and 26 million, in each country, respectively and, taken together, could result in 6 million to 8.5 million new cases of diabetes, 5.7 million to 7.3 million cases of heart disease and stroke, and 492,000-669,000 additional cases of cancer in both countries.

"We project that the medical costs will be substantial," said Dr. Wang, of the department of health policy and management at Columbia University, New York. Indeed, the cost of treating essential preventable diseases that result from obesity are estimated to increase by $48 billion to $66 billion per year in the United States and by £1.9 billion to £2 billion per year in the United Kingdom by 2030.

However, a 1% reduction in body mass index across the U.S. population could avoid up to 2.1 million to 2.4 million new cases of diabetes, 1.4 million to 1.7 million cardiovascular diseases, and 73,000-127,000 cases of cancer by 2030. Similar reductions could be achieved in the United Kingdom.

"Of course, what we have to do is to reverse this [rising obesity trend]," said Kevin Hall, Ph.D., the lead author of the third article in the series (Lancet 2011;378; 826-37). But it may not be as simple as "eat less, exercise more" suggested Dr. Hall, a senior investigator at the National Institute of Diabetes and Digestive and Kidney Diseases.

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References: 1. Fuchs GS, Mikkelsen S, Knudsen TK, Kappelgaard A-M. Ease of use and acceptability of a new pen device for the administration of growth hormone therapy in pediatric patients: an open-label, uncontrolled usability test. *Clin Ther.* 2009;31:2906-2914. **2.** Norditropin® FlexPro® [Instructions for Use]. Princeton, NJ: Novo Nordisk Inc; 2010. **3.** Data on file. PDS290 pen-injector for Norditropin® SimpleXx® container closure system: comparison to Norditropin NordiFlex®. Princeton, NJ: Novo Nordisk Inc; 2009.

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Standard guidance for weight loss is to reduce caloric intake by 500-1,000 kcal/day to lose 1-2 pounds per week, but this is fundamentally wrong. "The reason it's wrong is that it would only be true if nothing else happened to your metabolism," Dr. Hall maintained.

Dr. Hall's research shows that for every 10 calories that can be cut from the diet per day, there would more likely be a drop of just 1 pound over 3 years, so not eating a 250-calorie chocolate bar per day could would lead to a 25-pound weight loss over 3 years.

Dr. Hall presented a web-based model that could one day help clinicians advise their patients on how many calories would need to be cut from the diet and the level or exercise needed every day to attain a certain weight loss. Currently, the model can only be used as a research tool and will need to be updated as new data become available.

With all these new data and improved understanding of what causes obesity and its health care burden and costs, the lead author of the fourth and final article (Lancet 2011;378;838-47) in the series said that it's time for governments and policy makers to act.

"Governments certainly need to lead obesity prevention, but so far few have shown any leadership whatsoever," commented Steven L. Gortmaker, Ph.D., of the department of society, human development, and health at Harvard School of Public Health in Boston.

Dr. Gortmaker and his colleagues noted that the U.N. meeting on noncommunicable diseases "is an important opportunity for the international community to provide the leadership, global standards, and cross-agency structures needed to create a global food system that offers a healthy and secure food supply for all."

The research was conducted under the auspices of the Collaborative Obesity Modeling Network as part of the Envision Project and supported by the National Collaborative on Childhood Obesity Research, a collaboration of the National Institutes of Health, the Centers for Disease Control and Prevention, the United States Department of Agriculture, and the Robert Wood Johnson Foundation. The NIH and the NIDDK provided additional funding.

All authors declared that they had no conflicts of interest. ■

Indications and Usage

Norditropin® (somatropin [rDNA origin] injection) is indicated for the treatment of children with growth failure due to inadequate secretion of endogenous growth hormone, the treatment of children with short stature associated with Noonan syndrome or Turner syndrome, the treatment of children with short stature born small for gestational age (SGA) with no catch-up growth by age 2-4 years, and for the replacement of endogenous growth hormone in adults with growth hormone deficiency (GHD) who meet either of the following two criteria: 1. Adult Onset: Patients who have GHD, either alone or associated with multiple hormone deficiencies (hypopituitarism), as a result of pituitary disease, hypothalamic disease, surgery, radiation therapy, or trauma; or 2. Childhood Onset: Patients who were growth hormone deficient during childhood as a result of congenital, genetic, acquired, or idiopathic causes.

Important Safety Information

Somatropin should not be used to treat patients with acute critical illness due to complications following open heart or abdominal surgery, multiple accidental trauma or acute respiratory failure as increased mortality may occur.

Somatropin is contraindicated in patients with Prader-Willi syndrome who are severely obese, have a history of upper airway obstruction or sleep apnea, or have severe respiratory impairment. There have been reports of sudden death when somatropin was used in such patients. Norditropin® is not indicated for the treatment of patients who have growth failure due to genetically confirmed Prader-Willi syndrome.

Somatropin should not be used or should be discontinued with any evidence of active malignancy. Any preexisting malignancy should be inactive and its treatment complete prior to instituting therapy with somatropin. Somatropin should be discontinued if there is evidence of recurrent activity. Patients with preexisting tumors or GHD secondary to an intracranial lesion should be monitored routinely for progression or recurrence. In childhood cancer survivors, an increased risk of a second neoplasm, particularly meningiomas in patients treated with radiation to the head for their first neoplasm, has been reported in patients treated with somatropin.

Somatropin should not be used in patients with active proliferative or severe non-proliferative diabetic retinopathy, for growth promotion in pediatric patients with closed epiphyses, or in patients with known hypersensitivity to somatropin or any of its excipients.

Somatropin may decrease insulin sensitivity particularly at higher doses in susceptible patients. Glucose levels should be monitored periodically, including close monitoring of patients with preexisting diabetes or glucose intolerance. Doses of anti-hyperglycemic drugs (insulin or oral agents) may require adjustment for patients with diabetes on somatropin therapy.

Intracranial hypertension (IH) with papilledema, visual changes, headache, nausea and/or vomiting, usually occurring within the first eight (8) weeks after initiation of somatropin therapy, has been reported in a small number of patients. In all reported cases, rapid resolution has occurred after therapy cessation or a reduction of dose. Funduscopic examination should be performed routinely before and during somatropin therapy. If papilledema is observed, somatropin treatment should be discontinued.

Fluid retention during somatropin replacement therapy in adults may frequently occur. Clinical manifestations of fluid retention are usually transient and dose dependent.

In patients with GHD, central (secondary) hypothyroidism may first become evident or worsen during somatropin treatment. Periodic thyroid function tests are recommended and thyroid hormone replacement therapy should be initiated or adjusted as needed.

Slipped capital femoral epiphysis may occur more frequently in patients with endocrine disorders (including GHD and Turner syndrome) or with rapid growth. Onset of a limp or complaints of hip or knee pain in somatropin patients should be carefully evaluated. Rapid growth may also result in progression of preexisting scoliosis. Patients with a history of scoliosis or skeletal abnormalities, which may be present in untreated Noonan, Turner or Prader-Willi syndrome, should be monitored.

Patients with Turner Syndrome should be evaluated carefully for otitis media and other ear disorders since these patients have an increased risk of ear and hearing disorders. Somatropin treatment may increase the occurrence of otitis media in patients with Turner syndrome. Somatropin may also increase the risk of IH in Turner patients. In addition, patients with Turner syndrome should be monitored closely for cardiovascular disorders (e.g., stroke, aortic aneurysm/dissection, hypertension) as these patients are also at risk for these conditions.

Congenital heart disease is an inherent component of Noonan syndrome. Though a clinical study in Noonan syndrome reported no evidence of somatropin-induced ventricular hypertrophy or exacerbation of preexisting ventricular hypertrophy (as judged by echocardiography), the safety of Norditropin® in children with Noonan syndrome and significant cardiac disease is not known.

Cases of pancreatitis have been reported rarely in children and adults receiving somatropin treatment, with some evidence supporting a greater risk in children compared with adults. Girls who have Turner syndrome may be at greater risk than other somatropin-treated children. Pancreatitis should be considered in any somatropin-treated patient, especially a child, who develops abdominal pain.

Other somatropin-related adverse reactions include injection site reactions/rashes, lipoatrophy and headaches. Subcutaneous injection of somatropin at the same site repeatedly may result in tissue atrophy and can be avoided by rotating the injection site.

Somatropin inhibits 11ß-hydroxysteroid dehydrogenase type 1 (11ßHSD-1) in adipose/hepatic tissue, and may significantly impact the metabolism of cortisol and cortisone. In patients treated with somatropin, previously undiagnosed central (secondary) hypoadrenalism may be unmasked requiring glucocorticoid replacement therapy. In addition, patients treated with glucocorticoid replacement therapy, especially with cortisone acetate and prednisone, for previously diagnosed hypoadrenalism may require an increase in their maintenance or stress doses.

Careful monitoring is advisable when somatropin is administered in combination with other drugs known to be metabolized by CP450 liver enzymes (e.g., corticosteroids, sex steroids, anticonvulsants, cyclosporine) as limited published data suggest somatropin may alter clearance of these compounds.

In adult women on oral estrogen replacement, a larger dose of somatropin may be required to achieve the defined treatment goal.

The safety and effectiveness of Norditropin® in patients age 65 years and older has not been evaluated in clinical studies. Elderly patients may be more sensitive to the actions of somatropin and may be more prone to develop adverse reactions.

Please see Brief Summary of Prescribing Information on the following pages.

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