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Awareness of pathophysiological concepts of type 2 diabetes—A survey in 847 physicians

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Abstract

Aims: The aim of the study was to determine physicians' knowledge of specific concepts generally implicated in the pathophysiology of type 2 diabetes (T2D).
Methods: A multiple choice online survey was completed by 847 physicians, of which 516 were engaged in primary care (PCP) and 331 in specialized care (SCP) in the US, the UK, Germany and France (3–30 years in practice, at least 40 patients with T2D). A continuous rating system was used to measure familiarity (''totally familiar'' to ''never heard of'') or agreement with a statement (from ''totally agree'' to ''totally disagree'').
Results: The term ''insulin resistance'' was recognized by 74% of PCPs and 90% of SCPs ($p < 0.05$) and 76% felt that it was ''a key but not the sole determinant of T2D''. Only 47% agreed that ''beta cell dysfunction is a key determinant of T2D onset'' and 57% agreed with ''beta cell dysfunction being a key determinant of T2D progression''. Even among SCPs, 6% were not familiar with the term ''beta cell dysfunction'' (16% among PCPs, $p < 0.05$). The overall familiarity with the following terms was: 55% with ''beta cell dysfunction'', 56% with ''beta cells'', 38% with ''glucagon'', 32% with ''alpha cells'', 55% with ''hepatic glucose output'', 15% with ''incretins'' and 18% with ''GLP-1''. SCPs were significantly more familiar with all terms than PCPs (all p-values <0.05).
Conclusions: The pathogenetic role of beta cell dysfunction in the onset and progression of T2D did not seem to be well established. ''Insulin resistance'' was a well known concept even among PCPs, while ''hepatic glucose output'', ''pancreatic alpha cells'' and ''glucagon'' were not. Incretin hormones and GLP-1 were widely unknown. This may effect prescribing behaviour and how well an individual's therapy is based on pathophysiology.

Keywords: Beta cell dysfunction; Insulin resistance; Incretins; GLP-1; Glucacon

1. Introduction

Type 2 diabetes mellitus (T2D) has become an epidemic disease and there are probably very few physicians who are not confronted with diagnosis and treatment of this disease. Insulin insensivity and pancreatic islet dysfunction are attributed to play a major role in the development, onset and progression of diabetes [1].

Abbreviations: T2D, type 2 diabetes; PCP, primary care physician; SCP, specialized care physician; NGT, normal glucose tolerance; IGT, impaired glucose tolerance; GLP-1, glucagon like peptide; GIP, glucose-dependent insulinotropic peptide; DPP-IV, dipeptidyl peptidase-IV

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Table 1
Speciality of physicians

Specialty	Total	France	Germany	UK	USA
PCP	516	104	108	100	204
SPC	331	75	76	76	104
Total	847	179	184	176	308
Number of years in practice (mean ± S.D.)	17 ± 6.8	18 ± 7.4	18 ± 6.1	17 ± 6.8	17 ± 6.8

Insulin resistance is an early phenomenon partly related to obesity and is defined as a decreased biological response to insulin [2–4]. A number of driving mechanisms have been proposed including elevated non-esterified fatty acids, inflammatory cytokines, adipocytokines, and mitochondrial dysfunction [5–9]. In contrast, pancreatic beta cell function declines steadily in the progress from normal glucose tolerance (NGT) to impaired glucose tolerance (IGT) and finally overt T2D, even before the onset of clinical hyperglycemia [10,11]. Glucotoxicity, lipotoxicity, and amyloid formation hadesly beta cell dysfunction [12–14].

Drug therapy based on the pathophysiologic concepts should be superimposed on lifestyle changes with weight loss and exercise [15,16]. Therapeutical options which target hyperglycaemia by enhancing tissue insulin sensitivity are metformin and the thiazolidinediones. The sulfonylureas and the shorter acting secretagogues, i.e. the ''glinides'', improve pancreatic insulin secretion [17].

Novel therapeutic approaches involve incretin hormones which are secreted in a nutrient-dependent manner [18]. The GLP-1 analogues exendin-4 or liraglutide and the DPP-IV-inhibitors (''gliptins'') as incretin enhancers appear to stimulate insulin secretion effectively, inhibit glucagon secretion, promote proliferation and inhibit apoptosis of beta-cells [19–21]. The alpha-glucosidase inhibitor acarbose reduces intestinal glucose absorption and, to a minor degree, stimulates GLP-1 release [22,23].

For rationale-based management of T2D it is necessary to understand the basic principles of the pathophysiology of T2D. The aim of the present study was to determine the knowledge of physicians, both specialists and non-specialists, of specific concepts generally implicated in the pathophysiology of T2D.

2. Methods

A multiple choice online survey was sent to 4616 physicians in France, Germany, the UK and the US with a total response rate of 35.6% ($N = 1645$). Out of those, 1326 physicians met the eligible criteria and entered the questionnaire. However, only 847 physicians answered the questionnaire completely (51.5%, USA, $N = 308$, UK, $N = 179$, Germany $N = 184$ and France $N = 179$), of which 516 were engaged in primary care (PCP) and 331 in specialized care (SCP) (Table 1). Eligibility criteria were at least three and less than 30 years in medical practice and a minimum of 60 patients with type 2 diabetes (T2D) treated within the last month for specialists and 100 patients for general practitioners. Diabetologist, endocrinologist or physicians with a specialty in diabetes care were defined as specialized care physicians (SCPs). Exclusion criteria were participation in another research initiative in the previous three months. Cardiologists, nephrologists and other subspecialists not engaged in diabetes treatment were not contacted.

A continuous rating system was used to measure familiarity with a presented concept (ranking from 1 = ''totally familiar'' to 4 = ''never heard of before'') or an agreement with a statement (from 1 = ''totally agree'' to 7 = ''totally disagree''). The physicians were reassured that their answers were treated anonymously and they should keep in mind that there were no right or wrong answers.

Differences between PCPs and SCPs were assessed using T score statistics.

3. Results

The term ''insulin resistance'' was recognized by 74% of PCPs and 90% of SCPs (total 80%, $p < 0.05$) and 76% felt that it was ''a key but not the sole determinant of T2D''. Only 47% agreed with ''beta cell dysfunction is a key determinant of T2D onset'' and 57% considered ''beta cell dysfunction as being a key determinant of T2D progression'' (Table 2).

The overall familiarity with the following terms were as follows: 55% with ''beta cell dysfunction'', 56% with ''beta cells'', 38% with ''glucagon'', 32% with ''alpha cells'', 55% with ''hepatic glucose output'', 15% with ''incretins'' and 18% with ''GLP-1''. Even among SCPs, 6% were not familiar with the term ''beta cell dysfunction'' (16% among PCPs, $p < 0.05$). This is correlated with the knowledge of ''beta cells''. Only 61% of the specialists and 24% of GPs were totally familiar with the term 'glucagon' hormone, even less with the term 'alpha cells' (both PCPs versus SCPs $p < 0.05$).