2 diabetes, almost no correlation between BMI and non-fasting blood glucose levels was found [15]. Fasting blood glucose, glycohemoglobin, EI and oral medication use were not determined simultaneously in these studies. Our analyses, while revealing that the differences in BMI or EI did not reflect the averaged glycemic control status of patients, also support the earlier studies and provide evidence of relationships between BMI and EI and parameters such as serum lipids and blood pressure. It is also clear from our analyses that it is impossible to ascertain the glycemic control status of an individual patient from a single assessment of their BMI or EI. The lack of significant differences in EI and the relatively small differences in physical activity in the face of a large discrepancy in BMI seen in our patients (Table 1) suggested that lifestyle-related factors play relatively limited roles in determining the current BMI of the patients.

Glycemic control was poorly correlated with BMI while blood pressure and serum lipids showed significant step-wise elevations with increased BMI (Table 1), despite all three parameters reportedly improving with weight loss in intervention studies of diabetic patients [1]. This suggests that the relationship between obesity and hyperglycemia is quite complex. The higher proportion of insulin therapy, and also the longer diabetes duration and the lower fasting plasma insulin levels, seen in patients in the lower BMI categories (Table 1) suggest that Japanese diabetic patients have quite limited insulin secretory capacity and are becoming obese only during the early stages of the disease. This supports previous speculation that the disease process profoundly influences the BMI of patients [4] and could also explain the much lower average BMI in Japanese patients than in white patients [9,10].

Several potential sources of bias need to be considered in interpreting our data. One is BMI-dependent underreporting of energy intake, which has been observed mainly in 24 h dietary recalls [16–18], but also in food frequency questionnaires [18]. However, we combined food recording with FFQ in our dietary survey and observed differences in energy intake (Table 2) that were much broader than the reported BMI-dependent effect (20–25% over the BMI range of approximately 10 kg/m^2) [18]. Another limitation of this study is that we only included patients who had completed a dietary survey for analysis. Neither could we discuss the role of ethnicity because comparable analyses of other ethnic groups could not be found. Such a comparison would have aided our understanding of the underlying pathophysiological relationship between energy intake and obesity in patients with type 2 diabetes and the influence of genetic background on the pathophysiology of the disease.

Acknowledgments

This study was financially supported by the Health Sciences Research Grants (Research on Health Services) provided by the Ministry of Health, Labor and Welfare, Japan. We gratefully acknowledge all the patients, physicians, co-medical staffs and secretaries who are taking part in the JDCS.

Appendix A

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