

Journal of Nutritional Biochemistry 13 (2002) 539-542

Total salivary IgA, serum C3c and IgA in obese school children

Anabel Pallaro^a, Susana Barbeito^b, Patricia Taberner^b, Patricia Marino^b, Alejandra Franchello^b, Irene Strasnoy^b, Olga Ramos^b, Nora Slobodianik^{a,*}

^aDepartment Nutrition, School of Pharmacy and Biochemistry, University of Buenos Aires, Buenos Aires, Argentina ^bService of Nutrition and Diabetes, Dr. Pedro de Elizalde Hospital, Buenos Aires, Argentina

Received 5 June 2001; received in revised form 8 March 2002; accepted 12 April 2002

Abstract

Studies of the immunologic function in adult obese humans and experimental models indicate that excess adiposity is associated with impairments in host defense mechanisms. The aim of this work was to analyze the secretory and humoral immune system in obese children (n = 105, 55 boys, 50 girls), between 6 and 13 years of age. Samples of non-stimulated saliva and whole blood were collected from fasting patients. Total salivary IgA (IgAsal), serum C3 complement (C3c) and Immunoglobulin A (IgA) were determined by quantitative radial immunodifussion on agar gel layers (Diffu-plate, Biocientífica SA). Results, expressed as mg/dl, were compared to laboratory reference values from healthy children of either sex in the same range of age that belong to the same socioeconomic class (n = 60). Data (Mean \pm 1 SD) of the whole population were: IgAsal: 11.4 ± 4.8 vs 14.8 ± 6.9 ; C3c: 190.7 ± 53.1 vs 126.3 ± 45.5 ; IgA: 194.5 ± 101.5 vs 157.2 ± 19.9 . Data distribution showed higher frecuencies near the zone of the highest reference values for serum C3c; when results of IgA and IgAsal were expressed as percentage of the mean reference value, 51% and 48.6% of the whole studied population presented data lower than 100% and 75% respectively. These results show a compromised secretory immune system without incidence of clinical symptoms and infections, whereas humoral immunity might not be profoundly affected. © 2002 Elsevier Science Inc. All rights reserved.

Keywords: Total salivary IgA; Serum C3c and IgA; Obesity; Children

1. Introduction

Nutritional status may modify the metabolic response to infection or injury and may influence morbidity and mortality [1,2,3].

Obesity, the major public health problem and a common type of malnutrition in developed countries, may be associated with altered host defense mechanisms, being infection a frequent primary or contributing cause of death in this nutritional pathology [1,4,5].

Epidemiologic and clinical information suggest that the incidence and severity of infectious illness are higher in obese than in non-obese individuals [5,6,7,8].

In adults with severe uncomplicated obesity, normal concentrations of serum immunoglobulins (IgG, IgA, IgM, IgD) and complement components (C3, C4) with normal levels of secretory IgA and lysozyme in tears were observed [9]. Apparently, the T and B subpopula-

tions of lymphocytes were the same for the obese individuals and normal controls. However, it was reported that the immune response to immunization against the hepatitis B virus was decreased by obesity. Other investigators reported that the *in vitro* release of MIF (macrophage migration inhibitory factor) by lymphocytes was decreased while the mitogen induced blastogenic response of pheripheral blood lymphocytes was significantly diminished in obese subjects [5,8,10,11].

Finally, 38% of children and adolescents showed reduction of intracellular bacterial killing by polymorphonuclear leukocytes and variable immune responses in vivo and in vitro [12].

Despite such findings, which suggest that immunocompetence may be altered in obesity, little is known about specific components of the immune system in childhood obesity. Definitive studies on the effects of overnutrition on immune system function in humans are lacking [1,8]

The aim of this report was to analyze the secretory and humoral immune system, through the determination of total salivary IgA and serum levels of C3 complement and Immunoglobulin A, in obese school children.

^{*} Corresponding author. Tel.: +54-11-4-9648243; fax: +54-11-4-9648243.

E-mail address: nslobo@ffyb.uba.ar (N.H. Slobodianik).

^{0955-2863/02/\$ -} see front matter © 2002 Elsevier Science Inc. All rights reserved. PII: S0955-2863(02)00198-5

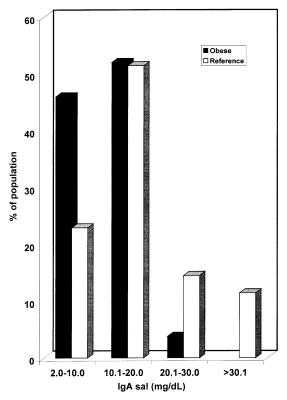


Fig. 1. Distribution of Total salivary IgA (mg/dl) in obese and healthy non-obese children.

2. Material and methods

2.1. Subjects

The study included one hundred and five obese children (50 girls and 55 boys)—diagnosed at the Service of Nutrition and Diabetes, Dr. Pedro de Elizalde Hospital, Buenos Aires, Argentina—, between 6 and 13 years of age and without concomitant pathologies.

The study was approved by the Ethics Committee of the University of Buenos Aires and all participants gave informed consent before recruitment into the study.

2.2. Anthropometric assessment and laboratory analyses

The anthropometric assessment of patients included present weight and height and it was calculated the relative body weight as the ratio between present weight/ideal body weight (RBW) RBW: weight/(50th percentile weight/ height) and were expressed as percentage. Obese children were defined as those with ratios higher than 120 according to our National Life Standard Tables [13]. The diagnosis of obesity was performed by the physicians (expert in nutrition) of the Service of Nutrition and Diabetes, Dr. Pedro de Elizalde Hospital. Obese children with clinical or hormonal disorders were not included in this study.

Samples of whole blood and non-stimulated saliva were collected from patients after a 12–15 hs fasting. Total sali-

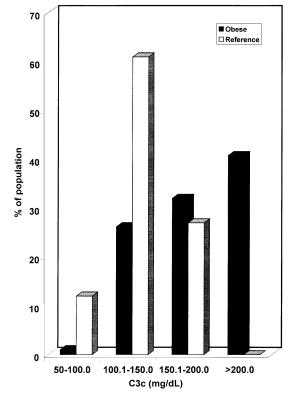


Fig. 2. Distribution of C3c serum levels (mg/dl) in obese and healthy non-obese children.

vary IgA (IgAsal), serum C3 complement (C3c) and Immunoglobulin A (IgA) were measured by single radial immunodiffusion techniques on agar gel layers (Diffu-Plate, Biocientífica SA, Buenos Aires, Argentina) [14].

For comparison, healthy non-obese children (n = 60) in the same age range, who belonged to the same socioeconomic class, were recruited to obtain the laboratory reference values.

All results were expressed as mean \pm SD. Statistical analysis was performed by the Student's t-test using a computer program [15].

3. Results

The results of RBW were: boys: 139.2 \pm 19.5; girls: 138.4 \pm 13.2.

Biochemical data, expressed as mg/dl, are presented in Table 1. No statistical differences were observed between boys and girls' biochemical parameters, so the populations were unified.

Statistical differences at a level of p < 0.05 were observed for total salivary IgA; obese children showed the lowest values. Fig. 1 shows that 46% of the population present values lower than 10 mg/dl.

Statistical differences at level of p < 0.01 were observed in C3c serum levels in the obese group when compared with



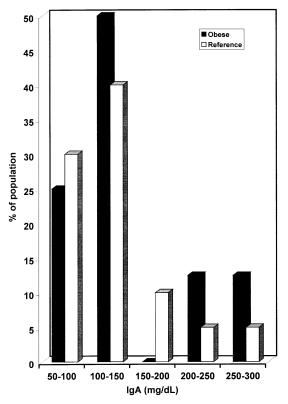


Fig. 3. Distribution of serum IgA levels (mg/dl) in obese and healthy non-obese children.

laboratory reference values; data distribution showed higher frecuencies near the zone of the highest reference values (Fig. 2).

No statistical differences were observed in IgA concentration between obese children and reference values. Data distrubution is presented in Fig. 3.

Moreover, when the results of IgA and IgAsal were expressed as percentage of the its respective mean reference value, as suggested by Cahen et al. [16]; 51% and 48.6% of the whole studied population presented data lower than 100% and 75% respectively (Figs. 4 and 5); on the other hand, 94.3% of the whole population showed values higher than 100% for C3c. (Fig. 6).

4. Discussion

Table 1

It is accepted that nutrient imbalance—deficit or excess—affects immune responses. Therefore, the relationship

Total salivary IgA, serum C3c and IgA of obese children and reference values.

Group	IgAsal	C3c	IgA
Girls (n = 50) Boys (n = 55) Whole population Reference	$\begin{array}{c} 11.8 \pm 5.1 \\ 11.1 \pm 4.6 \\ 11.4 \pm 4.8 \\ 14.8 \pm 6.9 \end{array}$	$189.6 \pm 100.5 \\ 192.0 \pm 55.0 \\ 190.7 \pm 53.1 \# \\ 126.3 \pm 45.5 \\$	$\begin{array}{c} 188.1 \pm 100.5 \\ 200.3 \pm 102.9 \\ 194.5 \pm 101.5 \\ 157.2 \pm 19.9 \end{array}$

Mean \pm SD; * p < 0.05; # p < 0.01.

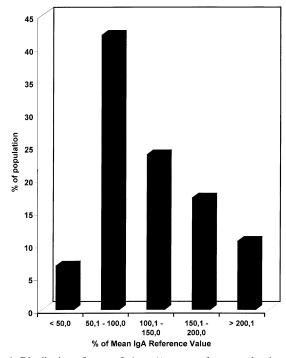


Fig. 4. Distribution of serum IgA as % mean reference value in obese children.

between excess adiposity and immunity has been studied primarily in animal models of obesity, particularly in genetically obese rodents. In humans, clinical survey data have provided information about the relationship between obesity and immunologic function and few studies have

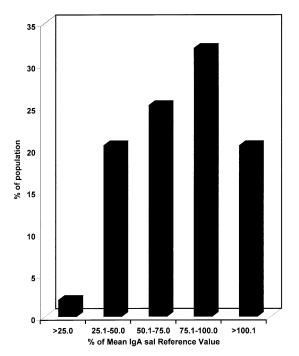


Fig. 5. Distribution of total salivary IgA as % Mean reference value in obese children.

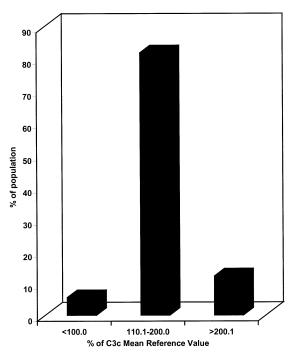


Fig. 6. Distribution of serum C3c as % mean reference value in obese children.

compared specific immune responses in obese and nonobese individuals [1,2,4,7]

Our study, performed on obese children without clinical concomitant complications, belonging to the same socioeconomic class and without environmental differences nor social customs with the reference group, show a compromised secretory immune system evaluated through the levels of total salivary IgA; this fact could explain the higher incidence of respiratory tract infections that have been reported, by other investigators, in obese infants [17]. However, no apparent symptoms of infections were observed in the studied population.

The serum concentrations of IgA and C3c suggest that humoral immunity might be not profoundly affected; moreover, C3c values were increased in the 94.3% of the whole population (Fig. 6).

It was reported by other authors, that serum levels of alternative complement pathway proteins (C3c, Factor B) in obese patients were 1.5–2 fold higher than in controls; it seems that body weight itself plays a role in determining levels of these components. It might be that factors influencing body weight and its changes, contribute to control the serum levels of these complement proteins, due to altered production in adipose tissue and/or other sites [18]. On the other hand, it is known that adipose cells can synthesize essential components of the alternative pathway including C3 and B [19]. These reports could explain the higher values of C3c that we have observed in this study.

As the relationship between obesity and immune function is multifactorial and complex, *involving neuroendo*- *crine, dietary, metabolic and psychological factors,* further studies will be required to clarify this issue.

Acknowledgments

This study was supported in part by University of Buenos Aires (Grants B 003) and Biocientífica SA (Buenos Aires, Argentina).

References

- R.K. Chandra, Nutrition and the immune system: an introduction, Am J Clin Nutr 66 (1997) 460S–463S.
- [2] N.S. Scrimshaw, P. SanGiovanni, Synergism of nutrition, infection and immunity: an overview, Am J Clin Nutr 66 (1997) 464S–477S.
- [3] P. Fraker, Impact of nutritional status on immune system (Chapter 12), in: M. Gershwin, J.B. German, C.L. Keen (Eds.), Nutritional Immunology: Principles and Practice, Humana Press Inc., Totowa, New Jersey, USA, 2000, pp. 147–156.
- [4] D. Stallone, The influence of obesity and its treatment on the immune system, Nutr Rev 52 (1994) 37–38.
- [5] P.A. Davis, J.S. Stern, Obesity and Immunity (Chapter 24), in: M. Gershwin, J.B. German. C.L. Keen (Eds.), Nutritional Immunology: Principles and Practice, Humana Press Inc., Totowa, New Jersey, USA, 2000, pp. 295–300.
- [6] A. Must, J. Spadano, E.H. Coakley, A.E. Field, G. Colditz, W.H. Dietz, The disease burden associated with overweight and obesity, JAMA 282 (1999) 1523–1529.
- [7] M.E. Keith, K.N. Jeejeebhoy, Immunonutrition, Baillieres Clin Endocrinol Metab 11 (1997) 709–738.
- [8] D.C. Nieman, D.A. Henson, S.L. Nehlsen-Cannarella, M. Ekkens, A.C. Utter, D.E. Butterworth, O.R. Fagoaga, Influence of obesity on immune system, J Am Diet Assoc 99 (1999) 1512–1516.
- [9] D.N. Mc Murray, P.A. Beskitt, S.R. Newmark, Immunologic status in severe obesity, Int J Obesity 6 (1982) 61–68.
- [10] J. Hirokawa, S. Sakaue, S. Tagami, Identification of macrophage migration inhibitory factor in adipose tissue and its induction by tumor necrosis factor-alpha, Biochem Biophys Res Commun 235 (1997) 94–98.
- [11] S. Tanaka, S. Inoue, F. Isoda, Impaired immuity in obesity: suppressed but reversible lymphocyte responsiveness, Int J Obesity Relat Metab Disorders 17 (1993) 631–636.
- [12] R.K. Chandra, K.M. Kutty, Immunocompetence in obesity, Acta Paediatr Scand 69 (1980) 25–30.
- [13] M. Cusminsky, E. Castro, G. Lozano, J. Lejarraga, Tablas nacionales de peso, estatura y perímetro cefálico desde el nacimiento hasta los 12 años de edad, Arch Arg Pediat 79 (1980) 291–295, 445–446.
- [14] G. Mancini, A.O. Carbonara, G.F. Heremans, Immunochemical quantitation of antigen by single radial immunodiffusion, Immunochemistry 2 (1965) 235–254.
- [15] D. Schwartz, Methodes statistiques a l'usage des medecins et des biologistes, Medicales Flammarion, Paris, 1963.
- [16] L. Cahen, D. Hellio, J.D. Berville, O. Schilliger, Le profil proteique ciblé nutritionnel, Méd et Nut XXVII (1991) 360–362.
- [17] B. Hutchinson-Smith, The relationship between the weight of an infant an lower respiratory infections, Med Office 123 (1970) 257– 262.
- [18] C. Pomeroy, J. Mitchell, E. Eckert, R. Raymond, R. Crosby, P. Dalmasso, Effect of body weight and caloric restriction on serum complement proteins, including factor D/adipsin: studies in anorexia nervosa and obesity, Clin Exp Immunol 108 (1997) 507–515.
- [19] L.N. Choy, B.S.Rosen, B.M. Spiegelman, Adipsin and an endogenous pathway of complement from adipose cells, J Biol Chem 267 (1992) 12736–12741.