NOMBRE:.....
Nº REGISTRO:.....

1º PARCIAL DE INGLÉS TÉCNICO

PRE-READING (1)

1. Read the title. Scan the text for acronyms. What do they stand for?
Scan the text for 12 key words. Advance a general reading hypothesis.

2. Scan the first sentence of each paragraph in the Background section. Write a specific reading hypothesis.

WHILE READING (5)

3. Read the text and complete the chart showing differences between MNA and SGA. (2)

MNA:	SGA:

4.Answer the following questions:

a. How would you define the concept of "functional capacity"? What does it involve? (1)

b. What is the conclusion of the paper?Provide a complete answer. (1)

NOMBRE:.....
Nº REGISTRO:.....

5. Select one connector that you consider very important in this text. Explain its use and the ideas connected. (1)

Connector: _____ Lines:_____
Logical meaning: _____

.....

.....

AFTER READING (4)

6. Write the main idea of the text in one well-written sentence. Be specific and concise

Nutritional status and functional capacity of hospitalized elderly

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Introduction

Deterioration of the nutritional status affects and is affected by disease, especially among the elderly [1]. Nutritional diagnosis and the identification of factors that contribute to this diagnosis are, therefore, essential but complex processes. This complexity is due to the occurrence of many changes, both physiological and pathological, which may be taken as inherent to the aging or disease process. However, indirect indicators that likely guarantee proper and healthy eating, such as economic, social, lifestyle and quality of life aspects may represent important tools for assessing nutritional risk [2]. I

The MNA (Mini Nutritional Assessment) [3] has been an extensively used method to identify risk of malnutrition in the elderly and in those that may benefit from early intervention. The MNA is a simple, low cost and non-invasive method that can be done at bedside [3]. Added MNA scores allow one to screen the elderly who have an adequate nutritional status, those who are at risk of malnutrition and those who are malnourished. The MNA consists of anthropometric and global indicators, including information on eating patterns and self-perception of health, such as: reduced food intake; weight loss of >3 kg body weight; mobility, bed- or chair-bound; psychological stress; neuropsychological problems; body mass index; inability to live independently; taking >3 prescription drugs; having pressure sores or skin ulcers; number of full meals eaten per day; consumption of high-protein foods; consumption of fruits & vegetables; amount of liquids consumed per day; inability to feed self; difficulty in self-feeding; self-view of nutritional status; self-view of health status; mid-arm circumference <21 cm; and calf circumference <31 cm [3]. The tool has been successfully used to assess the nutritional risk of elderly who live independently, receive home care services or are institutionalized, and of patients who are chronically ill, frail, have Alzheimer's disease or cognitive impairment [4]. (...) II

There are at least 40 screening and assessment tools for subjective nutritional status assessments, and some are for the general population and others for specific populations [11]. The most broadly used of these population-specific tools is the Subjective Global Assessment (SGA), developed by Baker et al in 1982 [3]. The SGA has proven to be one of the most efficient methods to determine nutritional status and make the prognosis of clinical complications [12]. Different from the MNA, the SGA was developed to assess hospitalized individuals, investigating recent weight loss, changes in food consumption, gastrointestinal symptoms, loss of functional capacity, disease-associated stress, and depletion, found upon physical examination [12]. III

Thus, the SGA focuses mainly on the effect of the disease on nutritional status. When the same population of elderly individuals is assessed by the SGA and MNA, the SGA detects already established malnutrition more precisely, while the MNA detects those who need preventive care [13]. (...)Thus, the MNA is considered a very useful instrument for assessing long-term nutritional risk but not as useful for short-term prognoses [15]. Regarding functional autonomy, the MNA considers the mobility of the elderly, if bedbound or wheelchair-bound or if he or she is capable of walking but does not leave the home. The MNA does not assess eating autonomy, that is, if the elderly can prepare his or her own food, if he or she eats without help, if he or she can cut the foods and even if he or she can bring the foods to the mouth. IV

Functional capacity assessment based on self-reported performance of daily tasks was first assessed by Katz, 1963 [16]. The multidimensional OARS (Older V

Americans Research Survey) [17] questionnaire was validated and has been used in Brazil [18] for some time now. The questionnaire takes into account the basic activities of daily living (ADL) and the instrumental activities of daily living (IADL). The lack of functional autonomy to look after oneself and to prepare and eat foods is a factor that can result in malnutrition and deserves the attention of professionals and family since functional capacity assessment can be an indicator of nutritional risk which is particularly associated with food intake [19].

The prognosis of elderly inpatients depends not only on the acute physiological conditions inherent to the disease but also on a number of preexisting factors, such as loss of functional independence, loss of cognitive functions, low body weight [20] and corrected arm muscle area [21]. Poor eating habits are predictive of a bad hospitalization prognosis among the elderly [1], suggesting that there is a relationship of interdependence with the other factors. Thus, the objective of this work was to assess the relationship between nutritional status and indicators of functional capacity among recently hospitalized elderly in a general hospital. VI

Discussion

This study presents data on the outcome of a study that assessed the relationship between nutritional status and functional capacity of hospitalized elderly and there is clearly the need to improve the knowledge on the mechanisms of association between these factors (nutritional and functional states). VII

(...)

The global MNA nature allows the inclusion of important factors which do not only classify the nutritional status but also indicate when intervention is necessary to guarantee proper care. Inadequate food intake is the cause of malnutrition while physical and cognitive limitations can prevent adequate food intake [20]. Cereda et al., 2008 [27], showed that the poorer functional status was associated with low BMI, sarcopenia and reduced oral intake and the MNA reliably identifies at-risk institutionalised elderly needing higher standards of care, particularly related to eating. Routine documentation of oral intakes and feeding assistance might be useful to prevent weight loss, sarcopenia and functional status deterioration. VIII

The large variability is due to differences in level of dependence and health status among the elderly. In hospital settings, a low MNA score is associated with an increase in mortality, prolonged length of stay and greater likelihood of discharge to nursing homes. Malnutrition is associated with functional and cognitive impairment and difficulties eating. The MNA detects risk of malnutrition before severe change in weight or serum proteins occurs [4]. IX

Functional capacity is interconnected with the quality and quantity of food consumed. The IADL include shopping and preparing meals. In the present study, malnourished individuals were 6 times more dependent on others to shop and prepare meals than those that were adequately nourished (Table 3). Being unable to buy and prepare meals not only interferes with the amount of food ingested but also with the diversity, which may result in boring and unattractive meals. Among the ADL, partial or complete dependence of more than half of the malnourished individuals (Table 3) to eat warn us of the importance to assess the functional capacity while providing nutritional care, as corroborated by the results of a study [28] done with 130 Japanese older than 65 years, where those (48) who totally depended on others to move around were also the ones with the lowest indicators of nutritional status (anthropometry, albumin and food intake). X

There is an interrelationship between nutritional and functional statuses. It has already been shown that malnutrition compromises the functional status of the individual [29]. At the same time, functional status impairment increases vulnerability and may affect food consumption negatively [19]. Functional capacity assessment tools have been included in studies that seek to assess nutritional risk [30]. XI

The MNA is a screening and assessment tool with a reliable scale and clearly defined thresholds, usable by health care professionals. It should be included in the geriatric assessment and is proposed in the minimum data set for nutritional XII

110	interventions4. This study reinforces the importance of the MNA as an instrument to assess the nutritional status of the elderly since it represents a global assessment instrument. It also warns us of the need to pay special attention to functional capacity indicators and food intake among the elderly when planning care for this group, especially when they are debilitated by disease. **Conclusion** A relationship of interdependence between nutritional status and functional status was observed among the studied elderly. Deterioration of the nutritional status was associated with reduced food consumption, recent weight loss, disease-associated stress, degree of self-sufficiency, and functional capacity. The IADL and ADL showed that malnourished elderly were more impaired regarding the activities of daily living, which emphasizes the importance of nutrition. Malnutrition prevalence among the elderly admitted to the hospital was high, probably because of their vulnerability before the disease. Nutritional status deterioration is accompanied by reduced functional capacity. Thus, it is necessary to pay special attention to functional capacity when planning nutritional care for this group, especially when they are debilitated by disease.	XIII

BMI: Body mass index

Nombre y apellido: .....

N° Registro: .....

Fecha: .....

INGLÉS – EXAMEN LIBRE

Before Reading (2)

1. Select two strategies to approach the text and justify your choice. Apply them.
2. State a general reading hypothesis.
3. Select a third strategy, justify your choice and apply it.
4. State a specific reading hypothesis.

While Reading (4)

1. Summarize the purpose and the results in your own words. Be precise, concise and clear.
2. How do the authors interpret the results? Be precise, concise and clear.
3. Select two very important logical connectors and justify your choice.
4. Group 10 (ten) lexical items semantically connected and establish the lexical domain for this paper.

After Reading (4)

1. Write the main idea of the text in a sentence. This sentence has to be complete, specific, and well written.

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Do Resistin and Tumor Necrosis Factor-α Relate to Changes in Insulin Resistance in Normal Pregnancy?

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Abstract: Problem statement: The purpose of this study was to evaluate the role of resistin and Tumor Necrosis Factor-α (TNF-α) in insulin resistance during pregnancy. **Approach:** Serum resistin and TNF-α concentrations were measured by ELISA in 86 healthy pregnant women (26, 23 and 37 of them in the 1st, 2nd and 3rd trimesters, respectively) and in 21 healthy non pregnant women in a cross sectional study. **Results:** Resistin concentration was significantly higher in the third trimester (9.5±3.3 ng mL^{-1}) as compared with non pregnant women (7±3.3 ng mL^{-1}). Serum TNF-α level were also significantly increase in pregnant women (2.6±1.9 pg mL^{-1}) as compared with maternal healthy controls (0.8 ±0.7 pg mL^{-1}). There were significant correlation between gestational age and BMI ($r = 0.28$, $p = 0.01$), resistin ($r = 0.36$, $p = 0.002$) and TNF-α ($r = -0.44$, $p<0.0001$). There was not significant correlation between gestational age and Insulin Resistance (IR). We also did not found correlation between IR and resistin as well as between IR and TNF-α in pregnant women. **Conclusion:** TNF-α and resistin do not appear to contribute greatly to pregnancy induced insulin resistance in healthy pregnancy.

Key words: Resistin, TNF-α, pregnancy, insulin resistance

INTRODUCTION

Pregnancy is related to glucose metabolism disorders and insulin resistance (Hadden and McLaughlin, 2009; Johnson, 2008; Stanley *et al*., 1998). Insulin resistance may facilitate supply of appropriate nutrients particularly of glucose to fetus for fetal growth and metabolism. The mechanism responsible for insulin resistance has not been clearly stated. Recent researches have been shown adipokinins include leptin (McLachlan *et al*., 2006) resistin (Lu *et al*., 2006; Caja *et al*., 2005) IL-6 (Senn *et al*., 2002) and TNF-α (Zinman *et al*., 1999; Kirwan *et al*., 2002; Melczer, 2002; Gwozdziewiczová *et al*., 2004) play an important role in insulin resistance. TNF-α is one of the most widely studied cytokinins produce by adipose tissue. This cytokinin is also secret by placenta (Zavalza-Gómez *et al*., 2008; Chen *et al*., 1991). TNf-α has an important role in obesity-induced insulin resistance and diabetes (Pereira *et al*., 2006; Zinman *et al*., 1999; Gwozdziewiczová *et al*., 2004; 2005). A few studies suggest that TNF-α may play role in insulin resistance in normal and diabetogenic pregnant women (Pereira *et al*., 2006; Xue-Lian *et al*., 2008; Kirwan *et al*., 2002). Resistin is another protein identified recently as a hormone secreted by adipocytes which has a controversial history regarding its role in the pathogenesis of obesity-mediated insulin resistance and type 2 diabetes (Lu *et al*., 2006; Hasegawa *et al*., 2005; Hivert *et al*., 2008). Insulin sensivity changes from an enhanced state during early pregnancy to an insulin resistant state in late pregnancy (Kirwan *et al*., 2002; Leturque *et al*., 1984; Stanley *et al*., 1998). Therefore it is inspected, subsequent to increase in IR during pregnancy, its related factors change, too. However, at the time of our study a few researches have been done about changes in serum resistin and TNF-α level during different trimesters of normal pregnancy and their relationships to insulin resistance. Therefore, the aim of this study was to examine whether serum TNF-α and resistin concentration change during normal pregnancy and, if so, to relate those changes corresponding alterations in insulin resistance and BMI.

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MATERIALS AND METHODS

This cross sectional study was done on eighty-six pregnant women with different gestational ages (first trimester: 26, second trimester: 23, third trimester: 37 and twenty-one healthy non pregnant women similar in age and BMI (control group: 23.8±0.8, patient groups: 23±0.6). All subjects met the following criteria: No history of pre-gestational diabetes; no history of liver, respiratory, thyroid or other illness and any current infectious condition. They were not on any medical therapy.

Body Mass Index (BMI, Kg m^{-2}) was calculated according to the maternal height and pre-pregnancy weight. Serum samples were analyzed for concentrations of resistin, TNF-α, insulin and glucose. Serum glucose was measured by GOD-POD method. Serum insulin was determined by ELISA (Diaplus). Serum resistin was measured by ELISA (Biovender, Germany Ref NO: RD 191016100R) and also serum TNF-α was assayed by ELISA (Bender med, Austria Ref NO: BMF 223). Insulin resistance value were calculated using the homeostasis model assessment, HOMA-IR, as (fasting insulin IU L^{-1}) × (fasting glucose mmol L^{-1}) /22.5 as previously reported by Matthews *et al.* (1985). All participants in the trial gave informed consent and the study was approved by University Ethics committee.

Statistical analysis: All results are displayed as Mean ± SEM (standard error of mean). Levene's test was shown there was not difference between groups. One way ANOVA analysis and Post hoc tests were used to compare mean among the groups and correlations were calculated using liner correlation (Pearson). Statistical analysis was performed using SPSS 12 for window. $P<0.05$ was considered statistically significant for all analysis.

RESULTS

A total of 86 pregnant women and 21 non pregnant subjects participated in the study. Clinical and laboratory characteristics of patients and controls are summarized in Table 1. BMI were found to be significantly increased in the 3rd trimester as compared with controls and women with 1st trimester of pregnancy (Fig. 1a and Table 2). Patients in the second and third trimester of pregnancy had significantly higher systolic pressure than non pregnant women (Fig. 1b). Serum resistin concentration were found to be significantly raised in the second and third trimester as compared with women with first trimester of pregnancy, but we were not found any statistical difference in serum resistin concentration between the healthy controls and patients with gestational age less than 24 weeks (Table 2 and Fig. 1c). TNF-α level was also significantly higher in patients in all gestational age as compared non pregnant women (Fig. 1d). However, during pregnancy TNF-α level were significantly decreased with increase in gestational age (Table 2). Patients exhibited higher score of HOMA IR compared control group, but there were not difference in this score between pregnant subjects in different gestational age (Table 1 and 2). There were significant correlation between gestational age and BMI ($r = 0.28$, $p = 0.01$), diastolic pressure ($r = 0.28$, $p = 0.01$) resistin ($r = 0.36$, $p = 0.002$) and TNF-α level ($r = -0.44$, $p<0.0001$). There was not significant correlation between gestational age and IR. Resistin level in pregnancy did not correlate with IR, fasting insulin, BMI and body weight. TNF-α level also did not correlate with IR, fasting insulin, BMI and body weight.

Table 1: Clinical and laboratory characteristic of patients and control

	Pregnant women Mean ± SE	Control Mean ± SE
Number of case	86.0	21.0
Age (year)	26.4±0.500	27.20±1.30
Gestational age (week)	23.9±1.200	0.00
HT (m)	1.6±0.007	1.58±0.01
WT (Kg)	65.0±1.300**	58.60±1.50
BMI (Kg m^{-2})	25.4±0.400*	23.40±0.70
SBP (mmHg)	117.0±0.900**	110.70±1.50
DBP (mmHg)	72.8±0.800	70.90±3.40
BGL (mg/100)	81.5±1.700	80.20±1.90
Insulin (μLU mL^{-1})	10.9±0.600	8.70±0.40
Resistin (ng mL^{-1})	8.3±0.300	7.00±0.70
TNF (pg mL^{-1})	2.8±0.200***	0.80±0.10
IR	2.1±0.100**	1.70±0.10

BMI: Body Max Index; HT: Height of women; WT: Weight of body; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BGL: Blood Glucose Level; IR: Insulin Resistance; *: p<0.05 (control); **: p<0.01 (control); ***: p<0.0001 (control)

Table 2: Clinical and laboratory characteristics of pregnant women with different gestational age

	1st trimester Mean ± SE	2nd trimester Mean ± SE	3rd trimester Mean ± SE
Number of cases	26.00	23.00	37.00
Age (year)	25.50±0.87	24.90±0.50	27.60±0.80
GA (week)	11.20±0.30	22.20±2.70	32.90±0.80
WT (Kg)	59.20±2.20	65.70±3.10	68.20±1.80**
HT (m)	1.58±0.01	1.58±0.01	1.60±0.01
SBP (mmHg)	114.20±1.90	117.50±1.80	118.30±1.20*
DBP (mmHg)	70.50±1.50	71.70±1.70	74.70±1.10
BMI (Kg m^{-2})	23.60±0.80	25.70±0.90	26.20±0.60**
BGL (mg/100)	78.60±1.30	79.40±2.30	84.10±3.50
Insulin (μL mL^{-1})	10.50±0.70	10.60±0.80	10.05± 0.60
Resistin (ng mL^{-1})	6.70±0.20	8.60±0.40*	9.50±0.50***
TNF (pg mL^{-1})	3.93±0.40	2.88±0.30*	2.02±0.20***
IR	2.00±0.12	2.02±0.14	2.10± 0.01

BMI: Body Mass Index; GA: Gestational Age; WT: Wight of body during pregnancy; HT: Height; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BGL: Blood Glucose Level; IR: Insulin Resistance; *: p<0.05; **: p<0.01; ***: p<0.001 (significantly different from pregnant women in 1st trimester)

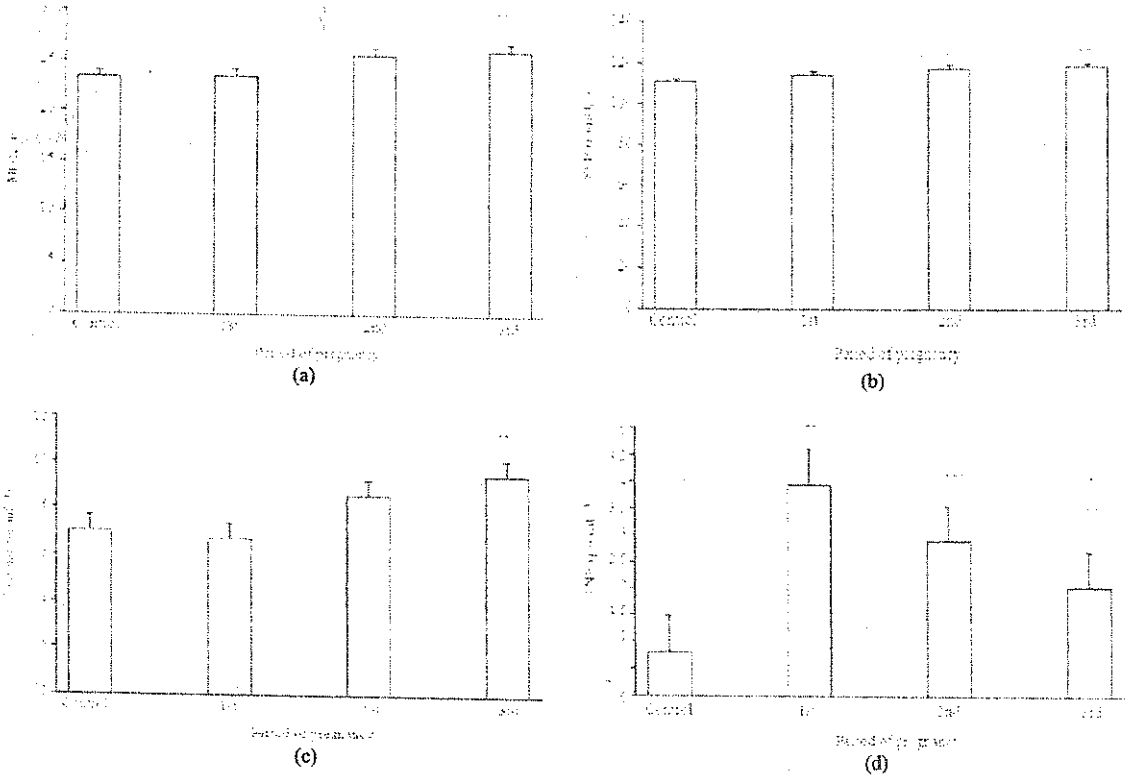

Fig. 1: (a) BMI in patients in different trimesters of pregnancy and controls. BMI were significantly higher in 3rd trimesters compared with the control (**: $p<0.01$). (b) Systolic blood pressure in different trimesters of pregnancy and controls. Systolic blood pressure were significantly higher in the 2nd and 3rd trimesters as compared with control group (*: $p<0.05$, **: $p<0.01$). (c) Serum resistin level in different trimesters of pregnancy and control. Serum resistin level significantly higher in the 3rd trimester as compared control (*: $p<0.05$). (d) Serum TNF level in different trimesters of pregnancy. TNF level significantly higher in the 1st, 2nd and 3rd trimesters of pregnancy as compared control (***: $p<0.0001$, **: $p<0.01$)

DISCUSSION

Glucose metabolism disorder is a common complication during pregnancy and its pathology is associated with IR and deficiency of insulin secretion (Johnson, 2008). In this study, insulin resistance significantly was higher in total group of healthy pregnant than in non pregnant women. In spite of previous report we did not found correlation between gestational age and insulin resistance. Kirwan *et al.* (2002); Melczer (2002); Stanley *et al.* (1998) have been shown insulin resistance was significantly increased in late pregnancy compared with either control or early pregnancy. This difference may be related to differences in dietary composition, life style between western and eastern societies (Clapp, 2006), variability between insulin assays in different experimental researches (Manley *et al.*, 2008), differences in the population studied and sampling time during pregnancy.

A number of studies have been reported concentrations of resistin in pregnant subjects (Palik *et al.*, 2007; Nien *et al.*, 2007; Hendler *et al.*, 2005; Chen *et al.*, 2005). Our findings of higher maternal resistin concentration only in third trimester compared to non pregnant subjects and women in the first trimester are consistent with report by Chen *et al.* (2005) but, Palik *et al.* (2007) reported that differences between resistin concentration of non pregnant and pregnant women are significantly in 1st, 2nd and 3rd trimester. Higher plasma concentration of resistin in second and third trimester of pregnancy compared first trimester as well as the positive correlation with gestational age were showed that placenta has important role in resistin

production during pregnancy. In addition a number of studies have been shown, resistin level decrease after deliver (Megia *et al*., 2008). In present study similar to recent reports, we could not found correlation between maternal resistin and insulin resistance (Megia *et al*., 2008; Kulik-Rechberger and Mora-Janiszewska, 2009; Kuzmicki *et al*., 2009). Our results are in agreement with several previous observations that found an increase in TNF-α level in pregnant as compared non pregnant subjects (Melczer, 2002; Xue-Lian *et al*., 2008; Daher *et al*., 1999) but in contradiction with these studies, we found negative correlation between gestational age and TNF-α level. Explanation account for this finding may be related to lifestyle of our subjects. Clapp and Kiess (2000) reported that regular weight bearing exercise during pregnancy suppressed the usual pregnancy-associated changes in the circulating level of TNF-α (Clapp and Kiess, 2000). In present study, most of patients were villager and naturally had high level of physical activity. In addition pregnancy to be associated with changes of several hormones, including, estrogen, progesterone, cortisol and 1, 25 dihydroxyvitamin D3 (Henricks *et al*., 1972; Smith *et al*., 1973; Ardawi *et al*., 1997). Some of these hormones such as cortisol and catecholamines and 1, 25 dihydroxy D3 are potent inhibitor of TNF-α production by monocyte/macrophage (DeRijk *et al*., 1997; Guirao *et al*., 1997; Anand *et al*., 2009; Ito *et al*., 2002). In other study has been shown, plasma concentration of cortisol was elevated more during late pregnancy than early pregnancy (Kirwan *et al*., 2002). Therefore it is possible increased production of these hormones during pregnancy to be responsible for reduced maternal TNF-α production.

CONCLUSION

In conclusion, our findings suggest that TNF-α and resistin do not appear to contribute greatly to pregnancy induced insulin resistance in healthy pregnancy.

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REFERENCES

Anand, S.P., P. Selvarajand and P.R. Narayanan, 2009. Effect of 1,25 dihydroxyvitamin D_3 on intracellular IFN-γ and TNF-α positive T cell subsets in pulmonary tuberculosis. Cytokine, 45: 105-110. PMID: 19091593

Ardawi, M.S., H.A. Nasrat and H.S. BA'Aqueel, 1997. Calcium-regulating hormones and parathyroid hormone-related peptide in normal human pregnancy and postpartum: A longitudinal study. Eur. J. Endocrinol., 137: 402-409. PMID: 9368509

Caja, S., I. Martinez, M. Abelenda and M. Puerta, 2005. Resistin expression and plasma concentration peak at different times during pregnancy in rats. J. Endocrinol., 185: 551-559. PMID: 15930181

Chen, H.L., Y.P. Yang, X.L., Hu, K.K. Yelavarthi and J.L. Fishback *et al*., 1991. Tumor necrosis factor alpha mRNA and protein are present in human placental and uterine cells at early and late stages of gestation. Am. J. Pathol., 139: 327-335. PMID: 1867321

Chen, D., M. Dong, Q. Fang, H.E. Jing, Z. Wang and Z.X. Yang, 2005. Alterations of serum resistinin normal pregnancy and pre-eclampsia. Clin. Sci. 108: 81-84. PMID: 15377276

Clapp, J.F. and W. Kiess, 2000. Effects of pregnancy and exercise on concentrations of the metabolic markers tumor necrosis factor alpha and leptin. Am. J. Obstet. Gynecol. 182: 300-306. PMID: 10694328

Clapp, J.F., 2006. Effects of diet and exercise on insulin resistance during pregnancy. Metab. Syndr. Relat. Disord., 4: 84-90. PMID: 18370754

Daher, S., F. Fonseca, O.G. Ribeiro, C.C. Musatti and M. Gerbase-DeLima, 1999. Tumor necrosis factor during pregnancy and at the onset of labor and spontaneous abortion. Eur. J. Obstet. Gynecol. Reprod. Biol., 83: 77-79. PMID: 10221614

DeRijk, R., D. Michelson, B. Karp, J. Petrides, E. Galliven and P. Deuster *et al*., 1997. Exercise and circadian rhythm-induced variations in plasma cortisol differentially regulate Interleukin-1 beta (IL-1 beta), IL-6 and Tumor Necrosis Factor-alpha (TNF alpha) production in humans: high sensitivity of TNF alpha and resistance of IL-6. Clin. Endocrinol. Metab., 82: 2182-2191. PMID: 9215292

Guirao, X., A. Kumar, J. Katz, M. Smith, E. Lin and C. Keogh *et al*., 1997. Catecholamines increase monocyte TNF receptors and inhibit TNF through β_2-adrenoreceptor activation. Am. J. Physiol., 273: E1203-8. PMID: 9435537

Gwozdziewiczová, S., R. Lichnovská, J. Hrebícek and F. Cesk, 2004. Tumor Necrosis Factor alpha (TNF alpha) and insulin resistance. Cesk Fysiol., 53: 167-175. PMID: 15704741

Gwozdziewiczova, S., R. Lichnovska, R.B. Yahia, R. Chlup and J. Hřebiček, 2005. TNF-α in the development of insulin resistance and other disorders in metabolic syndrome. Biomed. Pap. Med. Fac. Univ. Palacky Olomouc Czech Repub., 149: 109-017. PMID: 16170397

FACULTAD DE FARMACIA Y BIOQUÍMICA – UBA
1º CUATRIMESTRE DE 2010

NOMBRE:..... Nº REGISTRO:.....
PROFESOR:..... HORARIO:.....

2º PARCIAL DE INGLÉS TÉCNICO

PRE-READING (1)

You have 15 minutes to answer this section. Then your teacher will collect the sheets.

1. Read the title and the bibliography.

2. Find the purpose of the paper and indicate lines.

3. Write a reading hypothesis

FACULTAD DE FARMACIA Y BIOQUÍMICA – UBA
1º CUATRIMESTRE DE 2010

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PROFESOR:..... HORARIO:.....

2º PARCIAL DE INGLÉS TÉCNICO

WHILE READING

4. Scan the Introduction for the following figures and explain what they refer to: (0,80)

30.000	
70.000	
232,090	
30,350	
60 times	
Sixth	
Third	
Half a million	

Answer the following questions:

5. Read the Introduction and state the importance of this study according to the authors. (1)
6. Has PSA testing helped to stop prostate cancer in the Golestan province? (1)
7. Mention the three risk factors for prostate cancer. (1)
8. Mention the two recommendations given to physicians. (0.60)

9. Choose a connector with semantic importance and explain the concepts connected in your own words. (0.60)

Connector:
Line:

Idea:

10. Write the main idea of the text. Be precise and concise. (4)

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ISSN 1553-3468

Prostate cancer incidence in Golestan province, Iran (2004)

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[2]Department of Social Medicine, Golestan University of Medical Sciences

Abstract: Prostate cancer is the most common cancer in men and therefore represents a major problem in public health.The aim of this study was to find and evaluate province-specific estimate of incidence in males by age groups for prostate cancer in Golestan province, Iran. The data used in this study were collected in a cancer registry program that was conducted by Health Deputy of Golestan province in IRAN for a period of 1 year (2004). Prostate cancer data was identified and collected in the population based cancer registries through the 18 Pathology Laboratories (where male populations referred to these centers) and using a structured questionnaire, trained personnel conducted in-person interviews to collect information on prostate cancer in Golestan province. Prostate cancer incidence among males in Golestan province was 5.17/100000 in gerenal. But the highest rate (ASR: 215.87/100,000) among males were showen to be in age 80-85. The incidence of prostate cancer in age 80-84 has risen sharply and it was the lowest in age 50-54 (ASR: 5.18/100,000). According to this information Golestan province harbor a rather incidence for prostate cancer (in age 80-84), comparable to the lower incidence rate reported in the world. For the present time it can be said that prostate cancer in males appear to be one of the most prevalent and serious type of cancer in Golestan province.

Key words: Prostate cancer incidence - Golestan province – Iran

INTRODUCTION

At present, cancer is a serious public health problem in many countries of the world, imposing a large economical and psychological burden as well as loss of life and productivity[1]. Lots of effort and money have been put in the fields of clinical, epidemiological, pharmacological, and biological research on cancer in the recent decades. Cancer is the third most common cause of death in Iran and annually 30000 of Iranian die due to cancer[2]. It is estimated that more than 70000 new cases of cancer *occur* in the country and it is estimated that the cancer incidence in next decade suspected to be increased due to increase in elderly population within the country [3]. Prostate cancer is the second leading cause of male cancer death in most industrialized countries [4].It is the most common cancer diagnosed among men in the United States. In 2005, an estimated 232,090 new cases diagnosed and 30,350 men will die from prostate cancer in the United States [5]. African-Americans have the highest reported rates of prostate cancer in the world (With a worldwide incidence of 25.3 per 100,000), with an age-adjusted rate of 137 per 100,000, which is 60 times higher than the reported incidence in Shanghai, China, where the reported incidence is the lowest in the world (2 per 100,000)[6-7].The distribution of cancers vary significantly from country to country all over the world. The latest estimates of global cancer incidence show that prostate cancer is the sixth most common cancer in the world, the third most common cancer in men, the most common cancer in men in Europe, North America, and some parts of Africa with half a million new cases each year, almost 10% of all cancers in men [8-10]. Prostate cancer incidence is characterized by a very large geographical variability. Asian countries have much lower rates of occurrence of the disease than North American, and north and western European countries, with southern European and South American countries displaying an intermediate incidence rate[11]. Japanese and Chinese men are less likely to develop prostate cancer [12]. The incidence of prostate cancer was lowest among Asians (Japanese, 39/100 000 person-years) and Chinese (28/100 000 person-

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and healthcare personnel; public health workers; or those who have had recognized, unprotected, close-contact exposure to an infected person (confirmed, probable or suspected) during that person's infectious period.

Characteristics of Novel S-OIV A (H1N1) in Humans

Where did the swine influenza virus come from?

Influenza A virus can infect various host species, including birds, humans, and swine. Influenza A H1N1 virus was first isolated from swine in 1930[6] and from humans in 1933.[7] Swine influenza A viruses are antigenically very similar to the 1918 human influenza A virus, and they may all have originated from a common ancestor.[8,9] From 1930 to the late 1990s, swine influenza A viruses were called "classical swine influenza" and they have remained relatively stable antigenically.[10,11]

In around 1998, the classical swine influenza virus resorted with human influenza A H3N2 virus and a North American Lineage avian influenza virus (unknown subtype), which resulted in the emergence of a triple resorted H3N2 swine virus. This resorted virus has been circulating in the swine population throughout North America.[12–14] Also in around 1998, the triple resorted H3N2 virus resorted again with the classical swine influenza virus. This generated two new subtypes of swine influenza A virus, the H1N1 and the H1N2 viruses,[11] which have been circulating in the Asian swine population. Although human and swine H1N1 viruses are all of avian origin, they have evolved in different host species. Antigenic drift has occurred amongst different lineages of H1N1 viruses; therefore, cross-protection antibodies against avian, swine, and human H1N1 viruses are not expected to exist. Indeed, a recent study has demonstrated that ferret post-infection antisera raised against the currently circulating, seasonal human H1N1 viruses did not react with the novel S-OIV, according to a hemagglutination inhibition assay.[15]

The newly emerged S-OIV A (H1N1) contains a combination of gene segments that have not been previously identified in swine or human influenza viruses. The PB2 and PA genes originated from an avian virus that was introduced into swine viruses around 1998. PB1 originated from the human H3N2 virus, which acquired the gene from an avian virus in 1968. HA, NP, and NS genes came from classical swine virus and these three genes are closely related to the 1918 human influenza A virus. The other two genes, NA and M, were from the Eurasian swine virus and were introduced to swine viruses in 1979.[16] The Figure depicts the origins of each gene segment of S-OIV A (H1N1).

NA and M are the targets of two classes of clinically used antivirals, oseltamivir (Tamiflu)/zanamivir (Relenza) and amantadine/rimantadine. Eurasian swine viruses are oseltamivir-sensitive and amantadine-resistant. The novel S-OIV A (H1N1) also has inherited sensitivity to oseltamivir and resistance to amantadine.[16]

Virulence factors of S-OIV A (H1N1)

The mortality rate for infection with S-OIV A (H1N1) appears not to be particularly high. However, virulence may change as the number of adaptive gene mutations increases, and the virus may have more opportunities to replicate in the new host species. Like other influenza A viruses, swine influenza virus enters host cells by binding to receptors that contain sialic acid. Swine are known to contain two types of receptors, 2,6-linked sialic acids that appear abundantly in the human respiratory tract, and 2,3-linked sialic acids that tend to be found in avian cells. The binding affinity of S-OIV A (H1N1) to different sialic acids is unclear. However, since the S-OIV A (H1N1) has been transmitted from human to human, this virus is expected to bind to human receptors. However, adaptive mutations may occur that promote the binding of S-OIV A (H1N1) to 2,6-linked sialic acids, if more humans become infected in the near future.

Adaptive mutations may occur in any other gene segments apart from the receptor binding

a stop codon at position 220. Hence, NS1 protein in S-OIV does not have the PDZ ligand domain. It is difficult to predict whether a further mutation in humans will change the sequence at position 220 and thereby alter the virulence of S-OIV A (H1N1).

How Did S-OIV A (H1N1) Overcome Host Restriction and Pass from Swine to Humans?

The crossing of host species by the novel S-OIV A (H1N1) is very important and interesting. Given the known virulence factors discussed above, the causes of human infection and its spread among humans remain unknown. Clearly, other previously unrecognized molecular determinants are responsible for the ability of S-OIV A (H1N1) to replicate and be transmitted in humans. The so-called species-specific signatures of avian and human influenza A viruses have been reported:[23] We examined the amino acid sequences of S-OIV A (H1N1) at those species-specific positions and found that most of the sequences were avian-like signatures. However, some of them had changed from avian- to human-like signatures. For example, at position 271 of the PB2 gene, the avian-like signature is T (threonine); whereas the human-like signature is A (alanine). Most swine viruses contain T at this position, whereas S-OIV in humans has A at position 271 of PB2. More studies should be conducted to identify the unrecognized molecular markers and thus help to determine the mechanism by which an animal influenza A virus crossed the species barrier to infect humans. Additionally, these molecular determinants will be used to predict viral virulence and pathogenicity for diagnosis.

Combating the Pandemic: Vaccines

As the S-OIV A (H1N1) infection has become a pandemic, the most critical question is how to contain it. From the experience so far, it is impossible to prevent the virus from spreading further because the first wave of the epidemic hit many developed countries and containment has been a failure. Although this virus remains sensitive to oseltamivir, the medication is for treatment and short-term prophylaxis rather than epidemic control. The only way to control this pandemic is through large-scale immunization. The production of vaccines against S-OIV A (H1N1) is feasible; however, several questions remain unanswered. First, how many doses are needed to induce effective protection? For seasonal influenza vaccine, one dose is sufficient for those aged > 8 years. For pandemic vaccines such as H5N1, two doses are needed.[24] Second, what is the optimal antigen content in the vaccine? Seasonal influenza vaccine contains 15 μg per strain and 45 μg in total. Without adjuvant, even at 90 μg, H5N1 vaccine is not sufficiently immunogenic. It is not known whether adjuvant is needed, or the optimal amount of antigen in the vaccine. Finally, there is a historic precedent of rushed production of influenza vaccine to contain swine influenza (as demonstrated in 1976). Unfortunately, an increase in the incidence of Guillain-Barré syndrome was demonstrated in the same year and vaccination had to be stopped.[25] The mechanism remains uncertain, although the antiganglioside antibody was raised as a possible explanation.[26] How to prevent this from being repeated in 2009 is an area of concern.

Conclusion

Forty-one years after the last influenza pandemic, we have witnessed the first pandemic caused by a novel S-OIV A (H1N1) in the 21st century. With our knowledge and experience about influenza viruses, we should be able to cope with this pandemic with the least possible morbidity and mortality. Vaccination is the only effective way to stop this pandemic and will be available in late 2009. More understanding of influenza viruses and continuous development of broad-spectrum influenza vaccines are of critical importance.