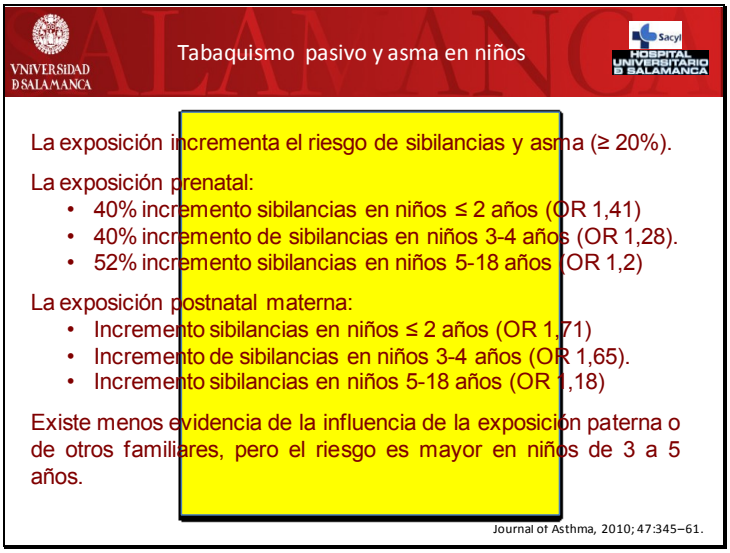
Diapositiva 21



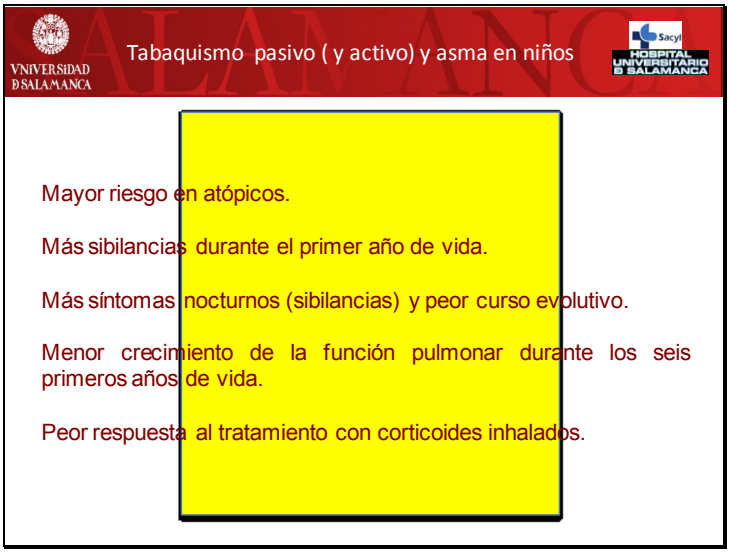
Se ha relacionado con síntomas de asma en niños asmáticos **(Morkjaroenpong V, Rand CS, Butz AM, et al. Environmental tobacco smoke exposure and nocturnal symptoms among inner-city children with asthma. J Allergy Clin Immunol 2002;110:147–53.)** Se ha relacionado con un curso evolutivo peor en niños, con más síntomas nocturnos, **(Martinez FD, Wright AL, Taussig LM, et al. Asthma and wheezing in the first six years of life. N Engl J Med 1995;332:133– 8)** Más sibilancias especialmente durante el primer año de vida **(Gilliland FD, Berhane K, Li YF, et al. Effects of early onset asthma and in utero exposure to maternal smoking on childhood lung function. Am J Respir Crit Care Med 2003;167:917–24.)** descenso de la función pulmonar durante los seis primaros años de vida.

(Halterman JS, Szilagyi PG, Yoos JL, et al. Benefits of a school-based asthma treatment program in the absence of secondhand smoke exposure. Arch Pediatr Adolesc med 2004;158:460 –7). Los niños no expuestos al humo del tabaco obtienen mejor respuesta al tratamiento con corticoides inhalados **Gilliland FD, Islan T, Berthane K, et al. Regular smoking and asthma incidence in adolescents. Am J Respir Crit Care Med 2006;174:1094 –100.** En niños no atópicos el riesgo es mayor que en atópicos.

Hannah Burke et al. PEDIATRICS Volume 129, Number 4, April 2012. Prenatal and passive smoking increases the incidence of wheeze and asthma in children and young people by at least 20%.

Exposure to prenatal maternal smoking was associated with 40% increase in risk of wheeze in children aged **≤ 2** years (OR = 1.41, 95% CI = 1.20–1.67, I2 = 82.5%, 14 studies). A similar magnitude of effect was observed for the relation between prenatal maternal smoking and incidence of wheeze between ages 3 and 4 (OR = 1.28, 95% CI = 1.14–1.44, I2 = 65.5%, 8 studies). Prenatal passive smoke exposure was associated with a 52% increased risk of wheeze in children aged 5 to 18 years (OR = 1.52, 95% CI = 1.23–1.87, I2 = 21.1%, 5 studies). Exposure to postnatal maternal smoking was associated with the strongest effects on the incidence of wheeze, effects on incidence of wheeze in children aged ≤ 2 years (OR = 1.70, 95% CI = 1.24–2.35, I2 = 0%, 4 studies), on incidence of wheeze in children aged 3 to 4 years (OR = 1.65, 95%CI = 1.20–2.68,I2 = 48.5%, 4 studies), and on the incidence of wheeze in children aged 5 to 18 years (OR = 1.18, 95% CI = 0.99–1.40, I2 = 1.4%, 3 studies).

Diapositiva 22



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The strongest significant effect was for prenatal maternal smoking and incidence of asthma in children aged ≤ 2 years (OR = 1.85, 95% CI = 1.35–2.53, I2 = 41.9%, 5 studies). The effect of prenatal maternal smoking became progressively weaker in relation to asthma incidence with increasing age but remained significantly associated with asthma onset between the ages of 5 and 18 years (OR = 1.23, 95% CI = 1.12– 1.36, I2 = 50%, 11 studies).

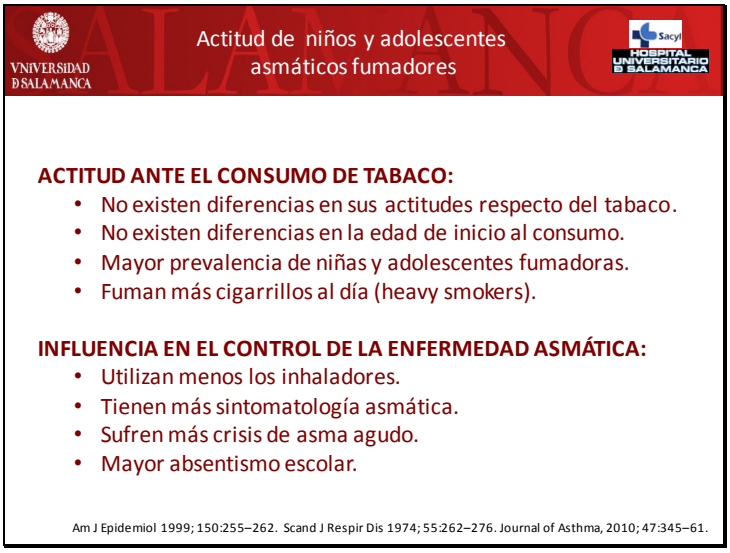
Exposure to postnatal maternal passive smoking was not significantly associated with incidence of asthma in children aged ≤ 2 years or 3 to 4 years but demonstrated a borderline significant association

with incidence of asthma in children aged 5 to 18 years (OR = 1.20, 95% CI = 0.98–1.46, P = .08, I2 = 65.3%, 8 studies)

There were more limited data on the effect of exposure to paternal smoking with no studies with data for children aged ≤ 2 years and only 1 study for children aged 3 to 4 years that revealed a significant effect (OR = 1.34, 95% CI = 1.23–1.46). Paternal exposure was not associated with incidence of asthma in children aged 5 to 18 years (OR = 0.98, 95% CI = 0.71–1.36, I2 = 0%, 4 studies).

Household passive smoke exposure was not significantly associated with incidence of asthma in children aged ≤ 2 years (OR = 1.14, 95% CI = 0.94– 1.38, I2 = 0.1%, 3 studies) but was associated with incidence of asthma in children aged 3 to 4 years (OR = 1.21, 95% CI = 1.00–1.47, I2 = 72.7%, 5 studies) and aged 5 to 18 years (OR = 1.30, 95% CI = 1.04–1.62, I2 = 37.7%, 5 studies).

Diapositiva 23

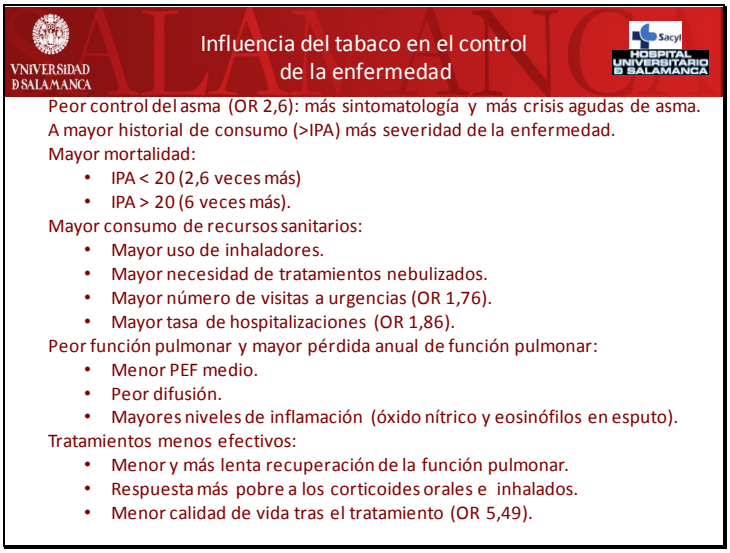


Kiviloog J, Irnell L, Eklund G. The prevalence of bronchial asthma and chronic bronchitis in smokers and non-smokers in a representative local Swedish population. Scand J Respir Dis 1974; 55:262–276. Examined rates of asthma and smoking among Swedish individuals between . the ages of 35 and 54Los resultados indicaron que la prevalencia de asma entre las mujeres fumadoras fue significativamente mayor que entre los no fumadores (4,5% y 1,7%, respectivamente). Sin embargo, no hubo diferencia en las tasas de asma entre los hombres fumadores y no fumadores (3,1% y 2,3%, respectivamente).

Chen Y, Dales R, Krewski D, Breithaupt K. Increased effects of smoking and obesity on asthma among female Canadians: the National Population Health Survey, 1994–1995. Am J Epidemiol 1999; 150:255–262.

Found similar results among participants in the National Population Health Survey conducted in Canada (13). Female smokers reported a significantly higher prevalence rate of asthma compared to nonsmokers (odds ratio [OR] = 1.7); this difference was particularly evident for females under the age of 25 (OR = 2.18) and among heavier as compared to lighter smokers or nonsmokers (OR = 2.0). There was no difference in asthma prevalence rates among male smokers and nonsmokers.

Diapositiva 29



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Piipari R, Jaakkola JJK, Jaakkola N, et al. Smoking and asthma in adults. Eur Respir J 2004;24:734 –9 El riesgo de desarrollar asma es superior en fumadores(OR 1,33) y exfumadores (OR 1.49) respecto a los no fumadores. Mayor susceptibilidad por las mujeres..

Gilliland FD, Islan T, Berthane K, et al. Regular smoking and asthma incidence in adolescents. Am J Respir Crit Care Med 2006;174:1094 –100.En niños no atópicos el riesgo es mayor que en atópicos.

Schatz M, Zeiger RS, Vollmer WM, et al. Determinants of future long-term asthma control. J Allergy Clin Immunol 2006;118:1048 –53. Peor control del asma a largo plazo (OR 2.46).

Strine TW, Balluz LS, Ford ES. The associations between smoking, physical inactivity, obesity, and asthma severity in the general US population. J Asthma 2007;44:651– 8. Más ataques de asma (OR 1,2) y más sintomatología nocturna (OR 2.0) .

Se ha relacionado con síntomas de asma en niños asmáticos

Martinez FD, Wright AL, Taussig LM, et al. Asthma and wheezing in the first six years of life. N Engl J Med 1995;332:133– 8 Se ha relacionado con un curso evolutivo peor en niños, con más síntomas y más sibilancias especialmente durante el primer año de vida.

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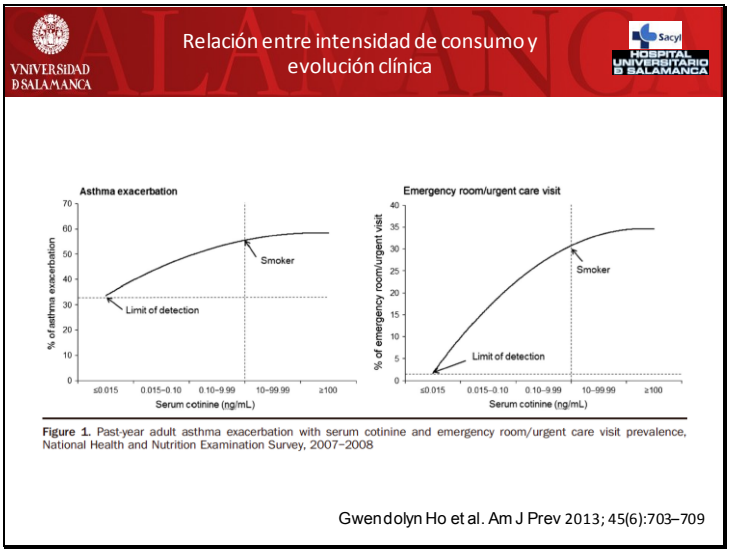
Halterman JS, Szilagyi PG, Yoos JL, et al. Benefits of a school-based asthma treatment program in the absence of secondhand smoke exposure. Arch Pediatr Adolesc med 2004;158:460 –7. Los niños no expuestos al humo del tabaco obtienen mejor respuesta al tratamiento con corticoides inhalados .

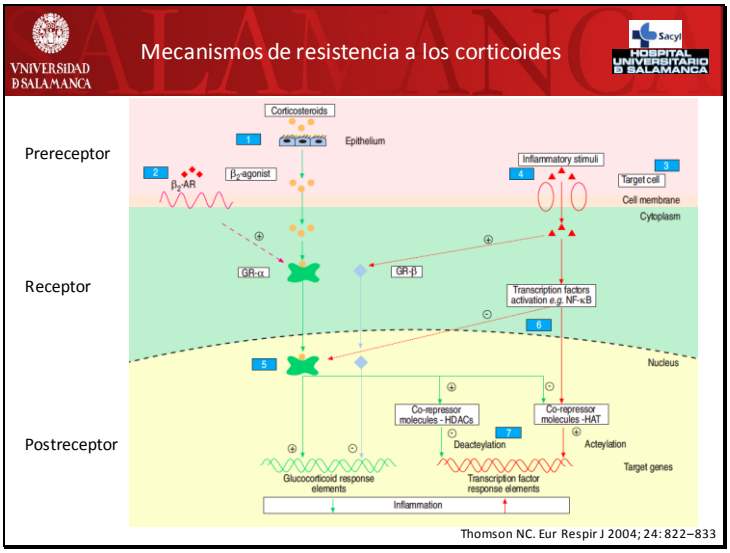
Diapositiva 25



(Gwendolyn Ho. Biomarkers of Tobacco Smoke Exposure and Asthma Severity in Adults. Am J Prev Med 2013;45(6):703–709)

Among adult asthmatics, 50.3% reported a past-year asthma attack (61.8% smokers, 46.6% nonsmokers, **p.** 0.029). Among these, 24.7% reported a past-year emergency/urgent visit for asthma (34.7% smokers, 20.1% nonsmokers, **p.** 0.034). Median concentrations of cotinine and creatinineadjusted NNAL (NNAL/Cr) were signifi cantly higher in those with a past-year asthma attack (0.43 ng/mL and 7.28 pg/mL) than in those without (0.06 ng/mL and 2.26 pg/mL), and highest in those with past-year emergency/urgent visits (0.93 ng/mL and 28.14 pg/mL).

Diapositiva 26



Los corticoides tienen efecto anti-inflamatorio e inmunosupresor potente. Actúan mediante la unión a receptores intracitoplasmáticos que al ser traslocados al núcleo y unirse a los promotores de los genes blanco, pueden aumentar o disminuir la expresión de estos genes. La resistencia a glucocorticoides es muy compleja y sus causas pueden estar a nivel de pre-receptor, receptor y post receptor. Interesante es que la expresión de receptores esteroidales tienen varios isotipos. Dos de ellos han sido más estudiados los receptores α que tienen acción antiinflamatoria inhibiendo la acción inflamatoria de varias citoquinas y el receptor β que tiene acción inhibitoria sobre el receptor α. Estos cambios de expresión pueden ser modificados por exposición a tabaco, fármacos e infecciones. Así se explica en

parte la desigual respuesta frente al tratamiento antiinflamatorio de diferentes enfermedades y pacientes.

The anti-inflammatory effects of corticosteroids are mediated by activation of cytoplasmic glucocorticoid receptors (GRs) that act as ligand-activated transcription factors, which translocate into the nucleus to suppress or induce glucocorticoid target genes. GR-a acts by directly binding to DNA sequences (transactivation) or by interacting with pro-inflammatory transcription factors (transrepression). GR-b, which does not bind ligand, is predominately located in the nucleus and cannot transactivate glucocorticoid-sensitive genes. Potential pathways and mechanisms of corticosteroid resistance in asthmatic smokers include the following: 1) corticosteroid pharmacokinetics, e.g. increased airway mucosal permeability, increased bronchial secretions; 2) corticosteroid and b2-adrenergic receptor (b2AR) interactions, e.g. down-regulation of b2AR function; 3) inflammatory cell phenotypes, e.g. increased airway neutrophil or CD8z lymphocyte numbers, reduced airway eosinophil numbers; 4) cytokine and mediators levels, e.g. increased production of interleukin (IL)-4, IL-8, tumour necrosis factor-a, decreased production of IL-10, increased nitrosative stress; 5) GRs, e.g. overexpression of GR-b, reduced expression of GR-a; 6) pro-inflammatory transcription factor activation, e.g. nuclear factor-**k**B (NF-**k**B), activator protein-1, signal transduction-activated factor; and 7) corticosteroid cell-signalling systems, e.g. reduced histone deacetylase activity (HDAC), increased p38 mitogen-activated protein kinase activity. HAT: histone acetyltransferase.

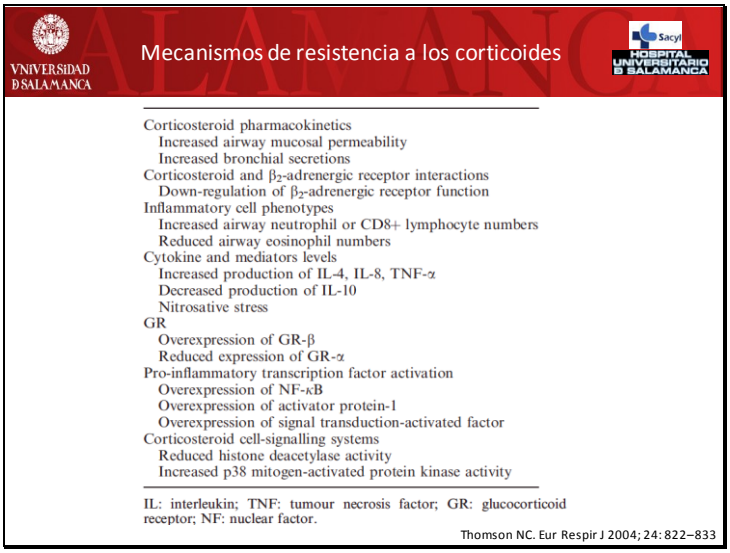
Existe un aumento de la permeabilidad de la mucosa en los fumadores con función pulmonar normal y en pacientes asmáticos no fumadores (Kennedy S, Elwood R, Wiggs B, et al. Increasedvairway mucosal permeability of smokers: relationship to airway reactivity. Am Rev Respir Dis 1984; 129:143–8 & Ilowite J, Bennett W, Sheetz M, et al. Permeability of the bronchial mucosa to 99mTc-DTPA in asthma. Am Rev Respir Dis 1988;139:1139–43.

Changes in levels of cytokines and inflammatory mediators may also play a role. Cigarette smoke has been shown to increase the production of interleukin 4, interleukin 8, and tumor necrosis factor- and decrease the production of interleukin 10 (Byron K, Varigos G, Wootton A. IL-4 production is increased in cigarette smokers. Clin Exp Immunol 1994;95:333– 6. & Keatings V, Collins P, Scott D, et al. Differences in interleukin-8 and tumour necrosis factor- in induced sputum from patients with chronic obstructive pulmonary disease disease and asthma. Am J Respir Crit Care Med 1996;153:530–4. & Churg A, Dai J, Changshi X, et al. Tumour necrosis factor- is central to acute cigarette smoke-induced inflammation and connective tissue breakdown. Am J Respir Crit Care Med 2002;166:849 –54. & Hawrylowicz C, Richards D, Loke T-K, et al. A defect in corticosteroid-induced IL-10 production in T lymphocytes from corticosteroid-resistant patients. J Allergy Clin Immunol 2002;109:369 –70.

Another potential mechanism for corticosteroid resistance is the overexpression of GR- receptors and underexpression of GR- receptors. GR- receptors are functional, whereas GR- receptors are not; therefore, an increase in the number of GR- receptors could lead to decreased binding and activity of glucocorticoids in vivo (Oakley RH, Jewell CM, Yudt MR, et al. The dominant negative activity of the human glucocorticoid receptor beta isoform. Specificity and mechanisms of action. J Biol Chem 1999;274:27857–66 & Pujols L, Mullol J, Perez M, et al. Expression of the human glucocorticoid receptor alpha and beta isoforms in human respiratory epithelial cells and their regulation by dexamethasone. Am J Respir Cell Mol Biol 2001;24:49 –57.

Altered corticosteroid cell-signaling systems may also play a role. (Megan Stapleton et al. Smoking and Asthma (JABFM May–June 2011 Vol. 24 No. 3)

Diapositiva 27



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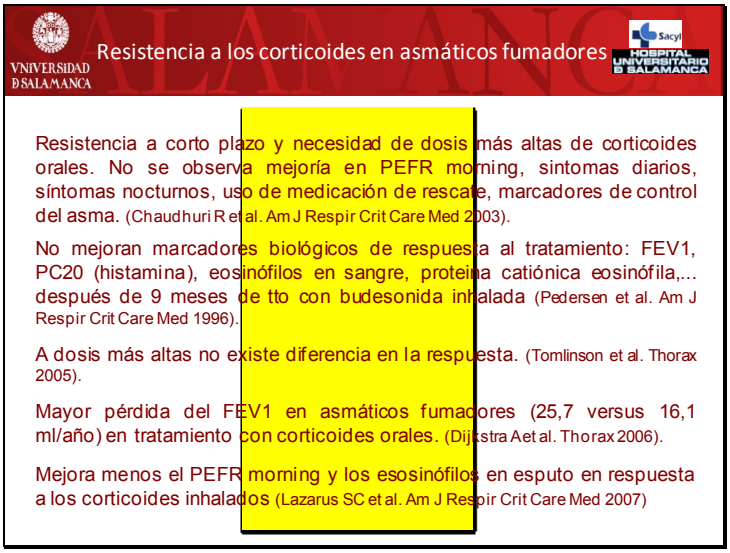
Diapositiva 28



The smokers had no improvement in FEV1 , histamine PC20, blood eosinophil counts, eosinophil cationic protein, or eosinophil protein x after 9 months of treatment with either dose of inhaled budesonide. This was the first study to report corticosteroid resistance in asthmatic smokers (Pedersen B, Dahl R, Karlstrom R, et al. Eosinophil and neutrophil activity in asthma in a one-year trial with inhaled budesonide. Am J Respir Crit Care Med 1996;153:1519 –29).

Resistencia a corto plazo y necesidad de dosis más altas . No se observa mejoría en PEFR morning, sintomas diarios, sintomas nocturnos, uso de medicación de resctate, marcadores de control del asma. (Chaudhuri R, Livingston E, McMahon AD, et al. Cigarette smoking impairs the therapeutic response to oral corticosteroids in chronic asthma. Am J Respir Crit Care Med 2003;168:1308–11.

Efecto dosis dependiente (a dosis más altas no existe diferencia en la respuesta entre fumadores y no fumadores (Tomlinson JEM, McMahon AD, Chaudhuri R, et al. Efficacy of low and high dose inhaled corticosteroid in smokers versus non-smokers with mild asthma. Thorax 2005;60:282–7)

Mayor perdida anual del FEV1 en asmáticos fumadores (25,7 ml/año) que en no fumadores (16,1 ml/año). Dijkstra A, Vonk JM, Jongepier H, et al. Lung function decline in asthma: association with inhaled corticosteroids, smoking, and sex. Thorax 2006;61:105–10.

Asmáticos no fumadores mejoran más el PEFR morning y disminuye el número de esosinófilos en esputo (Lazarus SC, Chinchilli VM, Rollings NJ, et al. Smoking affects response to inhaled corticosteroids or leukotriene receptor antagonists in asthma. Am J Respir Crit Care Med 2007;175:783–90.

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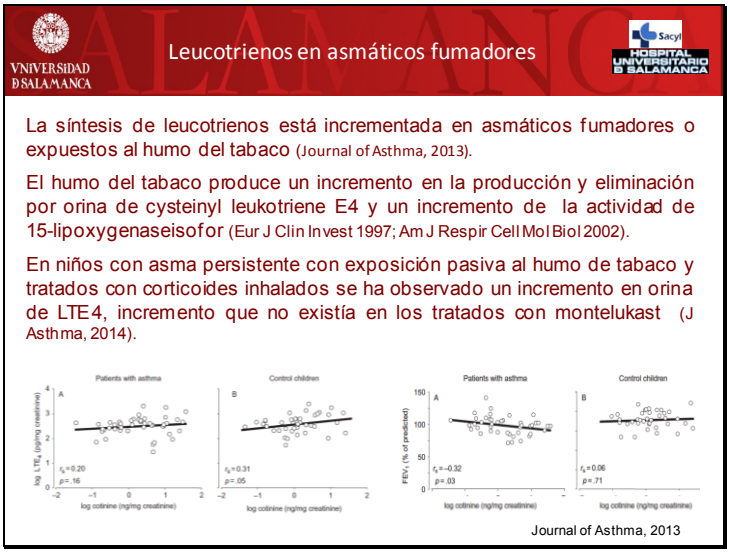
Chan

Diapositiva 29



Fauler J, Frolich J. Cigarette smoking stimulates cysteinyl leukotriene production in man. Eur J Clin Invest 1997; 27: 43–47. Cigarette smoking causes a dose-related increase in urinary cysteinyl leukotriene E4 production.

Zhu J, Kilty I, Granger H, et al. Gene expression and immunolocalization of 15-lipoxygenase isozymes in the airway mucosa of smokers with chronic bronchitis. Am J Respir Cell Mol Biol 2002; 27: 666–677.

15-lipoxygenaseisoform activity is increased in the airways of healthy smokers These findings might point to a role for leukotriene receptor antagonists in asthmatic smokers.

Ramneet Gill Low-level environmental tobacco smoke exposure and inflammatory biomarkers in children with asthma. J Asthma, 2014; 51(4): 355–359 A majority of school-age children with persistent asthma may be exposed to ETS, as measured by urinary cotinine, even if their parents insist they don't smoke in the home. Urinary LTE4 was higher in the ETS-exposed children treated with ICS, but not in children treated with montelukast.

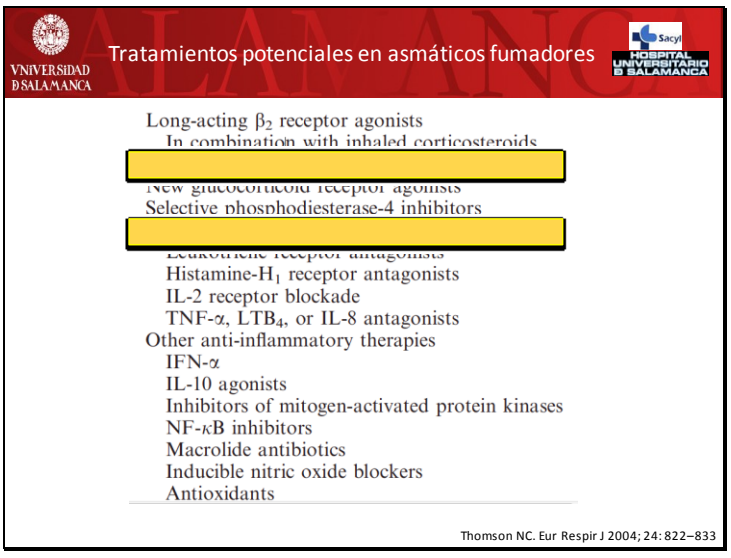
Diapositiva 30

Ito K, Caramori G, Cosio M, Chung K, Adcock IM, Barnes P. A molecular mechanism of action of theophylline: induction of histone deacetylase to decrease inflammatory gene expression. Proc Natl Acad Sci USA 2002; 99: 8921– 8926

Ito K, Lim S, Chung KF, Barnes PJ, Adcock I. Theophylline enhances histone deacetylase activity and restores glucocorticoid function during oxidative stress. Am J Respir Crit Care Med 2002; 165: 625.

La Teofilina a dosis bajas restaura la actividad de la Histona Deacetilasa (HADC), disminuyendo la expresión génica de la inflamación que es reclutado por los corticoides para disminuir la inflamación y restaura la actividad antiinflamatoia de los corticoides

Diapositiva 31



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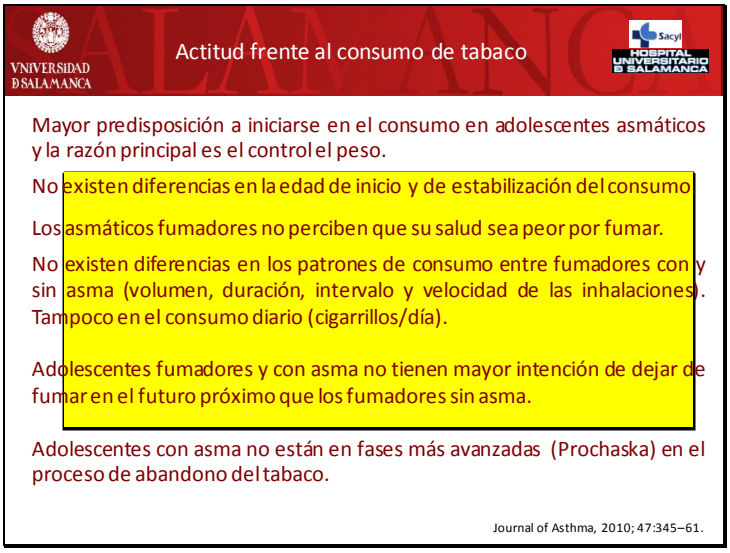
Diapositiva 32



Kiviloog J, Irnell L, Eklund G. The prevalence of bronchial asthma and chronic bronchitis in smokers and non-smokers in a representative local Swedish population. Scand J Respir Dis 1974; 55:262–276. Examined rates of asthma and smoking among Swedish individuals between . the ages of 35 and 54Los resultados indicaron que la prevalencia de asma entre las mujeres fumadoras fue significativamente mayor que entre los no fumadores (4,5% y 1,7%, respectivamente). Sin embargo, no hubo diferencia en las tasas de asma entre los hombres fumadores y no fumadores (3,1% y 2,3%, respectivamente).

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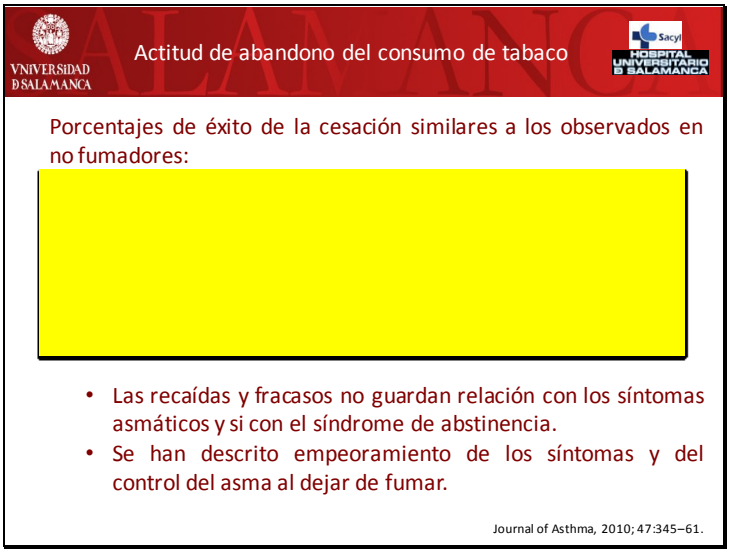
Diapositiva 33



Tonnesen P, Pisinger C, Hvidberg S, Wennike P, Bremann L, Westin A, Thomsen C, Nilsson F. Effects of smoking cessation and reduction in asthmatics. Nicotine Tob Res 2005; 7:139–148.
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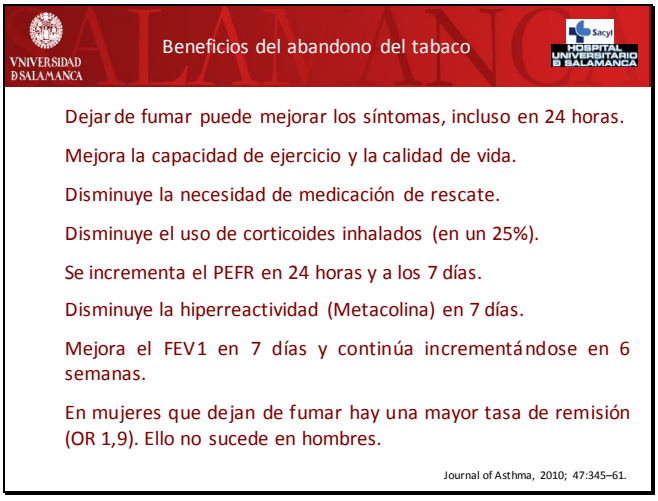
Results indicated that after 4 months, 12% had quit smoking and 15% had reduced their smoking to 7 or fewer cigarettes per day. Furthermore, at 4 months, biochemical parameters for smoking (exhaled CO, plasma cotinine, and thiocynate) were reduced in both the cessation and reduction groups, with the decreases in CO and thiocynate significantly greater for the cessation compared to the reduction group. Individuals in the cessation group also reported significant improvements in both overall and asthma-related quality of life. Those in the cessation group also showed significant decreases in rescue inhaler use, frequency of daytime asthma symptoms, bronchial hyperreactivity, and a 25% reduction in inhaled steroids.
Godtfredsen NS, Lange P, Prescott E, Osler M, Vestbo J. Changes in smoking habits and risk of asthma: a longitudinal population based study. Eur Respir J 2001; 18:549–554.
One study that found an increased risk of developing asthma after smoking cessation
Diapositiva 34



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Chaudhuri R, Livingston E,McMahaon AD, Lafferty J, Fraser I, SpearsM, McSharry CP, Thomson NC. Effects of smoking cessation on lung function and airway inflammation in smokers with asthma. Am J Respir Crit Care Med 2006; 174:127–133.
Chaudhuri and colleagues examined the effects of smoking cessation on lung function and airway inflammation among 32 smokers with asthma (94). Results indicated that
14 of the 21 individuals in the cessation group were able to remain abstinent for 1 week and 10 were able to remain abstinent for 6 weeks. Individuals in the cessation group showed significant improvements in spirometry (FEV1 and peak expiratory flow) after 1 week of cessation and improvements continued through 6 weeks of cessation. Moreover, asthma control improved and there was a decrease in sputum neutrophils after 6 weeks of cessation. Lastly, a study on the effect of quitting smoking on asthma remission found that females who had quit smoking during the follow-up period had an increased rate of asthma remission (hazard ratio [HR] = 1.9). The remission rate for males who quit smoking was not significant (95).
Diapositiva 35

Kiviloog J, Irnell L, Eklund G. The prevalence of bronchial asthma and chronic bronchitis in smokers and non-smokers in a representative local Swedish population. Scand J Respir Dis 1974; 55:262–276. Examined rates of asthma and smoking among Swedish individuals between . the ages of 35 and 54Los resultados indicaron que la prevalencia de asma entre las mujeres fumadoras fue significativamente mayor que entre los no fumadores (4,5% y 1,7%, respectivamente). Sin embargo, no hubo diferencia en las tasas de asma entre los hombres fumadores y no fumadores (3,1% y 2,3%, respectivamente).
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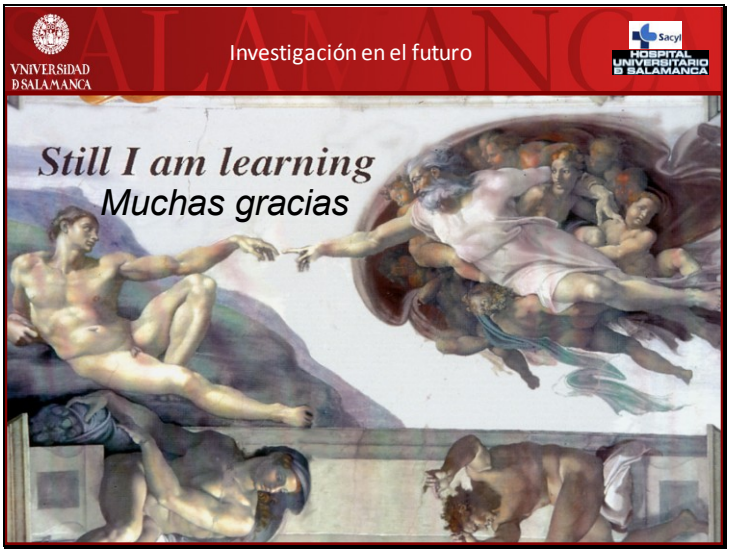

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