FEV1 decline and COPD and this agreed with Wang *et al*., (2009) who detected that the intrinsic Q-1 SNP may influence the splicing of the ss-variant and disturb the growth of *ADAM33*. Subsequent results on protease activity may cause a defect in tissue repair after inflammation-induced damage. This may lead to progressive damage of alveolar tissue and thereby enhance accelerated decrease in lung function [9]. In a Dutch population, the SNPs S1, S2, and Q-1 were associated with FEV1 decline, and the SNPs S1, S2, F+1, and T2 were associated with the presence of COPD while paper of Simpson *et al*., (2005) on European children showed that SNPs F+1, M+1, T1, and T2 significantly associated with lower FEV1 and COPD [13]. Current research in line with Xiao *et al*., (2005) who point out there is a relationship between *ADAM33*-V4 polymorphism and COPD in Tibetan population of china [17] but inconsistent with study of Pabst, *et al*., (2009) who show no genetic association between polymorphic variants in *ADAM33* and the onset or course of COPD [18,21]. Finally, only present study in Iraq are detected that *ADAM33*-V4 and Q-1 SNPs have role in development of COPD and other researches on genetics of COPD should be completed to determine the causative factors of COPD in this population.

CONCLUSION

COPD not causes by smoking or other environmental factors only but also strongly associated with genetic polymorphism. *ADAM33*/V4 and Q-1 SNP polymorphisms significantly correlated with COPD patients and this mean that individual who has *ADAM33*/V4 and Q-1 SNP polymorphisms more susceptible to COPD in this population.

REFERENCES

1. Rabe, K.F., Hurd, S., Anzueto, A., Barnes, P.J., Buist, S.A., Calverley, P., Fukuchi, Y., Jenkins, C., Rodriguez-Roisin, R., van Wee, C. and Zielinski, J., Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med.,2007, 176: 532–55.
2. Pauwels, R.A. and Rabe, K.F., Burden and clinical features of chronic obstructive pulmonary disease (COPD). Lancet, 2004, 364:613–20.
3. Buist, A. S., McBurnie, M.A., Vollmer, W.M., Gillespie, S., Burney, P., Mannino, D. M., Menezes, A. M., Sullivan, S.D., Lee, T.A., Weiss, K.B., Jensen, R.L., Marks, G.B, Gulsvik, A., Nizankowska- Mogilnicka, E. and BOLD Collaborative Research Group . International variation in the prevalence of COPD (the BOLD Study): a population based prevalence study. Lancet, 2007, 370: 741–50.
4. Sharma, N., Tripathi, P. and Awasthi, S., Role of ADAM*33* gene and associated single nucleotide polymorphisms in asthma. Allergy Rhinol., 2011, 2: e63-e70.
5. van Eerdewegh, P., Little, R.D, Dupuis, J. and Del Mastro, R.G., Association of the ADAM*33* gene with asthma and bronchial hyperresponsiveness. Nature, 2002, 418: 426-430.
6. Holgate, S.T., *ADAM metallopeptidase domain 33 (ADAM33):* identification and role in airways disease. Drug News Perspect., 2010, 23: 381-387.
7. Mocchegiani, E., Giacconi, R. and Costarelli, L., Metalloproteases/anti-metalloproteases imbalance in chronic obstructive pulmonary disease: genetic factors and treatment implications. Curr. Opin. Pulm. Med., 2011, 17 (Suppl 1): S11-S19.
8. van Diemen, C.C., Postma, D.S., Vonk, J.M. and Bruinenberg, M., A disintegrin and metalloprotease 33 polymorphisms and lung function decline in the general population. Am. J. Respir. Crit. Care Med.2005, 172: 329-333.
9. Wang, X., Li, L., Xiao, J., Jin, C., Huang, K., Kang, X., Wu, X. and Fuzhen, L., Association of ADAM*33* gene polymorphisms with COPD in a northe astern Chinese population. BMC Medical Genetics,2009, 10(132):1-7.
10. Lange, P., Nyboe, J., Jensen, G., Schnohr, P. and Appleyard, M., Ventilatory function impairment and risk of cardiovascular death and of fatal or non-fatal myocardial infarction. Eur. Respir. J.,1991, 4(9):1080-1087.
11. Lee, J.Y., Park, S.W., Chang, H.K., Kim, H.Y., Rhim, T., Lee, J.H., Jang, A.S., Koh, E.S. and Park, C.S., A disintegrin and metalloproteinase 33 protein in patients with asthma: Relevance to airflow limitation. American journal of respiratory and critical care medicine, 2006, 173(7):729-735.
12. Jongepier, H., Boezen, H.M., Dijkstra, A., Howard, T.D., Vonk, J.M., Koppelman G.H., Zheng, S.L., Meyers, D.A., Bleecker, E.R. and Postma, D.S., Polymorphisms of the ADAM*33* gene are associated with accelerated lung function decline in asthma. Clin Exp Allergy, 2004, 34(5):757-760.
13. Woodcock, A., Ollier, W.E., Collins, A., Custovic, A., Holloway, J.W. and John, S.L., Polymorphisms in a disintegrin and metalloprotease 33(*ADAM33*) predict impaired early-life lung function. American journal of respiratory and critical care medicine,2005, 172(1):55-60.
14. Foley, S., Mogas, A., Olivenstein, R. and Fiset, P., Increased expression of ADAM*33* and ADAM*8* with disease progression in asthma. J Allergy Clin Immunol., 2007, 119: 63-71.
15. Jackson, D., Sykes, A., Mallia, P. and Johnston, S., Asthma exacerbations: Origin, effect, and prevention. J allergy Clin Immunol., 2011, 128 (6): 1165-1174.
16. Postma, D. S., Kerkhof, M., Marike Boezen, H. and Koppelman, G. H., Asthma and Chronic Obstructive Pulmonary Disease Common Genes, Common Environments? American Journal of Respiratory and Critical Care Medicine, 2011,183:1-7.
17. Xiao, J., Han, J., Wang, X., Hua, D., Su, D., Bao, Y. and Lv, F., Association of *ADAM33* gene with susceptibility to COPD in Tibetan population of China. Mol. Biol. Rep., 2011, 38: 4941-4945.
18. Pabst, S., Touron, C. P., Gillissen, A., Lennarz, M., Tuleta, G., Skowasch, N. and Grohé, C., *ADAM33* gene polymorphisms in chronic obstructive pulmonary disease. Eur. J. Med Res., 2009, 14(Suppl 4): 182–186.
19. Bleecker, E.R., Similarities and differences in asthma and COPD. The Dutch hypothe sis. Chest, 2004, 126(2):93S-95S.
20. Lomas, D.A. and Silverman, E.K., The genetics of chronic obstructive pulmonary disease. Respir Res., 2001, 2: 20–6.
21. Mezher, M.M., Dakhil, A.S., Abdul Jawad, D.H. Role of Epstein-Bar virus(EBV) in Human Females with Breast Cancer. J Pharm Sci Res. 2017, 9: 1173-1177.