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Electronic nicotine delivery systems

To the Editor:

While reading the editorial on electronic nicotine delivery systems by Blasi and Ward [1], we noticed an inaccuracy in the section on The post-2016 regimen (p. 586). It is wrongly stated that "there will be a maximum nicotine volume for e-cigarettes of 10 mL for refillable cartridges". The volume of 10 mL was stated by the European Union in its 2014/40/EU Directive to be applicable to "refill containers" for the e-liquids that are used to refill the (maximally) 2 mL-containing refillable cartridges of an e-cigarette [2]. In the EU Directive 2014/40/EU, it is stated in article 20.3a that "nicotine-containing liquid is only placed on the market in dedicated refill containers not exceeding a volume of 10 ml, in disposable electronic cigarettes or in single use cartridges and that the cartridges or tanks do not exceed a volume of 2 ml" and in article 20.3b that "the nicotine-containing liquid does not contain nicotine in excess of 20 mg/ml."

@ERSpublications
A correction is needed on electronic nicotine delivery systems http://ow.ly/EWkzj

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1 Blasi F, Ward B. Electronic nicotine delivery systems (ENDS): the beginning of the end or the end of the beginning? *Eur Respir J* 2014; 44: 585–588.
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From the authors:

We thank K. Nackaerts and L. Joossens for correctly pointing out an error in our editorial "Electronic nicotine delivery systems (ENDS): the beginning of the end or the end of the beginning?" [1]. Where the Editorial states that "there will be a maximum nicotine volume for e-cigarettes (2 mL for single use and 10 mL for refillable cartridges), and a maximum nicotine concentration for refillable cartridges, tanks and containers of nicotine liquids (20 mg·mL^{-1})", it should read "according to Article 20 3 (a) and 20 3 (b) of Directive 2014/40/EU [2], 'Member States shall ensure that: (a) nicotine-containing liquid is only placed on the market in dedicated refill containers not exceeding a volume of 10 ml, in disposable electronic cigarettes or in single use cartridges and that the cartridges or tanks do not exceed a volume of 2 ml;

(b) the nicotine-containing liquid does not contain nicotine in excess of 20 mg/ml'." This error has also been addressed as an Author Correction in this issue of the *European Respiratory Journal*.

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A correction on electronic nicotine delivery systems http://ow.ly/EWkzj

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Conflict of interest: Brian Ward is an employee of the European Respiratory Society.

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Glucocorticoids induce the production of the chemoattractant CCL20 in airway epithelium

To the Editor:

We read with interest the report by Zijlstra *et al.* [1] in which the effects of glucocorticoids possibly contributed to airway neutrophilia in asthma. This study nicely adds to the research demonstrating that corticosteroids not only inhibit the production of several inflammatory chemokines and cytokines, but also corticosteroids increase levels of certain regulatory proteins; these neutrophil-active proteins are potentially involved in inflammation or, indeed, in host defence. Hence, the data reported by Zijlstra *et al.* [1] may deserve attention beyond the focus given by the authors.

Neutrophils are present in sputum of normal subjects. They are also increased in many respiratory conditions. Unsurprisingly, striking variability of sputum neutrophil counts has been demonstrated in corticosteroid-treated asthma [2] and was also recorded by Zijlstra *et al.* [1]. The notion that corticosteroids cause neutrophilia by inhibiting apoptosis of these cells is flourishing in the literature. It is so established that there is no longer any need to present supporting evidence [3]. Zijlstra *et al.* [1] do not discuss this aspect but their choice of reference regarding neutrophilic asthma is focussed on popular roles of apoptosis.

In vitro data have, for the last two decades, suggested the possibility that corticosteroids may reduce neutrophil apoptosis. Yet, there are no known data that compellingly support a role of this pharmacological treatment in patients; quite the opposite: in a careful biopsy study, Gizycki *et al.* [4] could not find any effect of corticosteroid treatment on neutrophil apoptosis compared with placebo treatment in chronic obstructive pulmonary disease. Furthermore, Uddin *et al.* [5] excluded a role of corticosteroid treatment as a factor in pro-survival activity for airway neutrophils in severe asthma. Lack of support for apoptosis-related effects actually lends weight to the findings of Zijlstra *et al.* [1], suggesting that a chemoattractant such as CCL20 could be involved in corticosteroid-induced airway neutrophilia.

On this note, it is of interest that severe asthma is associated with upregulation of CXCL5, possibly caused in part by corticosteroid treatment [6]. Furthermore, Fukakusa *et al.* [7] demonstrated that systemic