Review article

CED
Clinical and Experimental Dermatology

CPD

What's new in atopic eczema? An analysis of systematic reviews published in 2016. Part 1: treatment and prevention

L. Solman,[1] A. Lloyd-Lavery,[2] D. J. C. Grindlay,[3] N. K. Rogers,[3] K. S. Thomas[3] and K. E. Harman[3]

[1]*Department of Paediatric Dermatology, Great Ormond Street Hospital for Children, London, UK;* [2]*Churchill Hospital, Oxford University Hospitals NHS Foundation Trust, Oxford, UK; and* [3]*Centre of Evidence Based Dermatology, University of Nottingham, King's Meadow Campus, Lenton Lane, Nottingham, UK*

doi:10.1111/ced.13885

Summary

This review is part of a series of annual updates summarizing the evidence base for atopic eczema (AE). It provides a summary of key findings from 28 systematic reviews that were published or indexed during 2016 with a focus on treatment and prevention of AE. There is reasonable evidence of benefit for topical corticosteroids, calcineurin inhibitors, a glycyrrhetinic acid-containing preparation (Atopiclair®), oral ciclosporin, oral azathioprine, narrowband ultraviolet B radiation and education programmes. Overall, there is evidence that topical corticosteroids and calcineurin inhibitors have similar efficacy and that both can prevent AE flares when used twice weekly as maintenance therapy. However, topical calcineurin inhibitors are costlier and have more adverse reactions, thus topical corticosteroids should remain the standard of care for patients with AE. There is no evidence that multiple applications are better than once-daily application of topical corticosteroid. There is inconsistent evidence to support omalizumab and specific allergen immunotherapy use in AE. There is some evidence that vitamin D supplementation and synbiotics reduce AE severity, although the margin of improvement may not be clinically meaningful. There is little evidence to support the use of wet wraps or of complementary/alternative medicine (including Chinese herbal medicine). There is some evidence to suggest that a diet high in fish in infancy may be preventative for AE, but other dietary interventions for the prevention of AE show little promise. This review provides a succinct guide for clinicians and patients wishing to remain up to date with the latest evidence for the treatment and prevention of AE.

Correspondence: Dr Karen E. Harman, Centre of Evidence Based Dermatology, University of Nottingham King's Meadow Campus, Lenton Lane, Nottingham NG7 2NR, UK
E-mail: karen.harman@nottingham.ac.uk

Conflict of interest: The authors declare that they have no conflicts of interest.

This article presents independent research funded by the National Institute for Health Research Programme Grants for Applied Research (project number RP-PG-0216–20007). The views and opinions expressed in this article are those of the authors and do not necessarily reflect those of the NIHR, the National Health Service or the Department of Health.

Accepted for publication 18 September 2018

Background

Atopic eczema (AE) is a chronic, inflammatory, pruritic skin condition that frequently occurs in children.[1] The objective of this evidence update is to highlight the key findings of systematic reviews (SRs) on the treatment and prevention of AE. For SRs on epidemiology, aetiology and risk factors, see Part 2 of this update, and for nomenclature and outcome assessments, see Part 3. Similar evidence updates have been published previously, along with the search strategy details.[2–4]

Treatment of atopic eczema

Overview of all treatments

Nankervis *et al.*[5,6] reviewed 541 randomized controlled trials (RCTs) covering 92 AE interventions, and concluded that there is 'reasonable evidence of benefit' for topical corticosteroids, calcineurin inhibitors, a glycyrrhetinic acid-containing preparation (Atopiclair®; Valeant Pharmaceuticals Inc., Bridgewater, NJ, USA) that is likely to work by increasing cortisol levels in the skin, oral ciclosporin, oral azathioprine, narrowband ultraviolet B radiation, education programmes and use of topical corticosteroids or calcineurin inhibitors twice weekly for flare prevention. Interventions with reasonable evidence of 'no benefit' included dietary interventions (probiotics, evening primrose oil and borage oil), ion-exchange water softeners, twice-daily application of topical corticosteroids (compared with once-daily) and antibiotic-containing corticosteroids for noninfected AE. The evidence base for AE is still hampered by poor trial design. Research priorities with no current RCT evidence include dilution of topical corticosteroids, order of application of topical corticosteroids and emollients, allergy testing (followed by allergen avoidance) and modified bathing habits.

Systemic treatments

Omalizumab

We found three SRs that evaluated the effect of omalizumab in AE. These reviews were mixed in their assessment of omalizumab, as detailed below, probably reflecting differences in the range of studies included in their analyses. Only two RCTs have been performed, which were both small and showed that omalizumab was not significantly more effective than placebo,[7,8] while the observational studies have tended to demonstrate more positive effects, raising the possibility of publication bias.

Holm *et al.*[9] analysed 2 small RCTs, 5 case studies and 19 case series (174 participants) in their SR. Overall, 74% of patients experienced some kind of beneficial effect, ranging from 'little' to 'complete response'. This review concluded omalizumab to be a safe and well-tolerated, albeit expensive, treatment that could yield some clinical benefit for patients with severe recalcitrant AE.

A second SR and meta-analysis[10] evaluated 12 case studies and 1 RCT (103 participants). There was no conclusive evidence of the overall effectiveness of omalizumab (43% 'excellent' clinical response, 27% 'satisfying' and 30% no change or deterioration) but a preplanned multivariate logistic regression suggested that lower IgE concentrations (< 700 IU/mL) were significantly associated with an 'excellent' response compared with higher concentrations (700–5000 IU/mL) (OR = 12.3, 95% CI 2.46–62.5).

The third SR[11] assessed four small studies (2 RCTs, two nonrandomized open label studies, 69 participants), and concluded that results are conflicting; omalizumab may be beneficial for AE, but probably not in those with filaggrin (FLG) deficiency, based on the observation in one open-label trial[12] that none of the seven patients carrying an *FLG* mutation responded to omalizumab, whereas all eight responders were free of *FLG* mutations.

Immunotherapy/desensitization

A Cochrane review[13,14] evaluated specific allergen immunotherapy for the treatment of AE (12 RCTs, 733 participants). Some studies reported positive findings, but the quality of evidence in these studies was low due to the risk of bias and small sample size. The authors concluded that there was no consistent evidence of benefit for specific allergen immunotherapy.

Topical treatments

Topical corticosteroids and calcineurin inhibitors

We found three SRs that evaluated topical corticosteroids and calcineurin inhibitors in the treatment of AE.

One meta-analysis[15] compared topical corticosteroids with calcineurin inhibitors for AE treatment (12 RCTs, 6954 participants). The authors concluded that topical corticosteroids (all potencies included) and calcineurin inhibitors (tacrolimus and pimecrolimus pooled) have similar efficacy ('treatment success' 72% vs. 68%, relative risk [RR] = 1.15, 95% CI 1–1.31), but calcineurin inhibitors are associated with higher costs and have more adverse reactions (74% vs. 64%; RR = 1.28, 95% CI 1.05–1.58), such as skin burning (30% vs. 9%; RR = 3.27, 95% CI 2.48–4.31) and pruritus (12% vs. 8%; RR = 1.49, 95% CI 1.24–1.79). There were no differences in atrophy, skin infections or adverse events that were serious or required discontinuation of therapy. This study provides support for the continued use of topical corticosteroids as the therapy of choice for AE.

Siegfried *et al.*[16] assessed the long-term safety of topical calcineurin inhibitors and topical corticosteroids in the treatment of AE in children. All authors had received funding from at least one manufacturer of topical calcineurin inhibitor. Long-term use of topical calcineurin inhibitors and low- to mid-potency topical corticosteroids were concluded to be safe for paediatric patients with AE, with no evidence of cutaneous atrophy, systemic absorption or lymphoma. However, data were lacking on the safety of long-term use of higher-potency topical corticosteroids.

Wilkes *et al.*[17] mapped 74 RCTs that evaluated topical calcineurin inhibitors or corticosteroids. The study showed that topical calcineurin inhibitors were more commonly compared with placebo than with an active comparator. Of the 39 pimecrolimus and 41 tacrolimus trials, only 8 (20.5%) and 13 (31.7%), respectively had an active treatment arm (topical corticosteroids), vs. 25 (64.1%) and 15 (36.6%) with vehicle, respectively. The authors recommended the use of active comparators to determine the efficacy of new treatments in clinical practice. Placebo-controlled trials might be necessary for licensing and introduction of a new compound, but thereafter trials should be against appropriate potency active comparators (standard care) if they are to inform clinical practice.

Complementary and alternative treatments

Thandar *et al.*[18] assessed the evidence for topical herbal medicines. Of the eight RCTs included, only two small studies showed evidence of efficacy and were considered to have a low risk of bias: liquorice gel (90 participants) and *Hypericum perforatum* (St John's wort) cream (21 participants), both compared with placebo, prompting the conclusion that further high-quality studies are needed to assess these two treatments.

Another SR[19] evaluated the evidence for the effectiveness of complementary and alternative medicine for AE. It included 70 RCTs, the majority of which were underpowered. There were severe methodological limitations, making conclusions impossible. It was also limited to English-language publications so may have missed important contributions, particularly those in the Chinese literature.

In an overview review, Gu *et al.*[20] assessed three SRs: a Cochrane review without language restrictions and two RCTs on the use of Chinese herb medicine for AE. There was limited or inconclusive evidence for treatment of AE with Chinese herbal medicine.

Wet-wrap therapy

One meta-analysis[21] evaluated wet-wrap therapy for AE, consisting of topical steroid application under a layer of wet cotton bandages or garments (6 RCTs, 208 participants). A pooled estimate of the efficacy of wet-wrap therapy could not be calculated due to the high trial heterogeneity. A nonsignificant tendency toward an increased risk of mild skin infections was observed in those treated with wet-wrap therapy (pooled RR = 6.35, 95% CI 0.83–48.55). The overall quality of evidence for efficacy and safety outcomes was low, and the authors concluded that the evidence that wet-wrap therapy is more effective than standard treatment with topical corticosteroids is of low quality.

Dietary interventions and supplements for established atopic eczema

Probiotics. One meta-analysis[22] evaluated the evidence for the use of synbiotics (a mixture of prebiotics and probiotics) for treatment of established AE (6 RCTs, 369 children). Synbiotic intake was associated with reduced AE severity (change in SCORAD −6.56 points, 95% CI −11.43 to −1.68, $P = 0.008$), particularly in children aged ≥1 year (−7.37 points; 95% CI −14.66 to −0.07; $P = 0.048$) and where mixed bacterial strains were used (−7.32 points; 95% CI −13.98 to −0.66; $P = 0.03$). However, studies within the meta-analysis were heterogeneous, with wide confidence intervals. Furthermore, the improvement described may not be clinically meaningful: the minimal clinically important difference for SCORAD has been reported as approximately 8 points.

Vitamin D. One meta-analysis[23] of seven observational studies compared serum levels of vitamin D between 906 patients with AE and 657 controls. Serum vitamin D was lower in the patients with AE than in the controls [standardized mean difference (SMD) = −2.03 ng/mL, 95% CI −2.98 to −1.08], and this difference was significant in a subgroup analysis of children (690 children with AE vs. 657 controls; SMD = −3.03 ng/mL, 95% CI −4.76 to −1.29). In the same publication, they performed another meta-analysis (of four RCTs) and found significantly decreased SCORAD and EASI scores after vitamin D supplementation for both adults and children with AE (104 patients treated with vitamin D, 90 with placebo; SMD = −5.85, 95% CI −7.66 to −4.05).[23]

Another SR[24] also evaluated vitamin D supplementation in adults and children with AE (9 RCTs, 576

participants). A meta-analysis on four of these studies demonstrated that vitamin D supplementation resulted in a significant improvement in symptoms (mean difference 5.81, 95% CI −9.03 to −2.59, $P = 0.0004$). The authors concluded that vitamin D may be safe and beneficial in AE management, but larger studies of a longer duration are needed for confirmation.

Psychological and educational interventions. Saunder *et al.*[25] assessed the benefit of written action plans in the management of AE. The heterogeneity of the two included trials (1 RCT and 1 nonrandomized trial; 99 patients in total) precluded meta-analysis. In one trial, parents given written action plans had significantly better knowledge about AE than those given verbal instructions. The authors concluded that although the evidence is limited, the promise of benefit warrants large-scale RCTs assessing the effect of written action plans.

Pickett *et al.*[26] assessed educational interventions targeting health related quality of life (HRQoL) among people with chronic inflammatory skin conditions. In the three included RCTs assessing AE, no significant benefit was found by one trial, while the other studies showed a difference favouring educational interventions but did not reach statistical significance. In their treatment overview, Nankervis *et al.* analysed a larger number of studies and concluded that there is reasonable evidence of benefit for educational programmes.[5] This latter review focused more broadly on the effects of educational interventions.

Prevention of atopic eczema

Dietary interventions

Intake of fish during pregnancy or in infants. Zhang *et al.*[27] conducted a meta-analysis on the role of fish intake during pregnancy in the risk of AE in children (10 observational studies, 15 945 participants). There was no significant association between fish intake in pregnancy and childhood AE risk (RR = 0.88; 95% CI 0.75–1.04; $P = 0.13$), but studies were heterogeneous ($I^2 = 53\%$), making conclusions difficult.

In the same review, four studies (13 823 participants) explored the role of high fish intake in infancy on the development of AE. Three studies were able to be combined in meta-analysis, and the results suggested a protective effect of fish consumption in early life (RR = 0.71; 95% CI 0.61–0.82; $P < 0.00001$).

Omega-3 fatty acids. One SR[28] assessed omega-3 fatty acid supplementation on the risk of developing AE, including seven RCTs and seven observational studies (total number of participants unclear). Neither prenatal nor postnatal (maternal and infant) omega-3 fatty acid supplementation had a consistent effect on the risk for AE.

Polyunsaturated fatty acids. A Cochrane review[29] evaluated dietary supplementation with oils high in polyunsaturated fatty acids (PUFAs) on the risk of AE development. PUFA supplementation in infancy did not affect the incidence of AE in children up to 2 years of age (meta-analysis of 7 RCTs, 1906 infants; RR = 0.93, 95% CI 0.82–1.06) nor in children aged 2–5 years (meta-analysis of 2 RCTs, 154 infants; RR = 0.65, 95% CI 0.34–1.24). There was also no effect of PUFA supplementation on childhood prevalence of AE (635 infants; RR = 0.81, 95% CI 0.59–1.09).

Prebiotics and probiotics. Probiotics are live microorganisms that are believed to confer a health benefit, while prebiotics are indigestible food ingredients that promote the growth of beneficial microorganisms.

Chang *et al.*[22] conducted a meta-analysis on two RCTs (1320 children), which examined the use of synbiotics in infancy for AE prevention. The RCTs were moderately heterogeneous ($I^2 = 56.7\%$). Although one of the studies[30] showed synbiotic treatment significantly reduced AE incidence, the pooled risk showed no benefit (RR = 0.44, 95% CI 0.11–1.83).

The World Allergy Organisation–McMaster University Guidelines for Allergic Disease Prevention panel[31] suggested using prebiotic supplementation in infants who were not exclusively breastfed. This suggestion was conditional and based on very low certainty of the evidence, and they stated that they found no experimental or observational study of prebiotic supplementation in pregnant women or in breastfeeding mothers.

Hydrolysed formula. One meta-analysis[32] evaluated the effect of hydrolysed formula on AE risk (27 RCTs). The pooled data show no difference between hydrolysed formula and standard cow's milk formula for children aged 0–4 years with AE: OR = 0.84, 95% CI 0.67–1.07 for partially hydrolysed formula; OR = 0.55, 95% CI 0.28–1.09 for extensively hydrolysed casein-based formula; and OR = 1.12, 95% CI 0.88–1.42 for extensively hydrolysed whey-based formula.

Timing of allergenic food introduction. Ierodikanou *et al.*[33] assessed whether the timing of allergic food introduction during infancy influences risk of AE. Overall, there was no association shown by either

interventional (17 trials, 6798 participants) or observational studies (37 studies, 59 120 participants).

Conclusion

In this single year, there were several SRs published on the same topics, which is becoming increasingly common. The total number of SRs is also increasing year by year. This makes it very difficult for healthcare professionals to keep up with the latest literature and may overly inflate the importance of some interventions as a marketing tactic. We would like to encourage authors to register their protocols with the international prospective register of systematic reviews (PROSPERO, https://www.crd.york.ac.uk/PROSPERO/). Authors should also check the PROSPERO database before embarking on another review on the same topic. The Global Resource for EczemA Trials (GREAT) database is also a free comprehensive resource for identifying further SR and RCTs on AE treatments (http://www.greatdatabase.org.uk).

Acknowledgements

This work was carried out as part of the UK Dermatology Clinical Trials Network (UK DCTN) Fellowship scheme. The UK DCTN is grateful to the British Association of Dermatologists and the University of Nottingham for financial support of the Network. LS was a UK DCTN fellow for 2015.

Learning points

- There is reasonable evidence supporting the use of topical corticosteroids, calcineurin inhibitors and a glycyrrhetinic acid-containing preparation (Atopiclair®) that is likely to work by increasing cortisol levels in the skin, oral ciclosporin, azathioprine, narrowband ultraviolet B radiation and education programmes.
- Overall, topical corticosteroids and calcineurin inhibitors have similar efficacy, but topical calcineurin inhibitors are associated with higher costs and more AEs.
- Topical corticosteroids and calcineurin inhibitors used twice weekly can prevent eczema flares.
- There is inconsistent evidence of benefit for omalizumab, specific allergen immunotherapy and wet-wrap therapy in the treatment of AE.
- There is some evidence that synbiotics and vitamin D intake may improve the severity of AE.
- There is insufficient evidence that complementary/alternative medicine is beneficial in the treatment of AE.
- There is some evidence that a high fish diet in infancy (but not maternal fish intake) can be preventative for AE.
- There is insufficient evidence that the timing of allergenic food introduction or the intake of probiotics, hydrolysed formula milk, and omega 3 or polyunsaturated fatty acids in children or mothers reduces the risk of developing AE.

References

1 Hay RJ, Johns NE, Williams HC *et al.* The global burden of skin disease in 2010: an analysis of the prevalence and impact of skin conditions. *J Invest Dermatol* 2014; **134**: 1527–34.
2 University of Nottingham. Annual evidence updates for eczema – guidance. Available at: http://www.nottingham.ac.uk/research/groups/cebd/documents/methodological-resources/ebu-protocol.pdf (accessed 18 June 2018).
3 Lloyd-Lavery A, Rogers NK, Davies E *et al.* What's new in atopic eczema? An analysis of systematic reviews published in 2015. Part 2: Prevention and treatment. *Clin Exp Dermatol* 2018; **43**: 653–8.
4 Davies E, Rogers NK, Lloyd-Lavery A *et al.* What's new in atopic eczema? An analysis of systematic reviews published in 2015. Part 1: epidemiology and methodology. *Clin Exp Dermatol* 2018; **43**: 375–9.
5 Nankervis H, Thomas KS, Delamere FM *et al.* Scoping systematic review of treatments for eczema. *Program Grants Appl Res* 2016; **1**: 1–480. Available at: eprints.nottingham.ac.uk/40537/1/3009398-2.pdf (accessed 10 November 2018).
6 Nankervis H, Thomas KS, Delamere FM *et al.* What is the evidence base for atopic eczema treatments? A summary of published randomized controlled trials. *Br J Dermatol* 2017; **176**: 910–27.
7 Iyengar SR, Hoyte EG, Loza A *et al.* Immunologic effects of omalizumab in children with severe refractory atopic dermatitis: a randomized, placebo-controlled clinical trial. *Int Arch Allergy Immunol* 2013; **162**: 89–93.
8 Heil PM, Maurer D, Klein B *et al.* Omalizumab therapy in atopic dermatitis: depletion of IgE does not improve the clinical course — a randomized, placebo-controlled and double blind pilot study. *J Dtsch Dermatol Ges* 2010; **8**: 990–8.
9 Holm JG, Agner T, Sand C *et al.* Omalizumab for atopic dermatitis: case series and a systematic review of the literature. *Int J Dermatol* 2017; **56**: 18–26.
10 Wang HH, Li YC, Huang YC. Efficacy of omalizumab in patients with atopic dermatitis: a systematic review and

meta-analysis. *J Allergy Clin Immunol* 2016; **138**: 1719–22.e1.
11 Chia JC, Mydlarski PR. Dermatologic uses of omalizumab. *J Dermatolog Treat* 2017; **28**: 332–7.
12 Hotze M, Baurecht H, Rodriguez E *et al.* Increased efficacy of omalizumab in atopic dermatitis patients with wild-type filaggrin status and higher serum levels of phosphatidylcholines. *Allergy* 2014; **69**: 132–5.
13 Tam HH, Calderon MA, Manikam L *et al.* Specific allergen immunotherapy for the treatment of atopic eczema: a Cochrane systematic review. *Allergy* 2016; **71**: 1345–56.
14 Tam H, Calderon MA, Manikam L *et al.* Specific allergen immunotherapy for the treatment of atopic eczema. *Cochrane Database Syst Rev* 2016; **2**: CD008774.
15 Broeders JA, Ahmed Ali U, Fischer G. Systematic review and meta-analysis of randomized clinical trials (RCTs) comparing topical calcineurin inhibitors with topical corticosteroids for atopic dermatitis: A 15-year experience. *J Am Acad Dermatol* 2016; **75**: 410–19.e3.
16 Siegfried EC, Jaworski JC, Kaiser JD *et al.* Systematic review of published trials: long-term safety of topical corticosteroids and topical calcineurin inhibitors in pediatric patients with atopic dermatitis. *BMC Pediatr* 2016; **16**: 75.
17 Wilkes SR, Nankervis H, Tavernier E *et al.* How clinically relevant are treatment comparisons of topical calcineurin inhibitor trials for atopic eczema? *J Invest Dermatol* 2016; **136**: 1944–9.
18 Thandar Y, Gray A, Botha J *et al.* Topical herbal medicines for atopic eczema: a systematic review of randomized controlled trials. *Br J Dermatol* 2017; **176**: 330–43.
19 Vieira BL, Lim NR, Lohman ME *et al.* Complementary and alternative medicine for atopic dermatitis: an evidence-based review. *Am J Clin Dermatol* 2016; **17**: 557–81.
20 Gu SX, Zhang AL, Coyle ME *et al.* Chinese herbal medicine for atopic eczema: an overview of clinical evidence. *J Dermatolog Treat* 2017; **28**: 246–50.
21 Gonzalez-Lopez G, Ceballos-Rodriguez RM, Gonzalez-Lopez JJ *et al.* Efficacy and safety of wet wrap therapy for patients with atopic dermatitis: a systematic review and meta-analysis. *Br J Dermatol* 2017; **177**: 688–95.
22 Chang YS, Trivedi MK, Jha A *et al.* Synbiotics for prevention and treatment of atopic dermatitis: a meta-analysis of randomized clinical trials. *JAMA Pediatr* 2016; **170**: 236–42.
23 Kim MJ, Kim SN, Lee YW *et al.* Vitamin D status and efficacy of vitamin D supplementation in atopic dermatitis: a systematic review and meta-analysis. *Nutrients* 2016; **8**: 1–2.
24 Kim G, Bae JH. Vitamin D and atopic dermatitis: A systematic review and meta-analysis. *Nutrition* 2016; **32**: 913–20.
25 Sauder MB, McEvoy A, Sampson M *et al.* The effectiveness of written action plans in atopic dermatitis. *Pediatr Dermatol* 2016; **33**: e151–3.
26 Pickett K, Frampton G, Loveman E. Education to improve quality of life of people with chronic inflammatory skin conditions: a systematic review of the evidence. *Br J Dermatol* 2016; **174**: 1228–41.
27 Zhang GQ, Liu B, Li J, *et al.* Fish intake during pregnancy or infancy and allergic outcomes in children: A systematic review and meta-analysis. *Pediatr Allergy Immunol* 2017; **28**: 152–61.
28 Newberry SJ, Chung M, Booth M *et al. Omega-3 Fatty Acids and Maternal and Child Health: An Updated Systematic Review*. Rockville, MD: Agency for Healthcare Research and Quality (US), 2016 (Evidence Reports/ Technology Assessments No. 224). Available at: https://www.ncbi.nlm.nih.gov/books/NBK395921/ (accessed 1 December 2017).
29 Schindler T, Sinn JK, Osborn DA. Polyunsaturated fatty acid supplementation in infancy for the prevention of allergy. *Cochrane Database Syst Rev* 2016; **10**: CD010112.
30 Kukkonen K, Savilahti E, Haahtela T *et al.* Probiotics and prebiotic galacto-oligosaccharides in the prevention of allergic diseases: a randomized, double-blind, placebo-controlled trial. *J Allergy Clin Immunol* 2007; **119**: 192–8.
31 Cuello-Garcia CA, Fiocchi A, Pawankar R *et al.* World Allergy Organization-McMaster University guidelines for allergic disease prevention (GLAD-P): prebiotics. *World Allergy Organ J* 2016; **9**: 10.
32 Boyle RJ, Ierodiakonou D, Khan T *et al.* Hydrolysed formula and risk of allergic or autoimmune disease: systematic review and meta-analysis. *BMJ* 2016; **352**: i974.
33 Ierodiakonou D, Garcia-Larsen V, Logan A *et al.* Timing of allergenic food introduction to the infant diet and risk of allergic or autoimmune disease: a systematic review and meta-analysis. *JAMA* 2016; **316**: 1181–92.

CPD questions

Learning objective

To demonstrate up-to-date knowledge of prevention and treatment in atopic eczema.

Question 1

Which of the following statements about the topical treatment of atopic eczema (AE) is true?

(a) Topical corticosteroids or calcineurin inhibitors used twice weekly reduce the risk of AE flares.

(b) Ion exchange water softeners reduce the severity of AE.
(c) Topical calcineurin inhibitors are more effective than topical corticosteroids for AE.
(d) Topical corticosteroids are associated with higher rates of adverse reactions than topical calcineurin inhibitors.
(e) Use of a topical glycyrrhetinic acid containing emollient (Atopiclair®) is ineffective for the treatment of AE.

Question 2

What advice can you give to parents regarding the prevention of atopic eczema (AE) in their children?
(a) Prenatal (maternal) supplementation of omega-3 fatty acids decreases the risk of AE.
(b) Postnatal (infant) supplementation of omega-3 fatty acids decreases the risk of AE.
(c) Fish intake during pregnancy decreases the risk of AE.
(d) Use of extensively hydrolysed formula has no influence on the development of AE.
(e) Timing of allergic food introduction during infancy influences the risk of AE.

Question 3

Which of the following statements about the systemic treatment of atopic eczema (AE) is true?
(a) Omalizumab is effective in the treatment of patients with AE who have filaggrin deficiency.
(b) Specific immunotherapy is beneficial in patients with AE.
(c) Omalizumab is a well-tolerated, safe and inexpensive treatment.
(d) Omalizumab can be beneficial in patients with severe, recalcitrant eczema without filaggrin deficiency, although the data are conflicting.
(e) Ciclosporin is not beneficial in the treatment patient with AE.

Question 4

Which of the following complementary treatments for atopic eczema (AE) has an effect on the severity of AE?
(a) Chinese herbal medications.
(b) Liquorice gel.
(c) Borage oil.
(d) Evening primrose oil.
(e) Chamomile cream.

Question 5

For which of the following treatments of atopic eczema (AE) has ***no*** benefit been demonstrated in clinical trials?
(a) Topical corticosteroids.
(b) Oral ciclosporin.
(c) Oral azathioprine.
(d) Narrowband ultraviolet B (NB-UVB) radiation.
(e) Chinese herbal medications.

Instructions for answering questions

This learning activity is freely available online at http://www.wileyhealthlearning.com/ced

Users are encouraged to

- Read the article in print or online, paying particular attention to the learning points and any author conflict of interest disclosures
- Reflect on the article
- Register or login online at http://www.wileyhealthlearning.com/ced and answer the CPD questions
- Complete the required evaluation component of the activity

Once the test is passed, you will receive a certificate and the learning activity can be added to your RCP CPD diary as a self-certified entry.

This activity will be available for CPD credit for 2 years following its publication date. At that time, it will be reviewed and potentially updated and extended for an additional period.