

**NATIONAL INSTITUTE FOR HEALTH AND CARE
EXCELLENCE**

Appraisal consultation document

**Sodium zirconium cyclosilicate for treating
hyperkalaemia**

The Department of Health and Social Care has asked the National Institute for Health and Care Excellence (NICE) to produce guidance on using sodium zirconium cyclosilicate in the NHS in England. The appraisal committee has considered the evidence submitted by the company and the views of non-company consultees and commentators, clinical experts and patient experts.

This document has been prepared for consultation with the consultees. It summarises the evidence and views that have been considered, and sets out the recommendations made by the committee. NICE invites comments from the consultees and commentators for this appraisal and the public. This document should be read along with the evidence (see the [committee papers](#)).

The appraisal committee is interested in receiving comments on the following:

- Has all of the relevant evidence been taken into account?
- Are the summaries of clinical and cost effectiveness reasonable interpretations of the evidence?
- Are the recommendations sound and a suitable basis for guidance to the NHS?
- Are there any aspects of the recommendations that need particular consideration to ensure we avoid unlawful discrimination against any group of people on the grounds of race, gender, disability, religion or belief, sexual orientation, age, gender reassignment, pregnancy and maternity?

Note that this document is not NICE's final guidance on this technology. The recommendations in section 1 may change after consultation.

After consultation:

- The appraisal committee will meet again to consider the evidence, this appraisal consultation document and comments from the consultees.
- At that meeting, the committee will also consider comments made by people who are not consultees.
- After considering these comments, the committee will prepare the final appraisal document.
- Subject to any appeal by consultees, the final appraisal document may be used as the basis for NICE's guidance on using sodium zirconium cyclosilicate in the NHS in England.

For further details, see NICE's [guide to the processes of technology appraisal](#).

The key dates for this appraisal are:

Closing date for comments: 19th November 2018

Second appraisal committee meeting: 4th December 2018

Details of membership of the appraisal committee are given in section 5.

1 Recommendations

- 1.1 Sodium zirconium cyclosilicate is not recommended, within its marketing authorisation, for treating hyperkalaemia in adults.
- 1.2 This recommendation is not intended to affect treatment with sodium zirconium cyclosilicate that was started in the NHS before this guidance was published. People having treatment outside this recommendation may continue without change to the funding arrangements in place for them before this guidance was published, until they and their NHS clinician consider it appropriate to stop.

Why the committee made these recommendations

Sodium zirconium cyclosilicate is a treatment option for people with high blood serum potassium levels (hyperkalaemia). The company proposes that it would benefit people with heart failure or stages 3 to 5 chronic kidney disease who have high levels of serum potassium.

The evidence on how well sodium zirconium cyclosilicate works is not considered relevant to NHS clinical practice because it comes mostly from people with a level of serum potassium that would not be treated in the NHS. There is also no evidence to show that sodium zirconium cyclosilicate extends life or improves quality of life compared with standard care in people who would have treatment for hyperkalaemia in the NHS. In addition, the effectiveness estimates of sodium zirconium cyclosilicate for preventing heart attacks and strokes, and increasing survival, do not provide good evidence on what effect the drug or lowering serum potassium levels has on these outcomes.

Because of the lack of relevant clinical-effectiveness evidence, the cost-effectiveness estimates for sodium zirconium cyclosilicate are not valid. Therefore, the drug is not recommended for treating hyperkalaemia in adults.

2 Information about sodium zirconium cyclosilicate

Marketing authorisation indication	Sodium zirconium cyclosilicate (Lokelma, AstraZeneca) has a marketing authorisation 'for the treatment of hyperkalaemia in adult patients'.
Dosage in the marketing authorisation	<p>Correction phase:</p> <p>The recommended starting dose of sodium zirconium cyclosilicate is 10 g, administered 3 times a day orally as a suspension in water. When normal potassium levels are met, the maintenance regimen should be followed. If normal potassium levels are not met within 72 hours of treatment, sodium zirconium cyclosilicate should be stopped</p> <p>Maintenance phase:</p> <p>For people with normal potassium levels after the correction phase, the minimal effective dose of sodium zirconium cyclosilicate to prevent recurrence of hyperkalaemia should be established. A starting dose of 5 g once daily is recommended, with possible titration up to a maximum 10 g once daily or down to 5 g once every other day as needed to maintain a normal potassium level.</p>
Price	The price of sodium zirconium cyclosilicate has been agreed with the Department of Health, but is currently confidential and cannot be reported here.

3 Committee discussion

The appraisal committee (section 5) considered evidence submitted by AstraZeneca and a review of this submission by the evidence review group (ERG). See the [committee papers](#) for full details of the evidence.

Treatment of hyperkalaemia

Patients in the NHS with serum potassium levels above the normal range do not always have treatment to lower potassium

- 3.1 Hyperkalaemia is a high level of potassium in the blood serum, above 5.0 mmol/litre, and is classified by the European Resuscitation Council as mild (serum potassium level of 5.5 mmol/litre to 5.9 mmol/litre), moderate (6.0 mmol/litre to 6.4 mmol/litre) or severe (6.5 mmol/litre and above). Hyperkalaemia occurs most commonly in people with chronic kidney disease (stages 4 and 5), heart failure, liver disease and adrenal

insufficiency. It can also occur as a result of certain treatments for high blood pressure, chronic kidney disease, proteinuria (protein in the urine) and heart failure; specifically, potassium-sparing diuretics or renin-angiotensin-aldosterone system (RAAS) inhibitors. These inhibitors include angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs) and aldosterone receptor antagonists. Serum potassium levels in people with chronic kidney disease and in people having RAAS inhibitors are routinely monitored. Normal serum potassium levels are between 3.5 mmol/litre and 5.0 mmol/litre. However, the clinical and patient experts explained that people do not automatically have treatment to lower serum potassium if it is more than 5.0 mmol/litre. The committee and the clinical expert agreed they would not usually consider treating hyperkalaemia at serum potassium levels lower than 6.0 mmol/litre. This is because levels can be high for several reasons and may resolve fairly quickly without treatment or by treating the cause (rather than the hyperkalaemia itself). The committee understood that the decision to use potassium-lowering treatment would take into account the speed of onset of hyperkalaemia and changes on electrocardiogram, as well as serum potassium levels, because these show a patient's prognosis. It concluded that patients in the NHS with serum potassium levels above the normal range do not always have treatment to lower potassium.

Hyperkalaemia is treated as an emergency in hospital or, when it is non-life-threatening, in an outpatient setting: the treatments for these differ

3.2 The need for, and type of, treatment for hyperkalaemia depends on its severity:

- Life-threatening hyperkalaemia treated as an emergency in hospital: in this situation, there is often a rapid onset of very high levels of serum potassium and a risk of a cardiac arrest. It is treated with active potassium-lowering treatments, and by identifying and removing the cause. Treatments include:

- calcium gluconate intravenously to protect the heart from complications of hyperkalaemia
- insulin and glucose intravenously, which moves potassium from the blood into cells
- follow-up potassium-binding agents for 3 or more days (namely, calcium resonium given orally) to then remove potassium from the body
- stopping or reducing RAAS inhibitors (because these can increase potassium levels).

The clinical expert explained that people with normal serum potassium levels after hyperkalaemia has initially been corrected do not have maintenance treatment with a potassium-lowering drug in current clinical practice. This may be because potassium-binding treatments such as calcium resonium are poorly tolerated.

- Persistently raised, but non-life-threatening hyperkalaemia treated in an outpatient setting: potassium levels may be as high as with life-threatening hyperkalaemia, but that it is less of a risk than when potassium levels have risen quickly. Management aims to lower potassium levels to prevent hyperkalaemia needing hospital treatment.

Treatment includes:

- advice to people with chronic kidney disease to avoid foods high in potassium, as part of a wider restrictive diet
- stopping or reducing RAAS inhibitors.

The committee concluded that the management of emergency life-threatening hyperkalaemia differs from that of persistently raised but non-life-threatening hyperkalaemia, which justified the separate analyses for sodium zirconium cyclosilicate in these populations.

People would welcome an alternative to stopping RAAS inhibitors

3.3 The company proposed that people having sodium zirconium cyclosilicate will be less likely to stop RAAS inhibitors, so will live longer with fewer cardiovascular and renal complications. NICE's clinical guideline on

[chronic kidney disease in adults: assessment and management](#) states that RAAS inhibitors should not be routinely started in people with serum potassium levels of 5.0 mmol/litre or more, and should be stopped in people with levels of 6.0 mmol/litre or more. The committee noted that some people stop RAAS inhibitors for reasons other than hyperkalaemia. The committee and the clinical expert agreed that RAAS inhibitors would be used in the NHS for some people with serum potassium levels over 5.0 mmol/litre and would be stopped when serum potassium levels are 6.0 mmol/litre or more. At lower levels, the RAAS inhibitor dose would more likely be reduced, rather than stopped. This is because the perceived benefits of being on treatment outweigh the risks of having a serum potassium level of between 5.0 mmol/litre and 6.0 mmol/litre. The patient and clinical experts explained that some people with high blood pressure may switch to another type of blood pressure lowering treatment if they stop RAAS inhibitors. These may not be as effective for some people at delaying kidney disease. The committee concluded that patients and clinicians are keen for new treatments that would allow them to continue RAAS inhibitors, but that the harms and benefits of stopping a RAAS inhibitor and switching to an alternative blood pressure lowering treatment would need to be taken into account.

The long-term benefit of continuing RAAS inhibitors on quality of life and survival in people with hyperkalaemia may vary from person to person

- 3.4 The clinical expert explained that the benefit of being on RAAS inhibitor treatment depended on: the underlying population (for example, people with different stages of chronic kidney disease); the class of RAAS inhibitor (ACE inhibitors, ARBs, aldosterone receptor antagonists); and outcome (for example, cardiovascular disease, worsening of renal disease, death). Specifically, the clinical expert noted that the benefit of RAAS inhibitors in protecting the kidney had not been documented in people with chronic kidney disease stages 4 and 5. The committee also acknowledged that, in some people, the RAAS inhibitor dose may be reduced rather than stopped completely (see section 3.3), and that some

people may be on multiple RAAS inhibitors, only 1 of which is stopped because of hyperkalaemia. The committee concluded that factors affecting the harms and benefits of stopping RAAS inhibitors because of hyperkalaemia compared with using another antihypertensive (for people with high blood pressure) or with standard care (for people who would not normally be offered another blood pressure lowering drug) were affected by the:

- underlying condition
- type of RAAS inhibitor
- dose of RAAS inhibitor
- number of RAAS inhibitors
- reason for stopping RAAS inhibitor.

It further concluded that the long-term benefit of continuing RAAS inhibitors on quality of life and survival in people with hyperkalaemia may vary from person to person, and that the balance of benefits and harms should be taken into account in its decision-making (see section 3.11).

People with hyperkalaemia would welcome a treatment that allows a less strict low-potassium diet

3.5 The patient experts noted that a low-potassium diet is difficult to stick to because many foods contain potassium. The clinical expert explained that such a diet is considered worth trying to help manage serum potassium levels, is recommended by NICE. The diet lowers serum potassium compared with an unrestricted diet. They added that a new treatment option would not replace dietary advice but would most likely to be used alongside it, and may mean that the diet does not need to be so strict. The committee concluded that, although people may prefer a potassium-lowering drug to a strict diet, sodium zirconium cyclosilicate was unlikely to replace a low-potassium diet completely.

Company positioning of sodium zirconium cyclosilicate

The company proposes using sodium zirconium cyclosilicate in a population narrower than that in the marketing authorisation

3.6 The company focused its submission on using sodium zirconium cyclosilicate to treat hyperkalaemia in people with chronic kidney disease (stages 3 to 5, excluding those on dialysis) and in people with heart failure. The patient expert noted that there is a particular need for potassium-lowering treatment for people having dialysis. This is because the severity and frequency of symptoms of hyperkalaemia tend to increase between dialysis sessions. The committee agreed that there was a need for treatment options for people on dialysis but noted that the company had not provided evidence for this population. It noted that the population in the company's submission is narrower than the marketing authorisation for sodium zirconium cyclosilicate because people with other conditions also develop hyperkalaemia (see section 3.1). The committee recognised that chronic kidney disease and heart failure, and treatments used to manage these conditions, can be causes of hyperkalaemia. It also recognised that most people with heart failure will also have kidney disease (stage 3 or worse). The committee concluded that it would appraise sodium zirconium cyclosilicate within the population the company had proposed, which was narrower than that in the marketing authorisation.

The company proposes that sodium zirconium cyclosilicate will be used in either an emergency or outpatient setting

3.7 The marketing authorisation for sodium zirconium cyclosilicate covers its use as a corrective treatment for lowering serum potassium levels followed by its use as a maintenance treatment (at a lower dose) for people whose levels return to normal after the corrective treatment. The aim of the maintenance dose is to avoid repeat hyperkalaemia. The committee noted that the marketing authorisation does not specify whether sodium zirconium cyclosilicate should be used to treat life-

threatening hyperkalaemia considered to be an emergency, or persistent hyperkalaemia treated in the outpatient setting. It also noted that it does not specify the concentration of serum potassium at which treatment should be started. The company proposed that sodium zirconium cyclosilicate would be used:

- In an emergency setting as an alternative to calcium resonium and stopping RAAS inhibitors in people with very high levels of serum potassium and needing immediate hospital treatment to reduce levels. The committee heard from the company that sodium zirconium cyclosilicate would complement rather than replace the use of insulin and glucose in patients with life-threatening hyperkalaemia.
- In an outpatient setting as an alternative to stopping RAAS inhibitors and a strict low-potassium diet to manage non-life-threatening hyperkalaemia and prevent it developing into life-threatening hyperkalaemia in people with hyperkalaemia identified through routine monitoring. The committee noted that the clinical and patient experts did not expect sodium zirconium cyclosilicate to completely replace the need for a low-potassium diet (see section 3.5).

The company stated that its clinical advisers suggested that sodium zirconium cyclosilicate would be started, and RAAS inhibitors stopped or reduced, in people with serum potassium levels of 5.5 mmol/litre or more presenting with persistently high serum potassium levels through routine monitoring in an outpatient setting and in people admitted to hospital with serum potassium levels of 6.0 mmol/litre or more. It concluded that, in its deliberations, it should consider sodium zirconium cyclosilicate in both emergency and outpatient treatment settings. It further concluded that the comparators were calcium resonium and management of RAAS inhibitors in the emergency setting, and management of RAAS inhibitors in the outpatient treatment setting.

Clinical effectiveness

Trial evidence does not show whether sodium zirconium cyclosilicate is more clinically effective than current standard care in the NHS

3.8 The clinical-effectiveness evidence for sodium zirconium cyclosilicate came from the ZS004 and ZS005 trials. The committee noted that these trials did not include people with life-threatening hyperkalaemia that would be treated as an emergency, and that the trial population included people having treatments as outpatients. Both trials had 2 phases. The first phase was single arm and measured correction of hyperkalaemia. All patients with serum potassium values of 5.1 mmol/litre or more (but under 6.5 mmol/litre) had sodium zirconium cyclosilicate. The second phase measured how well sodium zirconium cyclosilicate maintained serum potassium levels in people whose levels had responded to the initial treatment and were between 3.5 mmol/litre to 5.0 mmol/litre. In ZS004, 'responders' were randomised to continue sodium zirconium cyclosilicate or placebo for 28 days. In ZS005, all 'responders' had sodium zirconium cyclosilicate for 52 weeks. The committee appreciated that the primary outcome measure in both trials was serum potassium levels. The single-arm part of ZS005 measured changing use of RAAS inhibitors as an exploratory endpoint. However, the single-arm design of this trial meant that there were no data on whether, compared with standard care, sodium zirconium cyclosilicate allowed a higher proportion of patients to maintain RAAS inhibitors, a key potential benefit suggested by the company (see section 3.3). The committee concluded that the company had not provided any data for the clinical effectiveness of treatments currently used in the NHS to correct hyperkalaemia and maintain normal serum potassium levels in the outpatient setting (that is, a low-potassium diet and management of RAAS inhibitors). It also noted that the trials did not include any data from people with life-threatening hyperkalaemia. Without these data, it could not determine whether sodium zirconium cyclosilicate

was more clinically effective than current standard care in the NHS in either an outpatient or emergency setting.

Trial results show sodium zirconium cyclosilicate may lower serum potassium levels but the benefit of this to patients is unclear

3.9 In ZS004 and ZS005, sodium zirconium cyclosilicate was associated with reduced serum potassium levels from over 5.0 mmol/litre to within the normal range of 3.5 mmol/litre to 5.0 mmol/litre in around 70% of patients in the correction phase of these trials. The average serum potassium levels in patients in ZS004, over the 28 days after the correction phase, was 5.1 mmol/litre in the placebo group, and 4.8 mmol/litre and 4.5 mmol/litre in the 5 g and 10 g daily doses of sodium zirconium cyclosilicate groups respectively, all of which fell within a range that would not be treated further in the NHS. The committee noted:

- There was no control group for the correction period of the trial. This meant that it was unknown whether the proportion of patients whose potassium returned to the normal range was similar to what is seen with standard care (see section 3.8).
- The trial started treatment in patients with serum potassium greater than 5.0 mmol/litre. Clinicians in the NHS would not typically offer treatment at this level (see section 3.1).
- Clinicians in the NHS may not always view a serum potassium level of below 5.0 mmol/litre as the target for treatment, as long as serum potassium levels are reduced to non-life-threatening levels, depending on the serum potassium level that precipitated treatment (see section 3.1).
- A key outcome for clinicians would be the proportion of people whose serum potassium levels dropped to below 6.0 mmol/litre, the level above which NICE recommends stopping RAAS inhibitors, but this was not an outcome in the trial.
- Hyperkalaemia may be asymptomatic; around 85% of patients in the clinical trials had serum potassium levels under 6.0 mmol/litre when

they started corrective treatment with sodium zirconium cyclosilicate. This meant the trial did not capture the effect of sodium zirconium cyclosilicate on symptoms of hyperkalaemia.

- Symptoms of hyperkalaemia may be similar to symptoms of the underlying condition, for example, heart failure. So, treating hyperkalaemia may not in itself result in a noticeable effect on symptoms.
- The follow-up in ZS004 trial was 28 days, during which the trial was placebo-controlled. The average serum potassium level in patients who were randomised to placebo during this time was around 5.1 mmol/litre, which was lower than the level that would need treatment. It is unclear whether, without further treatment, serum potassium levels would have risen to a level needing treatment after 28 days.

The committee concluded that, although the trial results showed that continuing sodium zirconium cyclosilicate was associated with lower serum potassium than stopping sodium zirconium cyclosilicate, the benefit of this to patients seen in clinical practice was unclear.

The company submission is not relevant to how hyperkalaemia is treated in NHS as an emergency or in an outpatient setting

3.10 Overall, the company focused its submission on a population with serum potassium levels reflecting a wider range than would be typically treated with potassium-lowering drugs or by stopping RAAS inhibitors in clinical practice in the NHS (see sections 3.1). In particular, the committee concluded that the population the company stated would be treated as outpatients because of non-life-threatening hyperkalaemia (that is, people with serum potassium levels of 5.5 mmol/litre to 6.0 mmol/litre) do not typically have potassium-lowering treatment in the NHS (see sections 3.1 and 3.7). Furthermore, ZS004 and ZS005 included patients with even lower serum potassium levels. The committee agreed that focusing on people with serum potassium levels of 6.0 mmol/litre and more would

better reflect clinical practice in England for treating hyperkalaemia in the outpatient setting. It noted that it had seen no data for people with life-threatening hyperkalaemia who would be treated in the emergency setting because this population was not included in the trial. The committee concluded that, overall, the company submission was not relevant to how hyperkalaemia is treated in the NHS as an emergency or in an outpatient setting.

There is no evidence that sodium zirconium cyclosilicate prolongs survival

3.11 ZS004 and ZS005 did not collect data on the effect of sodium zirconium cyclosilicate on long-term outcomes such as progression of chronic kidney disease or mortality. However, the company proposed that people with hyperkalaemia who have sodium zirconium cyclosilicate live longer than people who do not. It presented data from a range of literature sources:

- Published data from UK and US observational cohort studies: this showed a higher risk of death associated with high, but also with lower than normal, serum potassium levels, compared with a reference value of 4.5 mmol/litre to 4.9 mmol/litre. Using these data, the company assumed that sodium zirconium cyclosilicate prolongs life because it lowers serum potassium, and because patients with higher serum potassium compared with high normal values have a higher risk of death.
- Published data from a network meta-analysis of randomised controlled trials: this compared survival when people started treatment with a RAAS inhibitor with placebo. The company assumed that because this study showed that starting a RAAS inhibitor is associated with living longer, people who stop a RAAS inhibitor have a shorter life expectancy.
- Published data from a randomised controlled trial on the risk of progression of chronic kidney disease in people who started taking a RAAS inhibitor compared with placebo: the company used this study to support the assumption that people who stop a RAAS inhibitor do not

live as long as people who continue a RAAS inhibitor because RAAS inhibitor use was associated with slower chronic kidney disease progression.

- An assumption that people on current standard care in the outpatient setting would stop RAAS inhibitors if they developed hyperkalaemia but that people taking sodium zirconium cyclosilicate would continue RAAS inhibitors despite hyperkalaemia: the serum potassium level at which people stopped or reduced their RAAS inhibitor dosage was assumed to be 5.5 mmol/litre.

The committee noted several limitations with the company's assumptions:

- There was no evidence for a difference in serum potassium levels on standard care and while taking sodium zirconium cyclosilicate for a population that would be treated in the NHS (see section 3.8).
- There was no comparative evidence from a randomised trial that lowering serum potassium in people with hyperkalaemia, prolonged survival.
- The company provided evidence from a single observational study showing an association between serum potassium levels and death, but did not provide a systematic review of the evidence. The committee recognised that this observational data did not guarantee an independent association between high serum potassium levels and death, or provide evidence that lowering serum potassium extends life.
- The committee recognised that people with hyperkalaemia may have differences in other clinical characteristics compared with people without hyperkalaemia, which may increase their risk of death. These differences may be unmeasured or unknown, and the extent to which the company took these into account was unclear.
- The 'normal' range as defined by the company (3.8 mmol/litre to 5.1 mmol/litre) overlapped with the category of serum potassium

(3.5 mmol/litre to 3.9 mmol/litre) associated with an increased risk of death relative to people with serum potassium values of 4.5 mmol/litre to 4.9 mmol/litre. The committee was concerned that if the associations between serum potassium and death were true, using sodium zirconium cyclosilicate to lower blood serum potassium to the levels proposed by the company could actually increase the risk of death.

- It was unclear whether the benefits of starting RAAS inhibitors on survival and lower progression of chronic kidney disease (that had been assessed in the network meta-analyses of trials) were the same as the risks of stopping RAAS inhibitors to manage serum potassium levels. This was because patients may change to another antihypertensive drug.
- It was unclear whether the company had taken into account in its analyses the factors that may affect the balance of harms and benefits of continuing RAAS inhibitors in people with hyperkalaemia (see section 3.4).

The committee concluded that the company had not proven that treatment with sodium zirconium cyclosilicate prolongs survival.

Cost-effectiveness modelling

The model does not generate valid estimates of cost effectiveness for the NHS

3.12 The committee agreed that the company's modelling approach was of limited use for decision-making. The clinical trials providing evidence for the model did not reflect who would have treatment for hyperkalaemia in the NHS. Also, they did not provide randomised comparative data for starting compared with not starting sodium zirconium cyclosilicate in this population. In addition, the results of the cost-effectiveness model were driven by the assumed surrogate relationship between serum potassium levels and mortality and other long-term outcomes (see section 3.11) without evidence that lowering serum potassium with sodium zirconium cyclosilicate lowers the incidence of these outcomes. Because of this, the

cost-effectiveness results did not address the clinical problem. In addition, the committee noted further limitations:

- The company did not model the relationship between having a RAAS inhibitor and serum potassium.
- The model excluded outpatient follow-up of people who had had treatment for hyperkalaemia in hospital.
- The model did not account for the proportion of people whose hyperkalaemia was not corrected by sodium zirconium cyclosilicate.
- The modelling of sodium zirconium cyclosilicate in people with underlying chronic kidney disease or heart failure did not account for people having both conditions.
- The model did not address the possibility of a dose-dependent relationship between RAAS inhibitors and more advanced renal disease, cardiovascular disease and death.
- The model did not address the fact that some people may stop RAAS inhibitors for reasons other than hyperkalaemia.
- The ERG's utility values for chronic kidney disease were more plausible than the company's because utility is expected to decrease as kidney disease progresses.
- The ERG's assumptions on the costs of changing RAAS inhibitor dosage were more plausible than the company's because the consultations to change dosage would be expected to be done in an outpatient rather than an inpatient setting.

The committee concluded that the output of the company's cost-effectiveness modelling was not useful for decision-making.

Innovation

The company has not shown that sodium zirconium cyclosilicate is innovative

3.13 The company proposed several benefits of sodium zirconium cyclosilicate, including preventing the need to modify RAAS inhibitor treatment and

avoiding a restrictive low-potassium diet. The patient experts stated that if the company had shown evidence for these benefits, then sodium zirconium cyclosilicate would be innovative. The committee concluded, however, that the clinical effectiveness and evidence supporting the benefits of sodium zirconium cyclosilicate had not been shown, so it could not be considered innovative.

Conclusion

Sodium zirconium cyclosilicate is not recommended

3.14 There was insufficient evidence on how sodium zirconium cyclosilicate affects quality of life and survival compared with standard care in a population relevant to clinical practice in the NHS. This meant the committee was not presented with a plausible estimate of cost effectiveness. Therefore, the committee did not recommend sodium zirconium cyclosilicate for treating hyperkalaemia in adults.

4 Proposed date for review of guidance

4.1 NICE proposes that the guidance on this technology is considered for review by the guidance executive 3 years after publication of the guidance. NICE welcomes comment on this proposed date. The guidance executive will decide whether the technology should be reviewed based on information gathered by NICE, and in consultation with consultees and commentators.

Amanda Adler
Chair, Appraisal Committee
October 2018

5 Appraisal committee members and NICE project team

Appraisal committee members

The 4 technology appraisal committees are standing advisory committees of NICE. This topic was considered by [committee B](#).

Committee members are asked to declare any interests in the technology to be appraised. If it is considered there is a conflict of interest, the member is excluded from participating further in that appraisal.

The [minutes](#) of each appraisal committee meeting, which include the names of the members who attended and their declarations of interests, are posted on the NICE website.

NICE project team

Each technology appraisal is assigned to a team consisting of 1 or more health technology analysts (who act as technical leads for the appraisal), a technical adviser and a project manager.

Mary Hughes

Technical Lead

Ross Dent

Technical Adviser

Jeremy Powell

Project Manager

ISBN: **[to be added at publication]**