Adding abiraterone or docetaxel to long-term hormone therapy for prostate cancer: directly randomised data from the STAMPEDE multi-arm, multi-stage platform protocol


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Background: Adding abiraterone acetate with prednisolone (AAP) or docetaxel with prednisolone (DocP) to standard-of-care (SOC) each improved survival in systemic therapy for advanced or metastatic prostate cancer: evaluation of drug efficacy: a multi-arm multi-stage platform randomised controlled protocol recruiting patients with high-risk locally advanced or metastatic PCa starting long-term androgen deprivation therapy (ADT). The protocol provides the only direct, randomised comparative data of SOC + AAP versus SOC + DocP.

Method: Recruitment to SOC + DocP and SOC + AAP overlapped November 2011 to March 2013. SOC was long-term ADT or, for most non-metastatic cases, ADT for ≥2 years and RT to the primary tumour. Stratified randomisation allocated pts 2 : 1 : 2 to SOC + docetaxel 75 mg/m² 3-weekly × 6 + prednisolone 10 mg daily; or SOC + abiraterone acetate 1000 mg + prednisolone 5 mg daily. AAP duration depended on stage and intent to give radical RT. The primary outcome
measure was death from any cause. Analyses used Cox proportional hazards and flexible parametric models, adjusted for stratification factors. This was not a formally powered comparison. A hazard ratio (HR) < 1 favours SOC + AAP, and HR > 1 favours SOC + DocP.

**Results:** A total of 566 consenting patients were contemporaneously randomised: 189 SOC + DocP and 377 SOC + AAP. The patients, balanced by allocated treatment were: 342 (60%) M1; 429 (76%) Gleason 8–10; 449 (79%) WHO performance status 0; median age 66 years and median PSA 56 ng/ml. With median follow-up 4 years, 149 deaths were reported. For overall survival, HR = 1.16 (95% CI 0.82–1.65); failure-free survival HR = 0.51 (95% CI 0.39–0.67); progression-free survival HR = 0.65 (95% CI 0.48–0.88); metastasis-free survival HR = 0.77 (95% CI 0.57–1.03); prostate cancer-specific survival HR = 1.02 (0.70–1.49); and symptomatic skeletal events HR = 0.83 (95% CI 0.55–1.25). In the safety population, the proportion reporting ≥ 1 grade 3, 4 or 5 adverse events ever was 36%, 13% and 1% SOC + DocP, and 40%, 7% and 1% SOC + AAP; prevalence 11% at 1 and 2 years on both arms. Relapse treatment patterns varied by arm.

**Conclusions:** This direct, randomised comparative analysis of two new treatment standards for hormone-naïve prostate cancer showed no evidence of a difference in overall or prostate cancer-specific survival, nor in other important outcomes such as symptomatic skeletal events. Worst toxicity grade over entire time on trial was similar but comprised different toxicities in line with the known properties of the drugs.

**Trial registration:** Clinicaltrials.gov: NCT00268476.

**Key words:** prostate cancer, randomised, treatment, abiraterone, docetaxel, head-to-head

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**Research in context**

**Evidence before this study**

Abiraterone acetate plus prednisone/prednisolone (AAP) and docetaxel with prednisone/prednisolone (DocP) have separately been shown to improve survival when used in addition to the previous international standard-of-care (SOC) for hormone-sensitive prostate cancer of androgen deprivation therapy with further therapy such as AAP or DocP on relapse. This has been confirmed in a number of separate trials and on meta-analysis. The largest body of evidence for both AAP and DocP comes from the systemic therapy for advanced or metastatic prostate cancer: evaluation of drug efficacy (STAMPEDE) platform trial.

**Added value of this study**

Recruitment to DocP and AAP overlapped in STAMPEDE giving the only head-to-head evidence comparing these two new standard treatment approaches. We report data from the 566 patients who were directly randomised between these two treatment approaches while the two research arms were both open to recruitment. The data show strong evidence favouring SOC + AAP on earlier, more biochemically driven outcome measures (OMs). For longer-term, more clinically driven OMs, including bone complications, prostate cancer-specific and overall survival, there is no evidence of a significant difference between AAP and DocP.

**Implications of all the available evidence**

The reported trials and meta-analyses showed a larger effect on survival for AAP over the previous SOC than did DocP over the standard SOC. These data show that the story may be more complicated. No other directly randomised data on survival of these treatments are available. Individual patient data network meta-analysis using all of the published trials are warranted, accounting for differences in patient characteristics, treating clinicians and centres and salvage treatment access. The STAMPEDE team is collaborating with the STOPCAP meta-analysis group to achieve this.

**Introduction**

For several decades, the standard-of-care (SOC) for most patients with high-risk locally advanced or metastatic prostate cancer has been long-term androgen deprivation therapy (ADT) alone. The past few years, there have been great changes, first with results from randomised controlled trials (RCTs) showing a survival advantage compared with ADT alone for adding radiotherapy to the prostate in men with non-metastatic disease and no known nodal involvement [1–3]; then with systemic treatments for all men starting long-term hormone therapy: docetaxel plus prednisolone/prednisone (DocP) [4–9] and, most recently, abiraterone acetate plus prednisolone/prednisone (AAP) [10, 11]. As both therapeutic combinations are effective, there are now two distinct standards-of-care with little information to guide clinicians as to which is the more effective; there are no prospective, powered, RCTs that will deliver direct comparative data.

Systemic therapy for advanced or metastatic prostate cancer: evaluation of drug efficacy (STAMPEDE) is a multi-arm, multi-stage platform protocol which assessed both of these treatment approaches, separately, against the previous SOC [12, 13]. The ‘docetaxel comparison’ of STAMPEDE recruited patients allocated to SOC + DocP between October 2005 and March 2013. The ‘abiraterone comparison’, the first comparison to be added to STAMPEDE, recruited patients allocated to SOC or SOC + AAP between November 2011 and January 2014. Each of those comparisons had primary outcome measure (OM) of overall survival (OS) for the patients randomised contemporaneously to the control arm and the relevant research arm. Consequently, between 15 November 2011 and 31 March 2013, patients were directly randomised contemporaneously between these two research arms (and other research arms) and we now present these data.
Methods

Trial design

The STAMPEDE protocol and design have been described in detail elsewhere [7, 10, 12, 14]. Briefly, STAMPEDE comprises a series of multi-arm multi-stage (MAMS) comparisons that have overlapped in recruitment and follow-up time.

Patient selection

Eligible patients were those starting long-term ADT for the first time. This was defined as patients with metastatic disease, nodal involvement or node negative, non-metastatic disease with two or more of three high-risk features: T-category 3 or 4, Gleason sum score 8–10 or PSA > 40 ng/ml. Patients rapidly relapsing after previous local therapy were also permitted if they had PSA > 20 ng/ml or PSA > 4 ng/ml with a PSA doubling time <6 months or those who developed loco-regional or metastatic spread whilst not on hormone therapy.

As with all STAMPEDE comparisons, the primary OM of the two underpinning comparisons (against control) was OS. Failure-free survival (FFS) was an intermediate primary OM, defined as time from randomisation to the first of: rising PSA (where rising PSA was defined as a confirmed rise to >4 ng/ml, and >50% above the lowest value in the first 6 months after randomisation); new disease or progression of: distant metastases, lymph nodes or local disease; or death from prostate cancer. Progression-free survival (PFS) was defined as time from randomisation to the first of: new disease or progression of: distant metastases, lymph nodes or local disease; or death from prostate cancer [15]. Metastatic PFS (MPFS) was defined as time from randomisation to death from any cause, new metastases or progression of distant metastases.

All patients provided written informed consent; all versions of the protocol have been reviewed by the relevant research ethics committees and the regulatory agencies; the original protocol and all subsequent versions involving the introduction of a new research arm and comparison were independently peer-reviewed by Cancer Research UK (CRUK).

Patients have been allocated across a number of research treatments as depicted in Figure 1. Here we focus on those patients randomised between 15 November 2011 and 31 March 2013, while both the ‘docetaxel comparison’ and the ‘abiraterone comparison’ were open to recruitment, and who were allocated to either SOC + DocP or SOC + AAP.

Trial treatment, masking and follow-up

The SOC was long-term hormone therapy with LHRH analogues (with short term antiandrogen if relevant) or orchidectomy. Unless contraindi-
cated, radiotherapy to the prostate was mandated in all patients with N0M0 disease, encouraged in patient with N + M0 disease, and permitted in patients with M1 disease until the activation of the ‘M1|RT compare-

Statistical analysis

The comparison presented here is of SOC + AAP against SOC + DocP because both of these arms have demonstrated better OS than their contemporaneous controls in the population of men starting long-term hormo-

Results

Accrual and characteristics

The dataset for this comparison was frozen on 10 February 2017. Between 15 November 2011 and 31 March 2013, 1348 patients joined all open arms STAMPEDE. Of the 566 randomised to the comparison reported here, 189 (14%) were allocated to SOC + DocP, 377 (28%) to SOC + AAP. The flow of patients to this comparison is shown in Figure 2. Table 1 shows the baseline characteristics of patients in this comparison which differ only slightly from the previous papers (summarised in supplementary Table S1, available at Annals of Oncology online). Median follow-up, calculated by reverse censoring on survival, was 48 months.

Overall survival

There were 44/189 (23%) deaths on the SOC + DocP arm and 105/377 (28%) deaths on the SOC + AAP arm. The estimated HR = 1.16 (95% CI 0.82–1.65; P = 0.40) (Figure 3A). Estimates in patients with and without metastases are shown in Table 2, with HR = 1.51 (95% CI 0.58–3.93) in M0 patients and HR = 1.13 (95% CI 0.77–1.66) in M1 patients. There was no evidence of interaction in the treatment effect by baseline metastases (P = 0.69).

Totally, 126/149 deaths were attributed to prostate cancer, comprising 10/22 and 116/127 deaths in patients with M0 and M1 disease at entry, respectively. Competing risks regression shows no evidence of a difference in prostate cancer-specific
STAMPEDE: Docetaxel vs abiraterone -- direct comparison

Figure 1. Activity-by-time diagram: patients included in this comparison. SOC, standard-of-care; Doc, docetaxel; Abi, abiraterone acetate-prednisolone/prednisone. Boxes represents periods of recruitment (x-axis) to each of the trial arms (y-axis). The blue boxes represent recruitment periods contributing to this analysis; the green boxes other recruitment period, past and future, contributing to other aspects of the STAMPEDE. The squares represent the time point of the first key comparative analyses for each comparison in pink and for this comparison in blue.

Figure 2. CONSORT diagram. SOC, standard-of-care; DocP, docetaxel-prednisolone/prednisone; AAP, abiraterone acetate-prednisolone/prednisone. Selection of patients for this comparison.
Table 1. Baseline characteristics of patients allocated to SOC + DocP or SOC + AAP by whether contributing to the direct comparison

<table>
<thead>
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<th>Characteristic</th>
<th>SOC + DocP</th>
<th>SOC + AAP</th>
<th>Overall</th>
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<td>N</td>
<td>%</td>
<td>N</td>
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<tr>
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<td>25</td>
<td>97</td>
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<tr>
<td>Median (quartiles)</td>
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<td>(29–162)</td>
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<tr>
<td>Mean (SD)</td>
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<td>(421)</td>
<td>274</td>
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<tr>
<td>2013</td>
<td>36</td>
<td>19</td>
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</table>
For non-prostate cancer-specific survival, with 23/149 deaths attributed to other causes, the sub-HR was 2.33 (95% CI 0.78–6.99). There was no evidence of heterogeneity of treatment effect by baseline metastases in either outcome.

Other efficacy OMs
Table 2 shows the effect size overall and by whether the patients had metastases at entry for FFS, PFS, MPFS and skeletal-related events. There is no evidence of heterogeneity of the treatment
effect by baseline metastases in any of these OMs. Figure 4 summarises the effect for all OMs.

Safety

The safety population includes people who started their allocated treatment. While nearly all patients allocated to AAP started it, a proportion of those patients allocated to receive docetaxel declined to start it. Table 3 summarises the worst toxicity reported for patients over their time on trial in the safety population and shows differing patterns for adverse events according to treatment. The prevalence of grade 3 or 4 toxicity in patients with assessments at 1 year without a prior FFS event was 11% SOC + DocP and 11% SOC + AAP; at 2 years this was 11% SOC + DocP and 11% SOC + AAP.

Second-line treatment

Figure 5 shows time from randomisation to any subsequent exposure to docetaxel or AR-targeted therapy with AAP or enzalutamide. Figure 6 shows time from an FFS event to reported exposure to selected treatments that are licensed for CRPC: docetaxel, AAP, enzalutamide. There was limited reported use of cabazitaxel, radium and sipuleucel-T at this point (not shown).
We and others have previously shown a survival advantage for adding docetaxel (with or without prednisolone/prednisone) and for adding abiraterone acetate and prednisolone/prednisone, in patients starting long-term hormone therapy for the first time [4–11]. However, there is currently no direct evidence available to help clinicians or patients assess which combination might be better. Here, we reported a pre-specified (but not pre-powered) analysis using only patients who were randomised during a period of the study when recruitment to the two research arms overlapped. We used data collected prospectively from over 100 sites across two countries as part of a clinical trial protocol. The MAMS platform design of STAMPEDE, an approach sometimes referred to as a master protocol [16], facilitated this comparison. Separate, traditional, two-arm RCTs, would not have allowed any directly randomised comparative evidence to be available so soon.

Our recently reported overall treatment effect on survival, in STAMPEDE, for adding AAP compared with the SOC (HR = 0.63) [10] was larger than the previously-reported overall treatment effect, in STAMPEDE, on survival for adding DocP to the same SOC (HR = 0.78) [7]. The earlier secondary efficacy OMs favoured adding AAP over DocP, including FFS—perhaps unsurprising given the direct antiandrogenic action of AAP (around four in every five FFS events was driven only by a rise in PSA) and PFS (which excludes rising PSA). There was weak evidence favouring AAP for MPFS and no evidence of a difference in symptomatic skeletal events, prostate cancer-specific survival or OS.

Comparing the results indirectly of these two therapies by readers extracting data from STAMPEDE’s AAP and docetaxel papers [7, 10] may not be the most appropriate way to compare the relative effectiveness: the patient cohorts were all not randomised contemporaneously and there may be confounding biases when comparing the two datasets, in particular, many DocP patients had very limited salvage CRPC options compared with AAP patients, simply due to the timing of licences of new therapies (see below).

Importantly, the two therapies are being used in different ways. AAP is used until the patient has castrate-resistant prostate cancer (CRPC), often lasting many years and consequently exhausting a major therapy option for CRPC. In contrast, DocP is given as an 18-week course thus all CRPC options should remain available. Our data reveal important differences in the pattern of treatment failure yet we do not see any differences in survival, suggesting that the relative time spent before and after first-line treatment failure are quite different by initial treatment. This may explain why the early, often biochemically driven OMs, favour AAP but the later post CRPC end points such as skeletal events, prostate cancer-specific survival and OS show no good evidence of a difference. Men receiving DocP will thus spend longer with CRPC than men receiving AAP but with a broader range of more effective options available. Supplementary Figure S1, available at...
**Figure 5.** Time from randomisation to reported starting docetaxel, AAP, enzalutamide or AR-targeting therapy. Kaplan–Meier (survival) plots showing cumulative incidence of exposure to treatments after randomisation. Each step up the y-axis represents an event, namely starting that particular treatment. The number of patients contributing information (at risk) over time since randomisation is shown under the table. The number of patients with an event between these points is shown in brackets. For example, in Figure 4C between 24 and 36 months after randomisation, 4 patients on the SOC+DocP arm report starting abiraterone and (150—129) = 4 are 17 are censored and may start in the future.

**Figure 6.** Time from failure-free survival event to subsequent treatment by allocated treatment. Kaplan–Meier (survival) plots showing cumulative incidence of exposure to treatments after a failure-free survival (FFS) event. Doc, docetaxel; AAP, abiraterone acetate + prednisolone; Enz, enzalutamide. Each step up the y-axis represents an event, namely starting that particular treatment.
The unequal power to detect any heterogeneity is very limited, especially in later OMs with fewer events. The patterns of toxicity are quite different for the two treatment approaches, consistent with the known effects of the drugs. The proportion of patients reporting at least one grade 3 or worse toxicity was similar and in line with previously reported toxicities for these agents (Table 3). In patients who started their allocated treatment and who are without disease progression at 1 year, the prevalence of grade 3 or worse toxicity was about 11% on both arms and very similar to our previous estimate for SOC. Nearly all patients started their allocated abiraterone, whereas about 1 in 12 patients did not start their allocated docetaxel. Our results may change future compliance with both treatments in routine practice; but the lack of compliance with allocated treatment of docetaxel is likely to have had some impact on our estimated effect sizes.

A key limitation is that the comparison was opportunistic and not designed in the usual way, hence power is limited to detect any realistic differences. The trigger for the analysis was the re-analysis of our ‘abiraterone comparison’ data [10]. The unequal allocation ratio reflects the planned design of the comparisons. The allocated treatment being given was not masked for practical reasons. This, of course, allowed for relapse therapies to be given at the investigator’s discretion. We observed that after relapse, many patients received the treatment class that they had not received up-front.

Salvage options have changed over time: men recruited earlier on to DocP (2005–2013) will have had very different options to those recruited later to AAP (2011–2014) when there were more CRPC therapies likely available, including AAP [17, 18], cabazitaxel [19], docetaxel [20, 21], enzalutamide [22, 23], radium-223 [24] and sipuleucel-T [25] (although not widely accessible in Europe). For this analysis, we limited ourselves to patients contemporaneously randomised to either arm to make this comparison as fair as possible. However, FFS events generally happened sooner with DocP than with AAP in time from randomisation and, therefore, calendar year (Table 4) may partially influence outcomes. Furthermore, a FFS event was more of an indication to change treatments on DocP; AAP continued beyond this point.

As far as we are aware there are no ongoing randomised trials directly comparing adding AAP versus adding docetaxel for patients starting long-term ADT. All of our published STAMPEDE data have contributed to the STOPCaP aggregate data network meta-analysis that has used all of the reported RCTs in metastatic patients to perform indirect comparisons and allow some assessment of potential ranking of effective therapies. This aggregate data analysis (co-submitted) will be supplemented by a forthcoming individual patient data (IPD) network meta-analysis which will hopefully provide a more accurate reflection of the temporal interval between the application of the two different therapies, to which STAMPEDE will contribute all relevant data. We will continue to follow-up patients for long-term OMs.

Considering their mechanisms of action and their proven oncological benefits, the question is raised of whether a combination of AAP plus docetaxel might lead to an approximately additive benefit of using them both, further extending survival. Randomised data on docetaxel with or without abiraterone will emerge from a subset the PEACE-1 trial (https://clinicaltrials.gov/ct2/show/NCT01957436), as will non-randomised, time-stratified data on abiraterone with or without docetaxel. Similarly comparative data will also emerge for enzalutamide, another AR-targeted therapy, from the ENZAMET trial (https://clinicaltrials.gov/ct2/show/NCT02446405) and with the combination of enzalutamide and AAP in STAMPEDE (Figure 1).

In conclusion, there are now two systemic therapies, DocP and AAP, which have shown a survival benefit from RCTs when added to treatment of patients starting long-term ADT for the first time. The evidence from our directly randomised data comparing these two therapies showed no evidence of a difference in overall or prostate cancer-specific survival, nor in other important outcomes such as symptomatic skeletal survival, suggesting that both currently remain viable new standards-of-care.

Acknowledgements

Independent oversight committee members

Independent Data Monitoring Committee: John Yarnold (chair), Doug Altman, Ronald de Wit, Bertrand Tombal; Previous—Reg Hall, Chris Williams

Trial Steering Committee: Jonathan Ledermann (chair), Jan Erik Damber, Richard Emsley, Alan Horwich; Previous—John Fitzpatrick, David Kirk, Jim Paul

Participating site list

Structure: City, Hospital (Number of patients by data freeze: site PI; other investigators)
Aberystwyth, Bronglais General Hospital (4: Porfiri; Durrani)
Ashford William Harvey Hospital (19: Thomas; Mithal)
Aylesbury, Stoke Mandeville Hospital (14: Sabharwal; Camilleri)
Ayr Hospital (54: Glen; Ansari)
Barnet General Hospital (25: McGovern; Eichholz)
Basingstoke & N Hampshire Hospital (21: Shaffer)
Bath, Royal united Hospital (70: Frim; Beresford)
Belfast City (191: O’Sullivan; Mitchell, Stewart, Shum)
Birmingham, City Hospital (26: Sivoglo; Ford)
Birmingham, Good Hope Hospital (18: Ford)
Birmingham, Heartlands Hospital (38: Zarkar)
Birmingham, QE (180: James; Porfiri, Ford)
Blackburn East Lancashire Trust (180: Parikh; Charnley)
Bolton, Royal Bolton Hospital (30: Elliott, Maddineni)
Bournemouth, Royal Bournemouth Hospital (100: Brock)
Bradford Royal Infirmary (36: Brown)
Brighton, Royal Sussex County Hospital (92: Robinson; Robinson, Bloomfield)
Bristol Haematology & Oncology Centre (106: Bahl; Herbert, Masson)
Burton, Queen’s Hospital (108: Smith-Howell; Chetiyawardana, Pattu)
Bury St Edmunds, West Suffolk Hospital (21: Woodward)
Cardiff, Velindre (341: Lester; Staffurth, Barber, Kumar, Palaniappan, Button, Tanguay)
Chelmsford, Broomfield Hospital (88: Hamid; Panwar, Leone)
Cheltenham General Hospital (54: Bowen)
Chester, Countess of Chester Hospital (79: Ibrahim)
Coventry & Warwickshire, University Hospital (40: Worling; Stockdale)
Crewe & Warwickshire, University Hospital (54: Wylie)
Cumbria, Cumberland Infirmary (18: Kumar)
Darlington Memorial Hospital (49: Kagzi; Hardman, Peedell)
Derby, Royal Derby Hospital (130: Chakraborti; Pattu)
Devon, North Devon District Hospital (33: Sheehan)
Doncaster Royal Infirmary (35: Bowen; Ferguson)
Dorset County Hospital (30: Crellin; Afzal, Andrews)
Dudley, Russells Hall Hospital (81: Keng-Koh; Ramachandra)
Durham University Hospital (17: Heath; McMenemin)
Eastbourne District General Hospital (63: McKinna)
Edinburgh, Western General (112: McLaren)
Essex County Hospital (58: Muthukumar; Sizer, Kumar)
Exeter, Royal Devon & Exeter (189: Sheehan; Srinivasan)
Gillingham, Medway Hospital (29: Kumar; Taylor)
Glasgow, Beatson West of Scotland Cancer Centre (323: Graham; Venugopal, Wallace, Jones, Lamb, Glen, Russell)
Guildford, Royal Surrey County Hospital (12: Laing; Khaksar, Wood, Money-Kyrle)
Harlow, Princess Alexandra Hospital (54: Gupta; Melcher, Melcher)
Hereford County Hospital (71: Grant; Cook)
Huddersfield Royal Infirmary (105: Hofmann)
Hull, Castle Hill Hospital (119: Simms; Hetherington)
Inverness, Raigmore Hospital (88: McPhail; MacGregor)
Ipswich Hospital (103: Brierly; Venkitaraman, Scrase)
Keighley, Airedale Hospital (52: Brown; Crawford)
Kent and Canterbury Hospital (79: Thomas; Raman, Mithal, Malde)
Kent, Queen Elizabeth Queen Mother Hospital (27: Thomas; Raman)
Kidderminster General Hospital (40: Capaldi; Churn)
Larbert, Forth Valley Royal Hospital (36: Sidek)
Leeds, St James University Hospital (94: Cross; Loughrey, Bottomley, Prescott)
Lincoln County Hospital (50: Sreenivasan; Ballesteros-Quintain, Panades, Baria)
Liverpool, Royal Liv University Hospital (88: Malik; Robson, Esvar)
Liverpool, University Hospital Aintree (26: Robson)
London, Charing Cross Hospital (38: Falconer; Mangar)
London, Guy’s Hospital (161: Chowdhury)
London, Hammersmith Hospital (4: Falconer; Mangar)
London, North Middlesex Hospital (24: Gupta; Newby, Thompson)
London, Royal Free Hospital (44: Vilarino-Varela; Pigott)
London, St Georges Hospital (35: Pickering)
London, St Mary’s Hospital (8: Falconer; Stewart)
London, University College Hospital (46: McGovern)
Maidstone, Kent Oncology Centre (114: Beesley)
Manchester Christie Hospital (167: Clarke; Elliott, Livsey, Choudhury, Wylie)
Manchester Hope Hospital (59: Clarke; Elliott, Lau, Tran)
Manchester, Royal Oldham Hospital (54: Conroy; Livsey, Choudhury)
Manchester, Withington Hospital (7: Sangar)
Middlesbrough, James Cook UH (103: Pedell; Van der Voet, Hardman, Shakespeare)
Newcastle, Freeman Hospital (92: Azzabi; McMenemin, Frew)
North Staffordshire UH (80: Adab)
Northwood, Mount Vernon Hospital (126: Hoskin; Anyamene, Ostler, Alonzi)
Nottingham University Hospitals (City Campus) (141: Sundar; Mills)
Nuneaton, George Eliot Hospital (14: Khan; Chan)
Oxford, Churchill Hospital (165: Protheroe; Cole, Sabharwal, Sugden)
Poole Hospital (62: Davies)
Portsmouth, Q Alexandra Hospital (173: Gale)
Preston, Royal Preston Hospital (221: Birtle; Parikh, Wise)
Reading, Royal Berkshire Hospital (42: Rogers; O’Donnell, Brown, Brown)
Redditch, Alexandra Hospital (15: Capaldi; Hamilton)
Romford, Queen’s Hospital (127: Gibbs; Subramaniam)
Scarborough General Hospital (82: Hingorani)
Sheffield, Weston Park (142: Ferguson)
Shrewsbury, Royal Shrewsbury Hospital (192: Sриhari)
Somerset, Weston General Hospital (18: Hilman)
• Southampton General Hospital (75: Jones; Heath, Wheeler, Crabb)

• Southend University Hospital (114: Tsang; Ahmed, Chan)

• Southport and Formby District GH (46: Bhalla; Sivapalanitharam, Sivapalansantharam)

• St Georges-on-Sea, Conquest Hospital (42: McKinna; Beesley, Lees)

• Stevenage, Lister Hospital (35: Hughes)

• Stockport, Stepping Hill Hospital (106: Logue; Coyle)

• Stockton-on-Tees, UH North Tees (28: Leaning; Shakespeare)

• Sunderland Royal Hospital (45: Azzabi)

• Sutton-in-Ashford, King’s Mill Hospital (64: Saunders)

• Sutton and London, Royal Marsden Hospital (162: Dearnaley; Parker, Selvadurai)

• Swansea, Singleton (188: Wagstaff; Phan, Phan)

• Swindon, Great Western Hospital (52: Khan; Cole)

• Taunton, Musgrove Park Hospital (137: Gray; Graham; Varughese, Plataniotis)

• Torbay District General Hospital (135: Lydon; Srinivasan)

• Tyne & Wear, S Tyneside District Hospital (6: Azzabi)

• Warrington Hospital (111: Syndikus; Tolan)

• Warwick Hospital (17: Chan; Stockdale)

• Wigan, Royal Albert Edward Infirmary (37: Tran)

• Wirral, The Clatterbridge Cancer Centre NHS Foundation Trust (128: Tolan; Syndikus, Ibrahim, Montazeri, Littler)

• Wolverhampton, New Cross Hospital (53: Gray; Sayers)

• Woolwich, Queen Elizabeth Hospital (18: Hughes)

• Worcestershire Royal Hospital (57: Capaldi; Bowen)

• Worthing Hospital (90: Nikapota)

• Wycombe Hospital (52: Sabharwal; Protheroe, Pwint)

Switzerland

• Basel Universitatsklinik (5: Rentsch)

• Berne University Hospital (Inselspital) (5: Thalmann)

• Chur Kantonsspital Graubunden (31: Strebel; Cathomas)

• Kantonsspital St Gallen (10: Engeler)

• Lausanne, Centre Hospital Univ Vaudois (7: Berthold; Jichlinski)

Plus more than 3000 local site team staff across these hospitals.

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Funding

The trial was sponsored by the UK Medical Research Council (MRC) and conducted by the MRC Clinical Trials Unit at UCL. In the UK the trial was supported by the UK Clinical Research Network, and funded by CRUK and the MRC, and in Switzerland, by the Swiss Group for Cancer Clinical Research (SACK). Industry collaboration and support has been provided to STAMPEDE by Astellas, Clovis Oncology, Janssen, Novartis, Pfizer and Sanofi-Genzyme. MRC employees were central to the conduct of the trial and the development of this manuscript. Authors MRSy and MRSp accessed raw data. The funding bodies had no role in determining this publication.

Research support for trial: Cancer Research UK (CRUK_A12459), Medical Research Council (MRC_MC_UU_12023/25); Janssen, Sanofi-Aventis; Astellas, Clovis Oncology, Novartis, Pfizer. DPD, JSdeB, GA and CCP acknowledge NHS funding to the NIHR Biomedical Research Centre at the Royal Marsden NHS Foundation Trust and Institute of Cancer Research.

Disclosure

CA reports grants and non-financial support from Sanofi-Aventis, Novartis, Pfizer, Janssen, Astellas and Clovis Oncology during the conduct of the study. GA reports personal fees, grants and/or travel support from Janssen during the conduct of the study; personal fees and/or travel support from Astellas, Pfizer, Janssen, Millennium Pharmaceuticals, Ipsen, Ventana, Veridex, Novartis, Abbott Laboratories, ESSA Pharmaceuticals, Bayer Healthcare Pharmaceuticals, Takeda and Sanofi-Aventis and grant support from AstraZeneca, Innocrin Pharma and Arno Therapeutics, outside the submitted work; in addition, GA’s former employer, The Institute of Cancer Research, receives royalty income from abiraterone and GA receives a share of this income through the ICR’s Rewards to Discoverers Scheme. AB reports grants and personal fees from AstraZeneca, outside the submitted work. SB reports other...
from Janssen, outside the submitted work; and attendance at ESMO 2018 funded by Janssen. PC reports grants from Janssen, during the conduct of the study. AC reports funding from Prostate Cancer UK, Cancer Research UK, National Institute of Health Research, Medical Research Council and Astra Zeneca, outside the submitted work. SC reports grants and personal fees from Sanofi-Aventis and personal fees from Janssen Pharmaceutical, outside the submitted work. NWC reports personal fees from Janssen Pharmaceuticals, during the conduct of the study; personal fees from Janssen Pharmaceuticals, outside the submitted work; personal fees from Bayer and Astellas. WC reports personal fees from Janssen and Bayer, outside the submitted work. JdB reports other from ICR and Janssen, during the conduct of the study; other from AstraZeneca, Pfizer, GlaxoSmithKline, Taiho, Daiichi, Novartis, Genmab, Merck Serano, Merck and Genentech/Roche, outside the submitted work. DPP reports other from UK National Institute for Health Research Clinical Research Network (NIHR CRN), during the conduct of the study; grants from Cancer Research UK; personal fees and other from Takeda, Agen, Astellas and Sandoz, personal fees, non-financial support and other from Janssen, personal fees and other from Cadence Research, other from Clovis, personal fees and non-financial support from ISSECAM, outside the submitted work; in addition, DPD has a patent GB9305269-17-substituted steroids useful in cancer treatment with royalties paid to Janssen Pharmaceutical Company. DF reports honoraria from Janssen, Novartis and Sanofi, outside the submitted work. JG reports other support as a local principal investigator for a study of radium-223 in prostate cancer funded by Bayer, and other support as a local principal investigator for a study of LHRH antagonist in prostate cancer funded by Millennium Pharmaceuticals, outside the submitted work. SG reports personal fees from Bayer, other from Bayer and CureVac, personal fees from Janssen Cilag, other from Janssen Cilag, personal fees from Dendreon Corporation, other from Astellas, personal fees from Millennium Pharmaceuticals, personal fees from Orion, Sanofi and MaxiVax SA, other from AAA Advanced Accelerator Applications International, Bristol-Myers Squibb, Ferring, Roche, Orion, Innocrin Pharmaceuticals, Sanofi, Novartis, Nektar Therapeutics and ProteoMedix, outside the submitted work. CG reports grants from Clovis Oncology, outside the submitted work. NDJ reports grants and personal fees from Sanofi and Novartis, during the conduct of the study; grants and personal fees from Janssen, Astellas and Bayer, outside the submitted work. RJJ reports grants from Sanofi, and grants and non-financial support from Novartis, during the conduct of the study; grants, personal fees and non-financial support from Sanofi and Novartis, grants and personal fees from Janssen, Astellas and Bayer, outside the submitted work. Jl reports personal fees, non-financial support and other from Janssen, Astellas and Sanofi, outside the submitted work. ZIM reports personal fees from Janssen, outside the submitted work; in addition, DPD has a patent GB9305269-17-substituted steroids useful in cancer treatment with royalties paid to Janssen Pharmaceutical Company. CCP reports personal fees from AAA and Bayer and Astellas. CCP reports personal fees from AAA and Bayer, research funding and speaker’s honoraria from Bayer, outside the submitted work. MKBP reports grants and non-financial support from Janssen, during the conduct of the study; grants and non-financial support from Astellas, Clovis Oncology, Novartis, Pfizer and Sanofi, outside the submitted work. AP reports personal fees from Ipsen, Bayer, Roche and BMS, grants from Merck, personal fees from Merck, outside the submitted work. JMR reports personal fees from Janssen (lecture fee), outside the submitted work. DS reports conference travel costs from Ipsen and Astellas, outside the submitted work. MRSp reports grants and non-financial support from Sanofi-Aventis, Novartis, Pfizer, Janssen and Astellas, during the conduct of the study. MJ reports personal fees and non-financial support from Sanofi-Aventis, outside the submitted work. MRSy reports grants and non-financial support from Sanofi-Aventis, Novartis, Pfizer, Janssen and Astellas, during the conduct of the study; and personal fees from Eli-Lilly, outside the submitted work. ST reports other from Sanofi, other support from Astellas, personal fees from Astellas and other support from Janssen, outside the submitted work. MV reports travel grants from Janssen. JW reports a paid consultancy for Janssen. All remaining authors have declared no conflicts of interest.

References