SPORT RESOLUTIONS (UK)
SPECIALIST INDEPENDENT DISPUTE SERVICE

CASE 19 – Decision

NATIONAL ANTI-DOPING PANEL

1. This tribunal has been appointed to hear and determine a charge against Athlete R, a leading British cyclist, brought by UK Anti-Doping acting for the British Cycling Federation. The charge in a letter dated 23 December 2013 is that the Athlete R contravened article 21.2 of the UCI Anti-Doping Rules (ADR) in using a prohibited substance (an erythropoiesis-stimulating agent) and/or a prohibited method (blood doping and/or physical manipulation of blood) in around August/September 2012. It is alleged that he used a prohibited substance or method to boost the levels of haemoglobin in his blood for the Tour of Britain, staged from 9 to 16 September 2012, which he ended as the overall winner.

2. This very serious charge is made not on the basis of direct evidence from an adverse analytical sample detecting the presence of a prohibited substance in the blood or urine, but on the basis of expert opinion as to the conclusions to be drawn from a blood sample taken on 22 September 2012 under the UCI Athlete Biological Passport (ABP) programme. This is the first occasion on which the NADP has had to consider an ABP case although there have been a number of such cases decided by the Court of Arbitration for Sport.

3. The athlete biological passport involves regular monitoring of biological markers to enable indirect detection of the use of prohibited substances or methods, which may not be in use at the time when any particular sample is taken. The passport constructs a profile of the athlete's blood collated from a number of blood tests. The parameters taken into account include blood haemoglobin concentration, which is an indicator of the capacity of the blood to transport oxygen, and reticulocyte percentage, which indicates the recent red cell production from the bone marrow. There is a formula used to combine those two values to produce an OFF-score which is sensitive to changes in the process of red cell production, erythropoiesis. Over time the model, based on data derived from a general population of athletes, is adapted to adjust to the values obtained from samples taken from the individual athlete, so that his different physiological circumstances are taken into account. The purpose of the longitudinal profile generated is to provide reference data against which abnormalities in samples can be assessed by the adaptive model.

4. The sample taken on 22 September 2012 was the first to be taken from the rider after he entered the programme. Over the ensuing five months four further samples were taken to build up his longitudinal profile. Assessed against that profile three experts appointed by UCI concluded on 3 September 2013 that the readings from the first sample had been abnormal. It is alleged that the concentration of haemoglobin (Hb) (17.9 g/dL) and the percentage of immature blood cells, reticulocytes (0.15%) were well outside the parameters that would be expected for the rider in normal physiological circumstances. These two values combine to give a highly abnormal OFF- score value of 155.8. In the absence of a plausible explanation from the rider it is alleged that the inevitable inference is that he had engaged in some form of doping to increase his haemoglobin levels.

5. It is accepted in the expert evidence served on behalf of Athlete R that the values disclosed in the testing of the first sample were "wildly abnormal" and would be compelling evidence of the use of a prohibited substance or method, unless explained by some other factor.

6. The explanation put forward by Athlete R is that on the evening of 20 September 2012, approximately 32 hours before the sample was taken on 22 September, he went on an alcoholic binge. He then did not eat or drink on the following day, save for a few sips of water when he took painkillers. It is argued that this exceptional and extreme

intake of alcohol, followed by a period of severe dehydration, had had the effect, by the time the sample was taken on the morning of 22 September, both of decreasing the volume of plasma in his blood and hence the concentration of Hb, and inhibiting the release of reticulocytes from the bone marrow into his blood, thus causing a substantial decrease in the measured level of reticulocytes in the blood.

7. So the main issues in the case which we have to determine are:

(1) What was the level of alcohol intake of Athlete R on the evening of 20 September, and to what extent was he dehydrated at the time the sample was taken at 0830 on 22 September 2012?

(2) Would that amount of alcohol, and that state of dehydration, account for the abnormal values obtained from the sample both in respect of Hb and %RET?

The Anti- Doping Rules

8. Athlete R is a professional rider bound by the anti-doping rules of the UCI which include Article 21.2 which prohibits use or attempted use of a Prohibited Substance or Prohibited Method. That rule provides:

> "It is each Rider's personal duty to ensure that no Prohibited Substance enters his or her body and that he does not use any prohibited Method. Accordingly, it is not necessary that intent, fault, negligence or knowing Use on the Rider's part be demonstrated in order to establish an anti-doping rule violation for Use of a Prohibited Substance or a Prohibited Method."

9. The 2012 WADA Prohibited List at S2 includes as substances which are prohibited Erythropoiesis Stimulating Agents, including erythropoietin (EPO) and other substances with similar chemical structure or similar biological effect, and at M1 as methods which are prohibited all forms of blood doping, including autologous or homologous blood transfusion.

10. Under Article 22 the burden of proof lies on UKAD to prove the charge to the "comfortable satisfaction of the hearing panel bearing in mind the seriousness of the allegation which is made". That formula, taken from the WADA Code, requires more than a balance of probability, but less than proof beyond a reasonable doubt. The CAS in *Pechstein v ISU* CAS 2009/1912 rejected the argument that in a serious case the tribunal should apply the criminal law standard of proof beyond a reasonable doubt.

11. Under Article 23 facts relating to anti-doping rule violations may be established by any reliable means. The WADA Code commentary (at articles 2.2 and 3.2) states that a contravention may be established by reliable means, other than an adverse analytical finding, such as conclusions drawn from longitudinal profiling or profiling of a series of the athlete's blood or urine samples. The principle of treating the longitudinal profile in the athlete's biological passport as a reliable means of proof of contravention was explicitly accepted in *UCI v Valjavec* CAS 2010/A/2235 at paragraph 7 and in *De Bonis v CONI* CAS 2010/A/2174 at paragraphs 9.6 to 9.8.

12. Charges based on abnormalities detected under an ABP programme are fundamentally different from cases based on direct evidence from an adverse analytical finding. An adverse analytical finding is, in general, an objective fact,

whereas the conclusions to be drawn from deviations from a longitudinal profile require scientific judgement as to the significance of observed abnormalities. That is why the WADA Operating Guidelines require that each stage following the detection by the model of an atypical value should be the subject of expert review. A single expert reviews the atypical value against the passport to decide whether the abnormality is unlikely to be the result of a normal physiological condition or a pathological condition. A panel of three experts is then required to consider whether it can reach a unanimous opinion that it is highly likely that a prohibited substance or method has been used. The athlete is then asked for his explanation, following which the panel of three experts is required to consider whether it remains of the unanimous opinion, taking into account the explanation from the athlete, that it is highly likely that the athlete used a prohibited substance or method. So proof of an anti-doping contravention in ABP cases depends critically on expert evidence.

13. In *UCI v Valjavec* CAS 2010/A/2235 it was argued by the UCI that given the lack of scientific expertise of the Court of Arbitration for Sport panel it should confine itself to checking whether the expert panel had considered the correct issues and reached its decision in a manner which was not apparently arbitrary or illogical, and should not substitute the panel's subjective interpretation for that of the experts. That surprising submission, which appears implicitly to rule out the ability of the athlete to adduce expert evidence in his own defence, was soundly and properly rejected at paragraph 79.

14. In this case the NADP tribunal, which has been constituted so that it does possess both scientific and medical expertise, has ensured that the expert evidence submitted by UKAD is subject to full and critical scrutiny, as set out below. The role of the tribunal is to determine whether, on the basis of all the expert evidence adduced both by UKAD and by the defence, it is satisfied, to the required standard, that UKAD has proved that the results derived from the ABP programme demonstrate that a doping contravention was committed. The athlete does not have to prove that his explanation for the abnormalities disclosed in the sample is more likely to be the true explanation, for the burden of proof rests entirely on UKAD to disprove that explanation.

15. In this case, as noted above, it is not disputed that, absent explanation, the results of the sample taken on 22 September 2012, compared to the longitudinal profile shown in the passport, do provide compelling evidence of a contravention. The expert evidence has been directed solely to the plausibility of the explanation advanced on behalf of the rider.

Procedural History

16. By letter dated 18 September 2013 the UCI notified Athlete R that a panel of three experts had given a unanimous opinion that that it was highly likely, absent an explanation, that he had used a prohibited substance or method, and he was invited to submit an explanation. By letter dated 16 October 2013 solicitors acting for Athlete R gave that explanation and denied that he had ever taken or used a prohibited substance or method. That explanation was submitted to the expert panel which on 14 November 2013 gave its unanimous opinion that the explanation given on

behalf of the rider did not explain the variations observed in his profile, and that it was highly likely that the rider had used a prohibited substance or method. By letter dated 9 December 2013 the UCI notified the rider of that opinion and of the intention to commence disciplinary proceedings.

17. These proceedings were commenced by the letter dated 23 December 2013 from UKAD acting for the British Cycling Federation.

18. Procedural directions were made on 3 February 2014. Those directions were later varied when the substantive hearing was postponed to 1 July.

19. Factual witness evidence was produced in the form of statements from Athlete R and Witness A, who supported his evidence as to the circumstances of the evening of 20 September 2012, and Witness B, who acted as Athlete R's manager in 2012 and 2013. There was a number of statements as to character, including a statement from Witness C, the former manager of the Racing Team A. At the hearing both Athlete R and Witness C gave evidence.

20. The expert evidence for the rider consisted of reports from Expert A and Expert B. At the hearing only Expert A gave evidence, and in submissions no reliance was placed on the evidence of Expert B, which had been principally directed to the validity of the conclusion reached by the expert panel in its decision dated 14 November 2013.

21. The expert evidence adduced by UKAD consisted of joint reports by Expert D and Expert E, both of whom gave evidence at the hearing. There was also a report by Expert F, dealing with the statistical basis on which the ABP is constructed.

22. On 18 April the rider submitted a brief disputing that the tribunal could be comfortably satisfied on the evidence that the most likely explanation of the results derived from the blood sample taken on 22 September 2012 was the use of a prohibited substance or method.. It was submitted that the results could be explained by an acute physiological insult due to binge drinking. On 13 May UKAD submitted a lengthy brief in answer, which dealt exhaustively with the relevant rules, the issues, the facts and the expert evidence. The parties then submitted short skeleton arguments before the hearing which reiterated in summary form the arguments already advanced.

Facts

23. Athlete R is aged [redacted]. He started mountain bike racing when he was 15. In 2004 he was selected for the British U23 national team. In 2005 he was diagnosed as having infectious mononucleosis and took a few years out of the sport.

24. In 2011 he rode for Racing Team B, finishing fifth in the general classification in the Tour of Britain. In 2012 he rode for Racing Team A, winning the Tour Méditerranéen and the Tour du Haut Var. In July 2012 he won the Tour Alsace. Between 22 August and 2 September he spent 11 days training at altitude with Racing Team A in Catalunya.

25. He returned to the UK on 3 September. He then raced in the Tour of Britain between 9 and 16 September, taking the

yellow jersey on the fourth day, and finishing as the overall winner. Between 14 and 16 September he was subject to three in competition urine tests, and in each case the samples tested proved negative for any prohibited substance. However none of these samples were screened for EPO.

26. During 2012 Athlete R had been approached to join Team Sky, and there had been a number of discussions about a remuneration package. On 20 September his agent negotiated a two year remuneration package with bonuses, substantially in excess of an increased offer which had been made by Endura Racing following his winning the Tour of Britain. This offer was described by Athlete R as "incredible" and these were "by far the most successful few days of my career". In early September he had been selected to race for the Great Britain national team in the UCI Road World Championships on 23 September in Maastricht.

27. His evidence is that on the evening of 20 September he decided to celebrate with his girlfriend. Over dinner they had two bottles of wine, most of which were drunk by him. After dinner they went out in Bristol and visited several bars, where he drank heavily, both wine and spirits. He is unable to recall precisely what he drank but it included 6 or 7 double measures of gin before moving on to vodka. His evidence is that he does not drink often, but when he does he tends to "binge drink". His normal off season binge drinking would include a full bottle of spirits followed by further drinks in bars.

28. He says he woke the following morning with a hangover. He had not vomited during the night. He took aspirin and paracetamol throughout the day, which he spent in bed until he had to leave to catch a plane to Maastricht. He felt sick during the flight, but does not speak of vomiting. He did not disclose to his teammates that he was sick or had a hangover as he knew this would not meet with a favourable reaction. During the day he did not eat or drink, save for a few drops of water used to take the tablets.

29. His evidence is corroborated to an extent by evidence from his girlfriend. They had something to celebrate both in his cycling success and in her career. He did drink about 1½ bottles of wine before they walked out into Clifton to visit bars. They drank wine followed by vodka, but her memory is hazy. It appears they walked back home. She left him at 7am the following morning when he appeared not to be feeling great, but was not ill, so she left him without concern.

30. That evidence is interpreted by Dr. Hampton in his expert report as leading to the conclusion that Athlete R had a total alcohol intake in the evening of 335 grams, that is over 33 units of alcohol. This level of alcohol intake he describes as not social or normal drinking but binge drinking leading to an acute severe toxic insult to the physiological system, causing a "desperately abnormal" effect on the reticulocyte production. He does not dispute that this assumed level of alcohol intake would cause the blood alcohol levels to be raised for 60 hours and views this level of drinking as unusual in a professional athlete.

31. At 0830 on 22 September 2012 Athlete R gave a blood sample, the first sample to be taken from him under the ABP programme. On 23 September he raced in the UCI World Racing Championship, a 260 kilometre event, and finished in 19th place, the leading British rider. On 24 September, at the request of Team Sky, he gave a further blood sample which was analysed at Central Manchester University Hospital. That analysis was not carried out under the ABP programme, nor in accordance with WADA protocols, but there is no reason to question its accuracy, subject to the

accepted variability which may be expected from analysis derived from different technical processes.

32. Expert A is not the only one to view the level of alcohol intake asserted by Athlete R as unusual for a professional athlete. In the particular circumstances of this rider, at this point in his career, his conduct in embarking on a binge drinking session appears very surprising. He had just achieved the most important victory in his life and landed a highly remunerative contract with Team Sky. This was not a "normal off season binge drinking" session, as he had been selected to race for the national team three days later in Maastricht. This was an honour for him and it was a long distance 260km road race in which he would be keen to impress his team. His explanation is that he did not set out that evening with the aim of drinking to excess, he did not consider that drinking alcohol would affect his racing performance three days later, but once he had had a few drinks he got carried away. Surprising as it may seem the evidence given by Witness C, the former manager of Racing Team A, is that some top riders do on occasion, even during the season, drink very heavily.

33. The hypothesis advanced in the evidence of Expert A is dependent upon two factual premises. The first is that the rider imbibed 335 grams of alcohol over six hours. The evidence from Athlete R and his girlfriend cannot be specific as to the amount of alcohol consumed, but if that evidence is accepted it is not inconceivable that it did amount to the volume assumed by Expert A. The second is that the rider suffered severe dehydration the following day by vomiting and not drinking any water. However the evidence of Athlete R did not state that he vomited during the night or the following day, nor does his girlfriend suggest that he was ill during the night or when she left him in the morning. He clearly cannot have exhibited signs of illness in front of his teammates on the plane or at the hotel. Why a professional athlete suffering from a hangover and dehydration should not have drunk any amount of water over 32 hours is not easy to explain. His evidence was that he was well aware of the importance of keeping hydrated, but he says he felt unwell and feared that if he took water it would cause him to vomit. Yet on the morning of 23 September he says he felt back to normal, despite not having drunk any water the previous day, and was then able to train with the team for over three hours. His physical state on 23 September does not appear consistent with a state of severe dehydration.

34. We have considerable reservations as to this evidence. We are unable to dismiss as implausible the evidence that Athlete R did in fact imbibe a substantial amount of alcohol during the evening of 20 September. However we do not accept the evidence that he was in a state of severe dehydration when he gave the blood sample at 0830 on 22 September. It is inconceivable that a professional rider, selected for the first time to ride for his country at a senior level in the world championships, would not have ensured that by the time he arrived in the team hotel at Maastricht he was fit to race and had ensured that he had taken on sufficient water to deal with any hangover which he was still experiencing.

The expert evidence

35. At the suggestion of the parties the principal expert evidence, from Expert E and Expert D for UKAD and Expert A for

Athlete R, was taken together. That evidence consisted of a short presentation from each side on each of the issues, followed by a period of questioning and some argument. That process was useful to enable the tribunal to gain an understanding of the differences between the experts and to give an indication of the strengths and weaknesses in the respective arguments. However the issues were very fully explained in the written reports and we propose to base our findings mainly on the considered views expressed in writing in those reports.

36. Expert E and Expert D are clearly experts of considerable distinction and unparalleled knowledge in the field of anti-doping, in particular relating to the ABP programme. Expert E is a member of the medical commission of the UCI. Expert D was closely involved in the development of the ABP programme. Although their independence was not questioned in the hearing it should be borne in mind that Expert E was one of the three experts who compiled the report dated 14 November 2013 which rejected the athlete's explanation for the abnormal variations in his profile.

37. Expert A is a senior lecturer in cardiovascular medicine at the University of Sheffield, and consultant haematologist in the Sheffield Teaching Hospital Foundation Trust. He is clearly a very experienced and knowledgeable haematologist, but he does not have the depth of experience or expertise in the field of blood doping possessed by the experts deployed against him.

38. The thesis advanced by Expert A in his two reports, and as further explained in his evidence, is as follows:

(1) He accepts that the sample taken on 22 September 2012 exhibits a very high and abnormal Hb concentration, and a very low and abnormal %RET;

(2) The use of a prohibited substance or method, such as EPO or autologous transfusion, could have given rise to the abnormalities detected;

(3) An acute event constituted by the consumption of excess alcohol, which he computes at 335g, followed by a period of dehydration, during which the subject took no water, would explain the abnormalities exhibited by the sample;

(4) "For this (explanation) to be true I would have to show that the haemoglobin can be explained by a reduction in the plasma volume rather than an increase in the red call mass and also that there was transient suppression of reticulocytes from the bone marrow. Clearly if there was a prolonged suppression of reticulocytes in the bone marrow, this would be associated with a low, rather than a high, haemoglobin";

(5) The ingestion of 335 g of alcohol over 6 hours is likely to have had a significant effect on free water exclusion and on plasma volume; this is supported by a paper by Rubini (1954) which shows a 4.3% change in plasma volume of patients, who were chronic alcoholics, treated with moderate quantities of alcohol; a dose of alcohol 7 times as great would have had a greater effect on plasma volume;

(6) The acute reduction in the reticulocyte count was the direct result of alcohol toxicity on the bone marrow that resulted in an acute suppression of reticulocytes, an effect which was transient; the mechanism suggested is that alcohol has a direct inhibitory effect on haemoglobin synthesis, and the signal for release of the cytoplasm into the circulation "seems to be" that cytoplasm is adequately haemoglobinised.

39. In response to Expert A's first report Expert E and Expert D prepared a detailed report dated 13 May 2014, exhibiting a number of scientific papers, rebutting the thesis advanced by Expert A. The main points made in that report were:

(1) As to the haemoglobin value:

(a) The reliance by Expert A on changes in plasma volume due to physical exercise to support a contention that alcohol could have the same effect is misplaced, because the mechanisms are different;

(b) There is no scientific evidence for a plasma volume reduction after alcohol consumption; the scientific consensus as evidenced by the general review of the literature by Beard & Knott (1971) is that alcohol consumption does not cause a loss of plasma volume but may even elevate it; the Rubini paper has been misinterpreted as the results actually show a 4% increase in plasma volume;

(c) If alcohol induced dehydration had been a significant factor then that would be expected to have reduced the mean cell volume (MCV) of the red cells, whereas the analysis showed that MCV was well within normal parameters;

(2) As to the %RET value:

(a) Alcohol affects the bone marrow by acting on progenitor cells and immature erythroblasts, ie. reticulocytes in the course of formation; haemoglobinisation is not a trigger for reticulocyte release;

(b) There is no scientific evidence that acute alcohol intoxication has any effect on reticulocytes in healthy subjects;

(c) Even the most extreme damage to bone marrow cells, by myelosuppressive chemotherapy, causes only a gradual decrease of reticulocytes over 7 – 10 days, not an immediate severe reduction in reticulocyte levels as implied by Expert A's thesis.

40. It is notable that in a second report dated 2 June 2014 Expert A elected not to seek to engage with any of these detailed arguments, but instead produced a generalised paper asserting the toxicity of alcohol, that alcohol causes dehydration and that high haemoglobin could be due to a reduced plasma volume with a normal red cell mass. None of these points are in themselves controversial, but they do not explain by what mechanism alcohol is suggested to have had the effect both of raising haemoglobin at the same time as reducing the level of reticulocytes to a level which Expert Adescribed as "wildly abnormal". It is not sufficient to assume that because alcohol does have an effect on the blood it can affect the concentration of haemoglobin or the level of reticulocytes.

41. It is right to note that there is no scientific paper produced which explains the effect on the blood of healthy fit adults of an acute absorption of alcohol at the levels assumed by Dr. Hampton. That does not give any support to his thesis, but on the other hand it does require that Professors Schumacher and D'Onofrio produce compelling evidence, derived from study of analogous situations, which establish that the explanation advanced for the abnormal levels of Hb and %RET

cannot be established. This tribunal needs to be satisfied either that the mechanism suggested is scientifically implausible, or that the relevant scientific literature clearly provides evidence to contradict the effects suggested.

42. On the haemoglobin issue we did not find Dr. Hampton's argument at all persuasive. The presentationmade by Professor Schumacher was to the effect that alcohol does indeed inhibit the anti-diuretic hormone so that water is excreted, but electrolytes are retained, so that fluid will flow back into the circulation and protect the plasma volume. This contradicted the mechanism for loss of plasma volume assumed by Dr. Hampton, the basis of which was far from clear and not explained in his reports. In response Expert A appeared to accept the physiological analysis, but could not satisfactorily explain how this could lead to the conclusion that plasma volume could decrease. It was initially stated that a 23% loss of plasma volume could explain the level of Hb concentration found, but subsequently Expert A revised his figures to suggest that a 10% reduction in plasma volume would be sufficient. But whatever the quantification of this theory it is clear that it postulates a very substantial reduction in plasma volume. There was no satisfactory response to the point that if alcohol induced acute dehydration had been the cause of increased concentration of Hb then a decrease in MCV would also have been expected, a point supported by reference to a paper by Fehr, Galliard-Grigioni & Reinhart (2008). The fact that the sample showed no abnormality in MCV contradicts the suggestion that the rider was suffering from severe dehydration at the time the sample was taken.

43. The literature produced, both the Rubini paper and Beard & Knott review, does not give any support to the proposition that even a heavy dose of alcohol could lead to a diminution in plasma volume. Beard & Knott in dealing with cases of acute alcohol intoxication are clear that in all material reviewed by them there was an increase in plasma volume after alcohol administration. The tribunal has some reservations about reliance on the Rubini study, the subjects of which were a small number of chronic alcoholics, but the Beard & Knott review is more broadly based and concludes that there is not convincing evidence that acute intoxication results in fluid and electrolyte depletion, in the absence of vomiting and diarrhoea (which did not occur in this case). The Whitehead paper (1995) concludes that alcohol consumption did not have any consistent effect on haemoglobin concentration. Each of these papers cited contradicts the general suggestion that alcohol may reduce plasma volume.

44. On the reticulocytes issue we did not understand how the mechanism suggested by Witness A could bring about a severe and immediate reduction in the volume of reticulocytes, when it is accepted that a toxic attack on the bone marrow, by analogy with the effects of chemotherapy, would only gradually reduce reticulocytes levels over a longer period. Expert A sought, however, to contend that chemotherapy works in a different manner by inhibiting cell division, whereas alcohol has an inhibitory action on haemoglobin synthesis (see Ali and Brain, 1974), and by implication on the release of reticulocytes. However, there is no basis for the suggestion that haemoglobinisation is a trigger for reticulocyte release, a hypothesis only tentatively advanced by Expert A. Expert D gave a very clear presentation as to how the cell development of reticulocytes proceeds in line with haemoglobinisation but the trigger for release is not related to the process of haemoglobinisation. The Sullivan paper (1964), on which Expert A relied, related to a very different clinical scenario in which chronic folate deficient alcoholics were repleted with folic acid, leading to a dramatic rise in reticulocyte count, a response which was reversed over a period of 4-10 days by

concurrent alcohol administration. Expert D stated that any effect of alcohol in this situation was not in any way comparable to the putative response of the reticulocytes to alcohol, as advanced by Expert A.

45. It was argued that the result of the analysis made in Manchester on 24 September 2012 ought to have been taken into account by the experts, and that its results supported the conclusions of Expert A. On the first point, the experts are only permitted under the WADA operating guidelines to take into account results obtained from analyses conducted under the ABP programme, which comply with strict criteria. Expert A only argued in his report that the sample taken on 24 September was consistent with his thesis, in showing a recovery towards normal values. The UKAD experts observe that the reticulocyte levels are still reduced, but note that comparison may be difficult due to variations in the equipment used and that the apparent increase in reticulocyte readings is within the range of variation accepted by the guidelines. It was also submitted that the OFF score derived from the Manchester sample of 127.8 would not be abnormal. However on the basis of the report of Dr. Sottas, paragraph 3, such an OFF score would fall below a probability of 99.9999%, but would still be far outside the expected parameters. But no point derived from this sample can affect the conclusions on the central issue as to whether there is a plausible explanation for the accepted abnormalities found in the sample taken on 22 September.

46. This tribunal has reached the very clear conclusion that the expert opinions expressed by Expert E and Expert D were cogent and supported by the scientific evidence, whereas the suggestions advanced by Expert A were not persuasive as to the mechanism suggested, and not supported by the scientific papers on which he relied. We are clear that the explanation advanced for the rider does not explain the abnormal values of Hb and %RET shown in the sample taken on 22 September 2012. As Expert A accepted, in order for his hypothesis to be valid it is necessary to accept his explanation for both abnormalities. We have accepted neither, and in combination the inference to be drawn from the abnormalities in both Hb and %RET is overwhelming.

Conclusions

47. It is argued by Mr. Unsworth that this case is unprecedented in that it is the only case in which a contravention has been alleged under the ABP programme based on a single sample, and the sample relied upon is the first sample taken under the programme. That may be so, but it does not call into question either the reliability of the ABP model in general, or the validity of relying upon a single sample to prove a contravention. The purpose of establishing the ABP programme is to build a longitudinal profile providing parameters which, to a very high degree of probability, serve to detect results which are abnormal and call for explanation. Because blood doping may be transient and its effects very quickly cease to be evident in blood or urine it is essential to have a programme which can detect an isolated outlier. There is no logical difference between an abnormal value detected in the first of a series of tests, and an abnormal value detected at the end of series of tests, by which time the model will have been fully adapted. In each case the abnormality will be assessed against a reliable series, and to a very high degree of probability. In this case there is no issue that the

abnormalities shown in the sample would constitute compelling evidence of the use of a prohibited substance or method, unless explained by some other cause.

48. There was no dispute that the abnormalities in the sample were consistent with the use of an erythropoietic stimulant which had been discontinued approximately 10 to 14 days before the sample was taken. There were some further submissions from Mr. Taylor for UKAD directed to the point that the rider had both the motive and the opportunity to commit a doping offence at this stage in his career, given the importance to him of obtaining a remunerative contract with Team Sky. This tribunal declines to take those points into account. The facts of the case, as set out above, are relevant only to the issue whether the rider has produced a plausible explanation for the abnormalities in the sample. Whether that explanation is plausible must be decided on the basis of the scientific evidence alone, and cannot be influenced by circumstantial evidence as to the motive or opportunity for the rider to have used a prohibited substance or method.

49. On the basis of the expert evidence discussed above we are entirely satisfied, to the required standard of proof of comfortable satisfaction taking into account the seriousness of the case, that the explanation advanced on behalf of the rider cannot explain the abnormal values obtained from the sample taken on 20 September 2012. For the reasons set out at paragraph 34 above we have concluded that the rider was not in a state of severe dehydration at the time the sample was taken, so the assumptions made by Expert A in his report as to the dehydration of the rider were not actually substantiated by the evidence. It is the combination of the two factors, both an abnormally high Hb level and an abnormally low level of reticulocytes, neither of which can be explained, which compels the conclusion that a prohibited substance or method had been used by the rider.

Ineligibility

50. The tribunal has found the doping violation under Article 21 at 2.1 proved. This is the first anti-doping rule violation so under Article 293 a period of ineligibility of 2 years must be imposed.

51. The rider has not been subject to any effective provisional suspension, so Article 317 does not apply. Accordingly the period must start on the date of this decision, unless Article 315 is applied. There has been a substantial lapse of time since the taking of the sample, and some delay in the hearing process, not attributable to the rider, due to the complexity of the case and difficulty in arranging a hearing date which both the lawyers for the parties and the experts could attend. By a press statement issued by his management company on 17 December 2013, after the charge became public, the rider in effect suspended himself from competition and training. Taking all those factors into account the tribunal considers that it would be fair for the period of ineligibility to commence on 1 January 014.

Disqualification

52. The UCI Expert Panel concluded that the prohibited substance or method was probably used from the end of August 2012 and thus affected the rider's performance in the 2012 Tour of Britain and the 2012 UCI Road World Championships. On the basis of our finding that a prohibited substance or method had been used it must follow that those results be disqualified.

Fine and Costs

53. Under Article 326.1 (a) the rider must be fined 70% of his gross income during 2012. That fine is assessed at £15,400.

54. Under Article 275 the costs payable by the rider are assessed at CHF 2,500 for results management and €324 for laboratory documentation. The tribunal decides not to make any order for payment of the costs of these proceedings. The general principle under the NADP Procedural Rules is that the tribunal does not make any order that a party should pay these costs.

Decision

55. For the reasons given above, the tribunal makes the following decision:

(i) A doping offence contrary to Article 21.2 has been established;

(ii) Under Article 293 the period of ineligibility imposed is 2 years from 1st January 2014;

(iii) Under Article 313 the rider's competitive results in the 2012 Tour of Britain and the 2012 UCI Road World Championships are disqualified;

(iv) Under Article 326 a fine of £15,400 is imposed;

(v) Under Article 275 the rider is required to pay costs of €324 and CHF 2,500.

Right of Appeal

56. The rider has a right of appeal against this decision to the Court of Arbitration for Sport under Article 329, which must be made by statement of appeal within one month from receipt of this decision.

Charles Flint QC

Professor Peter Sever

Dr. Terry Crystal

signed on behalf of the tribunal

15 July 2014

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