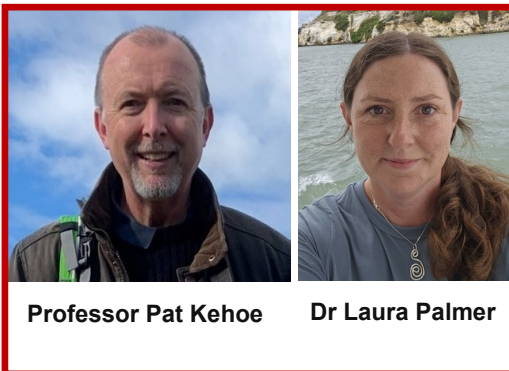


BDR has a new Directorship

This our first newsletter from the recently established BDR Lead Centre which is based at the University of Bristol, and we are delighted to announce the appointment of **Professor Patrick Kehoe** as the incoming BDR Director and **Dr Laura Palmer**, the new BDR Deputy Director.



Both Professor Kehoe (pictured far left) and Dr Palmer (left) are very familiar with the programme having led the BDR Centre in Bristol for many years. Supporting them in their new role is Nicky Barnett, former Manager at the Newcastle BDR Coordinating Centre.

During this transition period, there are no plans to make changes to the BDR programme. Instead, we want to ensure that we maintain study progress as we know how valuable the assessment data and tissue samples are to researchers. In addition, Participant and Study Partner retention and satisfaction, continue to be our primary focus, and although we currently plan to delay our next Engagement Event, we hope to schedule an event later in the year to allow us to update you on progress. As we have a new Lead Centre, we also have new contact details which are given below.



If you would like to contact the team
at the new Lead Centre, you can



Email us at: **BDR.Leadcentre@bristol.ac.uk** or Telephone: **0117 4147821**

For enquiries concerning appointments and donations,



please continue to contact your local BDR Centre

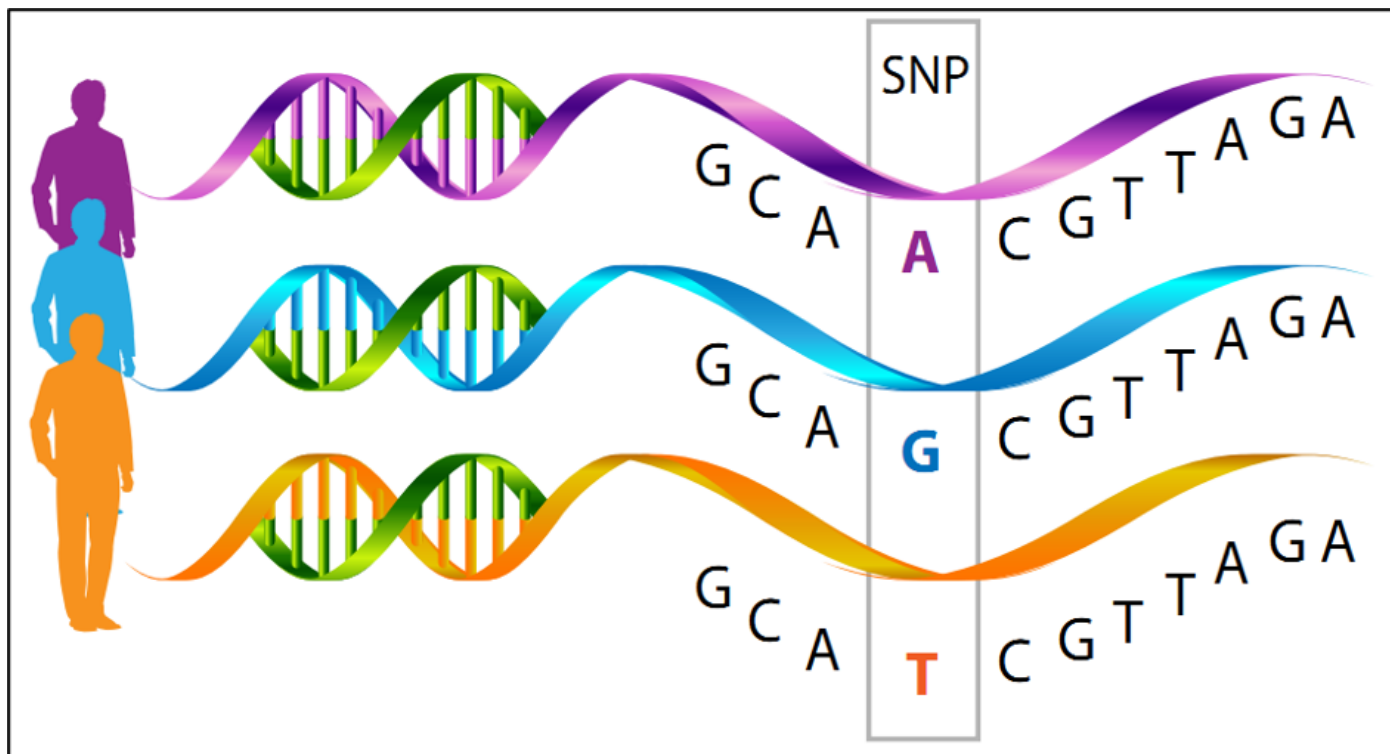


Developments in the genetic profile of BDR participants

In previous issues of the BDR newsletter, we have referenced the work of Associate Professor, Dr. Keeley Brookes at Nottingham Trent University and her significant contribution to the establishment of a BDR genetic dataset. Dr. Brookes recently announced increased genotyping on the BDR donors using a “Neurobooster” array and so we are proud to announce that we now have DNA samples ready for **genetic analysis from 1,112 of our deceased BDR participants and 313 of our living participants.**

What do we know about DNA and dementia?

Deoxyribonucleic acid, commonly known as “DNA” is the instruction manual for how to build and maintain a human being. Whilst we all have the same basic instructions, there can be small changes in them which enable us to be the unique people we are. We often refer to these differences as SNPs: **S**ingle **N**ucleotide **P**olymorphisms.



Dr Brookes has used the Neurobooster to inform us about the changes to the letters that are present in our BDR participants: around **1.8 million of the DNA possible changes have been looked at** to see if they are present in each of our samples. Having this data is crucial in helping to identify genes that may influence whether or not we develop dementia and, if we do, which type of dementia.

We now know that **version 4 of a gene known as “APOE”** is associated with dementia; we know this because we have seen the variation appear more frequently in people with dementia than in people without it in every study that has looked for this change. Because the effect of APOE 4 is so large, it is easily visible, but we also know that APOE 4 is not the only gene contributing to whether someone develops dementia. The effects of these other genes are smaller, and so are harder to see. That is why we need to look for these gene alterations in every dementia study group to identify those genes that come up consistently so that we can be as certain about these other genes as we can about APOE 4.

The BDR programme offers an extra level of information as we examine donated brains for specific pathological features that define different types of dementias. We can align the DNA changes with these specific features rather than with just a diagnosis, allowing us to investigate further the biological mechanisms that link the DNA change to the specific pathology seen. When we have identified the links between DNA alterations and pathological events, we can then look to see if we can target the changes with drugs in order to repair them. Finally, we can then use these DNA changes to identify people at risk of developing certain dementia pathologies and provide them with the right drugs before symptoms start, and thus **delay or perhaps even prevent the onset of dementia**.