Make Something Unreal Live 2013

This is the second Make Something Unreal Live game development competition, running from October 2012 and culminating at The Gadget Show Live 2013 with four teams vying for an Unreal Engine 4 (UE4) digital PC licence plus other prizes to be announced.

The competition is open to teams of six to 10 members consisting of current full-time university students. Any European student currently enrolled at a higher education institution is eligible to participate.

TEAM BRIEF

Goal

To develop a PC game demo broadly inspired by 'Mendelian inheritance' (see Attachment A, "Our Mendelian inheritance: genetics and genomics") using the Unreal Development Kit (UDK) – the free edition of Epic's award-winning Unreal Engine 3 (UE3) technology – along with scientific resources provided by the Wellcome Trust.

Key Dates

2nd October – Make Something Unreal Live 2013 launch and call for submissions

2nd November – 1st submission deadline – all project pitches must be submitted by this date

- Please register your intent to participate as soon as possible: http://www.staffs.ac.uk/gamesdesign/msul2013/
- Send pitch docs and concepts through MSUL submission form by deadline: http://www.staffs.ac.uk/gamesdesign/msul2013-submission/
- If needed, contact msul2013@staffs.ac.uk with enquiries.

7th November – Panel to review all submissions and trim to a shortlist of 12 teams

9th November – Qualifying teams to be announced; shortlist to receive extended information pack, including criteria for Presentation Day face-to-face pitches

3rd December – Presentation Day at Wellcome Trust, where all 12 teams pitch their projects to an industry panel

- At least one team member from each team must be present to pitch live
- Final four teams to be selected by end of day

Week of 17th December – Team Day 1: Meet the Scientists – All contestants to meet with Wellcome Trust experts and industry mentors

Week of 18th February – Team Day 2: Progress Review – All contestants to meet with mentors and MSUL judges, learn tips on presenting at the Gadget Show Live and demonstrate work in progress

2nd-7th April – Gadget Show Live 2013 **–** Four finalist teams to spend each day of the show improving their demos live in front of a public audience; present progress to select industry 'gurus' on the show floor for analysis and critique twice per day

- Kicks off with trade and press day on 2nd April
- Culminates in grand finale and crowning of winning team on 7th April

1st Submission contents

- The 'elevator pitch' documents should outline the high-level game concept, communicate key gameplay mechanics and scientific inspiration, and provide sample concept art.
- Team bios should include one paragraph per team member, indicating each person's skills, position on team, educational background and any special achievements.

1st Submission guidelines

- Game pitch documents should be succinct, i.e., two to three pages at the most.
- Be realistic in the scope of your concept: With barely six months of development time, it is recommended to concentrate on one or two key features.
- At this stage, the submission should not contain any code or playable demo material.
- Research and play with the theme.
    - Treat the subject matter as a starting point, using the links provided to gain a feel for the subject and where you might take it.
    - The demo should be based on the scientific principles of the subject. Feel free to extrapolate these forward or backward – these are not meant to be educational titles.
    - Think imaginatively, the theme has a rich historical as well as scientific resonance.
    - Prime examples of the use of real world science and math in fiction are the likes of Neal Stephenson's "Anathem" (see Wikipedia) and the Omni sequence in "Cloud Atlas" (see Wikipedia). It is this kind of use that we are looking for.
    - Do not go for the obvious game option. 'Humans versus mutants' will not make the cut!
- Above all, be realistic when setting scope. It is far better to implement a couple of cool features really well than to present a demo with lots of poorly executed features.

As the competition moves from stage to stage, there will be more resources and support provided from the organisers, including mentors from the games industry as well as the Wellcome Trust.

The Wellcome Trust is a global charitable foundation dedicated to achieving extraordinary improvements in human and animal health. It supports the brightest minds in biomedical research and the medical humanities. The Trust's breadth of support includes public engagement, education and the application of research to improve health. It is independent of both political and commercial interests.

MSUL 2013 is also supported by Staffordshire University, with staff providing support to the teams throughout the competition. UKIE, the games industry trade association, will continue to support the MSUL program with more details to follow.

Bearing all this in mind, keep the initial pitch simple and plan for it to evolve over the course of the competition.

Thanks and best of luck!

Kind regards from all of us,

Epic Games, Inc.

ATTACHMENT A

Our Mendelian inheritance: genetics and genomics

By Adam Rutherford

In the middle of the 19th century, an Austrian monk named Gregor Mendel crossbred around 30,000 pea plants. He was looking at how characteristics were passed from parent to offspring, and he noticed that some of them were inherited in strict patterns. In successive generations, for example, he saw three purple-flowering plants for every white-flowering one, and never a mix of the two colours.

Mendel concluded there was a discrete unit of inheritance for each colour. These units are what we call 'genes.' Each plant had two of them for flower colour: if both were 'purple', the plant was purple; if they were both 'white', it was white. But if the plant had one purple gene and one white, it would always be purple because the purple gene is dominant over the white. This also explained how crossing two purple plants could yield a white offspring, as it could inherit a recessive white gene from each parent. This happens, on average, one in four times, and only this combination produces a white flower. Mendel also worked out that genes in each plant must be separated when they produce sperm and eggs, so that the offspring only receive one from each parent.

His work was largely ignored until the beginning of the 20th century, and the birth of genetics. Genes were shown to sit on chromosomes, which are large chunks of DNA in each cell. Mendelian inheritance also provided a mechanism for evolution, which Darwin had outlined in 1859: genes are the basic unit of inheritance, and are passed down from parent to child. Evolution by natural selection requires change over generations, and the variation in genes, e.g. purple or white, showed how this was possible.

DNA, mutation and evolution

In 1953, Crick, Watson and Franklin worked out the structure of DNA – the iconic double helix – and how it encoded genes. DNA is made up of four chemical 'letters': A, C, G and T. Strings of letters constitute

genes, and these are decoded in our cells to make proteins. DNA is akin to a language, and genes contain the code to be translated into proteins: all life, every tissue, every hair or bone is made of or by proteins. Changes in the DNA of a gene will change the shape of a protein, and this can change how it behaves.

We have thousands of genes, and billions of letters of DNA. Every cell contains every gene whether it is needed in that cell or not, and so, every time a cell divides, every single piece of DNA has to be copied. This process is extremely accurate but not perfect. Sometimes, when the cell is dividing, errors can be introduced into DNA. These copy errors are called mutations. These are often harmful (like a spelling mistake that turns 'friend to 'fiend'), or neutral ('colour' to 'color'). They can be spontaneous, inherited or caused by factors such as smoking and radiation.

But occasionally they can change a protein in a way that makes the organism more successful than its peers, bringing adaptation to the changing environment. When this happens, the new version of the gene gets passed down to offspring, and will spread through the generations. Over long periods of time, accumulation of these useful mutations can transform one species into another. This is the molecular basis of evolution by natural selection.

- [Big Picture on Evolution](#)

Genetic mutation and disease

While genetic mutation is the basis of evolution, it can also cause disease. The first inherited diseases to be understood in terms of their genes were single-gene disorders like cystic fibrosis and Duchenne muscular dystrophy, as they demonstrate exactly the same clear pattern of inheritance as Mendel's peas. If the disease-causing mutation is recessive, then a couple might both be carrying one copy of the mutant version of the gene. If they have children, there is a one in four chance that the child inherits two mutant versions of the same gene and develops the condition, just as there was a one in four chance that Mendel's peas would be white.

Mutations reveal what healthy genes do

In biology, we often find out how things work only when they go wrong. Studying diseases helps us understand how our bodies work as much as studying 'normal' biology equips us to diagnose and treat diseases. The same is true in genetics, a science which started in Mendel's pea plants and today is one of the most exciting areas in all of science. All cancers are caused by genes acquiring DNA mutations that instruct cells to reproduce more and more and form tumours. We understand what many of these genes do in health by working out how they cause disease when they are defective.

The human genome

- [Landmarks in human genetics](#)

In the 1990s, geneticists decided that looking at one gene at a time was going to make slow progress. So a huge international consortium decided to read the entire human genome and account for every single

gene. This was probably the grandest project in biology's history, and in 2001 the first complete sequence of our 3 billion letters of code was published. There were two major surprises: first, we only have 23,000 genes (about the same as a mouse, fewer than a banana); and second, 97 per cent of the genome was not genes. It was DNA, but not code to make proteins.

So it turned out that although Mendel's laws of inheritance are categorically correct, most of our genes work in ways which are much more complex than pea flower colours, or even cystic fibrosis. We appear to have too few genes to account for all our complexity. Most of our genes are involved in networks of activity, performing many roles, or different roles depending on when and where they are needed and how they react to the environment. Sometimes people talk about 'nature versus nurture', but the truth is that we are the product of our biology (nature) expressed in the context of our environment (nurture). So a better phrase would be 'nature *via* nurture.'

It seems that much of the 97 per cent of the genome contains instructions for when and where genes should be active. This is a recent discovery, and part of the ongoing journey to understand human genetics. Our genomes are immensely complicated, and we will be exploring them for decades to come.

- The basics: YourGenome.org
  Types of mutation: Human Genome website
  Rates of human mutation: Wellcome Trust news story

- Big Picture on Genes, Genomes and Health
  List of genetic disorders on Wikipedia
  Wellcome Trust blog post on a recent study of cancer genetics
  Blog post on bacterial evolution in cystic fibrosis
  The man who could not walk backwards: A single genetic mutation causes Neuroferritinopathy

Most of our DNA is held in the nucleus of the cell, but a small percentage is in our mitochondria, small units of cellular machinery outside the nucleus that provide energy for the cell to function. Mutations in mitochondrial DNA can cause a wide range of disorders and disease.

- Feature on mitochondrial inherited disease

Gene therapy alters a patient's genes to treat their illness. The most common form replaces a mutated gene with a working version of the same gene. It is no mean feat; however, gene therapy has been successful used for certain conditions in recent years.

- Novel gene therapy for type of blindness

Race against resistance

Drugs used to treat infections exploit weaknesses in the properties of bacteria, viruses and parasites. However, bacteria and viruses especially have high rates of reproduction and mutation in their genomes, which means they can relatively quickly adapt and develop different properties that render our drugs ineffective. In short, they evolve.

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- [Patterns of bacterial adaptation](#)
  [Big Picture on Influenza](#)
  [Emergence of artemisinin resistance in malaria](#)

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