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Supplementary reading for biologists

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Учебное пособие “Supplementary reading for biologists” предназначено для студентов первого и второго курсов направления «Биология» Казанского Федерального Университета для занятия на уроках английского языка. Уровень сложности изучаемого материала может быть определен как Intermediate.

Учебное пособие решает важные учебно-методические задачи и дополняет комплекс учебников английского языка. Данное учебное пособие может помочь студентам совершить первый шаг к овладению английским в профессиональной сфере, в сфере биологии. Пособие содержит большое количество биологической терминологии, которая будет полезна студентам в их учебной и профессиональной деятельности.

Пособие состоит из 12 уроков, а также текстов для дополнительного чтения. Каждый урок посвящен определенной теме и состоит из текста на биологическую тематику, упражнений на отработку лексики текста, на понимание текста, а также упражнения на развитие навыков говорения. В некоторых уроках содержатся задания на поиск дополнительной информации, что должно стимулировать познавательную активность студентов.

В каждом уроке отдельным списком вынесены биологические термины, встречающиеся в уроках.

Данное пособие является актуальным, содержит только самые передовые научные данные и часто употребляемую лексику. Данное пособие поможет студентам овладеть словарным запасом, необходимым для чтения специальной литературы на английском языке и устного обсуждения представленных тем.

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The Content

Text 1. Snakebite Antivenom Development

Doctors Without Borders now describes snakebites as "one of the world's most neglected public health emergencies"

Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them. Then quickly scan the text to check your answers.**

1. *What is venom?*
2. *What do you know about snakebites?*
3. *Do you know, in what countries people most often meet snakes?*

■ **Read the given text and make your essential assignments:**

Modern medicine can grow kidneys from scratch, halt the spread of infectious diseases such as Ebola and diagnose the cause of a cough with a smartphone, yet snakebites still thwart us. Every year venom from snakes kills nearly 200,000 people and leaves hundreds of thousands disfigured or disabled, making these legless squamates the second deadliest animal. Only mosquitoes may kill more people every year (by spreading the protozoa that cause malaria).

Venomous snakes recently slithered their way back into the news when it came to light that leaders in the pharmaceutical world had ceased developing antidotes. French drug company Sanofi Pasteur, for example, made headlines in September, when Doctors Without Borders pointed out that the final batch of FAV-Afrique—the only antivenom proved to effectively treat snakebite victims in sub-Saharan Africa—expires in June 2016. Sanofi, its sole manufacturer, had ended production in 2014 because the drug was not making enough money. Others in the industry had already made similar moves, including Behringwerke and Wyeth Pharmaceuticals (now part of Pfizer).

The treatment situation has become so dire that Doctors Without Borders now describes snakebites as "one of the world's most neglected public health emergencies." And in October dozens of experts at the 18th World Congress of the International Society on Toxinology in Oxford, England, called for the World Health Organization to relist snakebite as a neglected tropical disease. Most bites occur in Africa and Southeast Asia.

Antivenom development is stuck in the 19th century because the field is underfunded, says David Williams, a clinical toxinologist and herpetologist who heads the Australian Venom Research Unit at the University of Melbourne and is also CEO of the Australian nonprofit Global Snakebite Initiative. To isolate compounds for treatment, researchers typically inject subtoxic levels of venom into animals, collect the antibodies formed by the immune response and purify the result. Antivenom must be tailored to an array of toxins across different regional snake species. No universal antidote exists.

Despite constraints, small research groups around the world are quietly working away at new, exciting solutions—waiting for a windfall of money and momentum. The most innovative of them is a targeted antivenom designed for sub-Saharan Africa that could serve as a blueprint for making cheaper compounds to counter bites from snakes found in other regions. Researchers from the U.K., Costa Rica and Spain started with proven "base antivenom" for three snakes and have begun screening it against toxins from additional snakes. Venom proteins that fail to bind to the base antivenom are screened for toxicity; only proteins identified as dangerous toxins become part of the immunizing mixture used to make the next antivenom batch more effective.

Such selective screening and iterative testing of specific proteins make for a stronger, targeted antidote compared with conventional antivenoms, which indiscriminately neutralize both toxic and nontoxic venom proteins. The group also plans to cut costs with a method pioneered in Costa Rica that requires fewer manufacturing steps. "Our goal is to make a product for sub-Saharan Africa that is cheaper or as cheap as $35 a vial," says Robert Harrison, head of the Alistair Reid Venom Research Unit at the Liverpool School of Tropical Medicine in England. Sanofi's product costs $150 per vial.

Other animals—and bacteria—may provide alternative antivenom. An opossum protein first identified in the 1990s has since been shown to protect mice from snake toxins that can cause widespread internal bleeding. Moreover, the protein neutralized hemorrhagic toxins from venomous snakes in both the U.S. and Pakistan. The finding suggests that the protein might possibly defend against all hemorrhagic snake toxins, says Claire Komives, a chemical engineer at San José State University. Komives has already demonstrated that she can engineer *Escherichia coli* bacteria to make the protein—which could reduce the cost of treatment to around $10 a dose. "I'm trying to make it in bacteria because we can scale [up production] cheaply," she says.

Groups elsewhere have turned away from traditional antidote development altogether. Matthew Lewin, director of the Center for Exploration and Travel Health at the California Academy of Sciences, has begun screening existing fda-

approved drugs for chemical ingredients that could form the basis of an injection or pill that stabilizes people bitten in the field or at least gives them time to reach a hospital. “If you had a pharmaceutical antidote, you could have it on your person,” Lewin says. Many snakebite deaths happen when victims cannot reach hospitals or clinics to receive an intravenous antivenom treatment.

Similarly, Sakthivel Vaiyapuri, a pharmacology researcher at the University of Reading in England, is screening for molecules that block the effects of snake venom. He also hopes to eventually develop a cocktail of chemical inhibitors that could lead to a universal antidote.

Modernized antivenom treatments would represent a solid first step toward reducing deaths from snakebites. Yet in the end, the best treatments in the world will fail without funding and distribution. “If the ministries of health responsible for health and well-being don't prioritize snakebite treatment,” says Williams of the Global Snakebite Initiative, “you're banging your head against a brick wall.”

Scientific American December 1, 2015

http://www.scientificamerican.com/article/snakebite-antivenom-development-is-stuck-in-the-19th-century-what-s-next/

■ Glossary of essential terms

№	English term	Russian equivalent
1.	snakebite	укус змеи
2.	venom	яд
3.	squamates	чешуйчатые
4.	protozoa	простейшие
5.	Doctors Without Borders	«Врачи без границ» (международная независимая гуманитарная медицинская организация)
6.	batch	партия
7.	kidney	почки
8.	scratch	ноль
9.	thwart	сорвать
10.	cough	кашель
11.	slither	уползать
12.	dire	остро
13.	underfunded	недофинансированный
14.	tailor	подогнать, приспособлять, адаптировать
15.	despite	несмотря
16.	widespread	широко распространенный
17.	hemorrhagic	геморрагический

18.	scale	масштаб
19.	pill	таблетка
20.	approved	утвержден
21.	bang	стучать
22.	halt	остановить
23.	victim	жертва
24.	expire	терять силу, истекать, заканчиваться
25.	treatment	лечение
26.	neglect	пренебречь, забросить
27.	toxinologist	токсиколог
28.	herpetologist	герпетолог
29.	nonprofit	некоммерческий
30.	antibodies	антитела
31.	target	цель, намечать
32.	blueprint	модель, шаблон
33.	mixture	смесь
34.	iterative	повторяющийся
35.	vial	флакон, пузырёк, ампула
36.	hemorrhagic	геморрагический
37.	FDA-approved Food and Drug Administration	Одобренный управлением по санитарному надзору за качеством пищевых продуктов и медикаментов
38.	brick	кирпичный
39.	rattlesnake	гремучая змея
40.	cords	связки
41.	recurved hooks	загнутые назад крючки

■ Your Essential Assignments

I. Quick check

1. Why did many pharmaceutical companies stop production of antidotes?
2. Briefly describe new tendencies in antidotes researches.
3. How are researchers going to reduce the price of the antidote?

II. Fill in the missing words:

Term (verb)	Noun	Adjective
exist	.....	.....
develop	.....	.....

demonstrate	.....	.....
neglected	.....	.....
select	.....	.....
produce	.....	.....
isolate	…..	……

III. Use monolingual English dictionary and write down what could the words given below mean:

nonprofit; antibodies; subtoxic; reduce; widespread.

IV. Find English equivalents to the following word combinations:

№	Russian term	English equivalent
1.	змеиные укусы всё ещё беспокоят нас	
2.	безногие чешуйчатые	
3.	прекратили разработку противоядий	
4.	единственный производитель	
5.	выделить (изолировать) соединение для лечения	
6.	иммунный ответ	
7.	несмотря на ограничения	
8.	без разбора нейтрализуют как токсичные, так и нетоксичные белки	
9.	животные и бактерии могут вырабатывать альтернативное противоядие	
10.	защищает от всех токсинов	
11.	внутривенное введение противоядия	
12.	сходным образом (так же)	
13.	коктейль из химических ингибиторов	

V. Give Russian equivalents to the following English phrases:

№	English phrase	Russian equivalent
1.	leaves hundreds of thousands disfigured or disabled	
2.	Malaria, which is transmitted by mosquitoes, kills the most people each year.	
3.	Venomous snakes recently slithered their way back into the news.	
4.	This is one of the world's neglected emergencies.	

5.	to counter bites	
6.	Antivenom must be tailored to an array of toxins across different regional snake species.	
7.	The researchers have already demonstrated that bacteria with altered genome can produce an antidote.	
8.	waiting for a windfall of money and momentum	
9.	that requires fewer manufacturing steps	
10.	is screening for molecules that block the effects of snake venom	
11.	The best treatments in the world will fail without funding and distribution.	

VI. Find synonyms among the pool of words:

Pool of words	Synonyms
1) 1.slither/2. explore/3.research/4. slip/5.glide	
2)1.innovative/2.introduce/3.advanced/4.progressive/5.inject/ 6.input	
3)1.characteristically /2.effective/3. acting/4. typically	
4)1.manufactur/2.identifiy/3.produce/4.fabricate/ 5.recognize	

VII. Answer the following questions. Use all information given before:

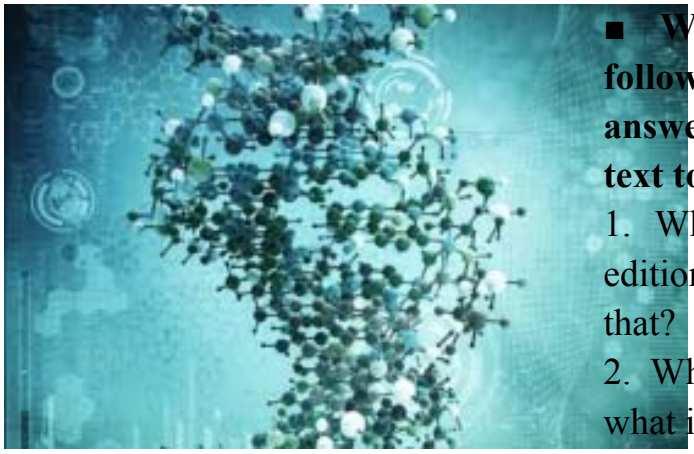
1. Can scientists introduce a universal antidote?
2. What animal kills the most people every year?
3. What is screening?
4. What do you think, why the sale of antidotes are not profitable for the pharmaceutical companies?
5. Do enough African countries receive vials of antidote against snake venom?
6. Why researchers want to use the modified bacterial gene for production of antivenoms?
7. Is it true that most people die, because of a lack of a vaccine in the hospitals?
8. In what countries do small groups of scientists work on development of innovative antidote?
9. Doctors without Borders tried to draw attention to the problem of snake bites. Why do they do it?

VIII. Match the sentence halves. Make complete sentences:

1.	Every year venom from snakes kills	**A.**	"one of the world's most neglected public health emergencies."
2.	Sanofi, its sole manufacturer	**B.**	nearly 200,000 people and leaves hundreds of thousands disfigured or disabled
3.	Doctors Without Borders now describes snakebites as	**C.**	collect the antibodies formed by the immune response and purify the result.
4.	Researchers typically inject subtoxic levels of venom into animals	**D.**	had ended production in 2014 because the drug was not making enough money.
5.	The most innovative of them is a targeted antivenom	**E.**	the protein might possibly defend against all hemorrhagic snake toxins
6.	The group also plans to cut costs with a method pioneered in Costa Rica	**F.**	that requires fewer manufacturing steps.
7.	The finding suggests that	**G.**	you could have it on your person.
8.	If you had a pharmaceutical antidote	**H.**	designed for sub-Saharan Africa.

Text 2. "Improving" Humans with Customized Genes Sparks Debate among Scientists"

Pre-reading



■ **Work in pairs. Discuss the following questions and try to answer them. Then quickly scan the text to check your answers.**

1. What do you know about gene edition? What do you think about that?
2. What is CRISPR? Do you know what is it?
3. What genetic diseases do you know?

Exercise A. Match the words with their definitions:

1.	gene	**A**	is the genetic material of an organism, it consists of DNA (or RNA in RNA viruses), includes both the genes and the non-coding sequences of the DNA/RNA.
2.	heredity	**B**	is the passing of phenotypic traits from parents to their offspring, either through asexual reproduction or sexual reproduction.
3.	mutation	**C**	is the product of reproduction of a new organism produced by one or, in the case of sexual reproduction, two parents.
4.	genome	**D**	are macromolecular biological catalysts.
5.	genomic mutation	**E**	is a molecule that carries most of the genetic instructions used in the development, functioning and reproduction of all known living organisms and many viruses.
6.	offspring	**F**	is a particular abnormal condition, a disorder of a structure or function, that affects part or all of an organism.
7.	enzyme	**G**	is a nucleic acid sequence on double-stranded DNA or RNA wherein reading 5' (five-prime) to 3' (three prime) forward on one strand matches the sequence reading backward 5' to 3' on the complementary strand with which it forms a double helix.
8.	DNA	**H**	is a packaged and organized structure containing most of the DNA of a living organism.
9.	RNA	**I**	changes in the number of chromosomes.
10.	chromosome	**K**	is a polymeric molecule implicated in various biological roles in coding, decoding, regulation, and expression of genes.
11.	disease	**L**	is a permanent alteration of the nucleotide sequence of the genome of an organism, virus, or extrachromosomal DNA or other genetic elements.
12.	palindromic sequence	**M**	is a locus (or region) of DNA that encodes a functional RNA or protein product, and is the molecular unit of heredity.

■ **Discuss these questions with your partner and your class.**

1. Do you know what eugenics is?
2. Do you know anything about the Convention on Biomedicine and Human Rights?
3. There is a positive and negative eugenics. What do you think about each of them?

■ **Read the given text and make your essential assignments:**

"Today we sense we are close to be being able to alter human heredity," Nobel Laureate and California Institute of Technology virologist David Baltimore said December 1 at the opening of a much-anticipated human gene editing summit taking place in Washington, D.C. this week. Gene editing, or tweaking the human genome with additions, subtractions or alterations, is becoming increasingly realistic with modern technologies. "When will we be prepared to say we are justified to use gene editing for human enhancement purposes?" he asked.

Part of the problem for researchers, doctors and ethicists alike is defining "enhancement" and deciding if it would be a move in the right direction, as the word would suggest. Is enhancement merely referring to boosting muscle tone and other desirable traits like achieving perfect pitch or does the term also encompass steps to guarantee better health by preventing disease? Some scientists disagreed over whether certain types of gene-editing would be important for helping patients, with one prominent researcher contending the technology would not often be needed, while another described dire current clinical needs for it. The international summit on gene editing, sponsored by Britain's Royal Society, the Chinese Academy of Sciences, and the US National Academies, has grappled with such thorny questions during three days of sometimes-heated discussion on editing the human genome that comes to a close today.

Baltimore, who is chairing the summit, believes that when it comes to drawing the line for enhancement, the determining factor is whether the change would be optional versus therapeutic. "To my mind enhancement is really optional," he says. "You are not solving a life-threatening issue." Making a change to the PCSK9 gene, for example, would lower the risk of cardiovascular disease and for someone with high LDL—the bad kind of cholesterol – it could be the difference between life and death, he says. In that case the intervention would be therapeutic. Whether or not to engage in future "improvements" that are considered more optional, however, remains murky.

Take the DEC2 gene. Tweaking it could make a person function like the rare individuals who are born with a variant that allows them to function well with just a few hours of sleep. That trait is not necessary for most people, but it could be

useful for a solider in the battlefield, for example. Ultimately, enhancements of many kinds will "definitely" happen in the future, says Fyodor Urnov of Sangamo BioSciences, a company that is working in the gene editing space. The bigger question, he says, is when it will happen.

Cheaper, and more efficient, gene editing technology that allows scientists to manipulate the human genome with greater ease and precision than ever before is forcing researchers to consider these questions quickly. Most notably, researchers are eyeing CRISPR—short for the cumbersomely-named clustered regularly interspaced short palindromic repeats. CRISPR is a powerful technology that allows editing—by way of replacing or repairing—of multiple genes at once in animal, plant and human cells. This biological dynamo could help unlock understanding of basic human biology and also help patients in need of medical care. The method has also sparked new ethical controversy. Last spring researchers in China announced they used CRISPR to alter the genomes of nonviable human embryos which could not develop into babies. They discovered the method is not yet accurate enough to be utilized in human embryos and also that it appeared to introduce unexpected mutations to other parts of the genome. Ultimately, this week's discourse will lead to a consensus statement providing some guidance on how to approach using this and older gene editing technologies such as zinc finger nucleases and enzymes called transcription activator-like effector nucleases, or TALENs. Meanwhile, the National Academies are working on separate studies related to how to address these questions for work in other species.

At issue this week is when and how to apply gene editing for research and clinical applications in humans. Gene editing could include altering genes in one person—say to treat leukemia in one patient or make a cosmetic change—but, more controversially, it could also include making changes to the germ line that would then alter the genome for an individual's children, grandchildren and the following generations, with potentially unknown repercussions.

Although most scientists at the meeting appear enthusiastic about conducting gene editing work to cure diseases in individual patients they remain more wary of making changes to eggs, sperm or embryos that would have lasting repercussions in future generations. The former target, say, using gene editing techniques to inactivate HIV receptors and achieve resistance of blood cells to the virus (which Sangamo BioSciences is working on in clinical trials) is different than helping parents who both carry genes for Huntington's Disease to have a child that is free of the disease (a change to the genome that would be passed on to future generations and would likely not be very commonly needed).

For his part, Eric Lander from the Broad Institute said in a December 1 presentation at the summit that the need to employ germ line editing would remain

very, very rare thanks to other already available reproductive technologies like in-vitro fertilization that could help most people. What's more important for avoiding genetic diseases is boosting access to gene testing so people can be more aware they are carriers for disease, he said. "What we should be thinking about is the vast majority of people with a recessive disease were not aware that they were carriers," he said. Armed with that genetic data, people could then employ existing aides like IVF or pre-implantation genetic diagnosis to conceive healthy offspring.

Yet at the clinical level the need for such germ line editing seems more real and arguably commonplace. George Daley of Harvard Medical School said on December 1 that he and his team have seen multiple patients affected by NEMO deficiency syndrome, a disorder where an inherited faulty gene results in a weak immune system and leaves patients prone to serious infections. To care for such patients, his team has seen families that try to have a second child– sometimes nicknamed a "savior sibling"—in the hopes that the second child's bone marrow can be employed to help the older sibling. Yet by the time families try to conceive their second child the parents are older and that likely contributes to why they have trouble getting pregnant quickly via IVF, he says. In such instances families could save time and perhaps achieve greater success if CRISPR was on the table to help ensure a single embryo with good results, he says. The question boils down to if one should consider purely the statistics or the human, says Baltimore. But either way, even if such circumstances are rare, they still exist and cannot be ignored. Perhaps the fact that CRISPR could be performed is reason enough to leave it on the table.

The need for guidance on how to attack these issues is undeniable considering the range of opinions at the conference. But guidance is not law. That may actually be a good thing. Guidance can remain more nimble than law and it is easier to get consensus among scientists working in the area than among individuals who do not work in the field, Baltimore says. In the absence of any international body that would be an obvious fit to enforce international regulations on gene editing there are historical precedents—like stem cell research—for providing guidance and then leaving the specifics up to regional authorities. Once the consensus statement is issued, either today or in the coming weeks and months, patients, lab scientists, ethicists and medical workers will be carefully watching what comes next.

Scientific American December 3, 2015
http://www.scientificamerican.com/article/improving-humans-with-customized-genes-sparks-debate-among-scientists1/

■ Glossary of essential terms

№	English term	Russian equivalent
1	heredity	наследственность
2	virologist	вирусолог
3	much-anticipated	долгожданный
4	gene editing	генное редактирование
5	tweaking	тонкая настройка
6	alteration	внесение изменений
7	subtraction	вычитание
8	prepared	подготовленный
9	justified	оправдано
10	enhancement	усиление, улучшение
11	purpose	цель
12	direction	направление
13	merely	просто
14	referring	ссылаться
15	boosting	повышение
16	desirable	желательный
17	pitch	подача
18	prominent	известный
19	dire	ужасный, кошмарный
20	current	текущий, современный
21	summit	саммит
22	grapple	сцепиться
23	thorny	тернистый, острый
24	chairing	возглавляющий
25	versus	против
26	solving	решение
27	cardiovascular disease	сердечно-сосудистое заболевание
28	intervention	вмешательство
29	murky	темный, туманный
30	trait	черта
31	battlefield	поле боя
32	efficient	эффективный
33	precision	точность

34	notably	особенно
35	sparked	загорелся
36	nonviable	нежизнеспособные
37	guidance	руководство
38	meanwhile	между тем
39	germ line	зародышевая линия
40	repercussions	последствия
41	former	бывший
42	resistance	устойчивость
43	very	очень
44	area	область
45	aware	известно
46	conceive	забеременеть
47	arguably	возможно
48	commonplace	банальный
49	disorder	расстройство
50	savior	спаситель
51	bone marrow	костный мозг
52	pregnant	беременная
53	ensure	убедиться
54	undeniable	бесспорно
55	consensus	консенсус
56	absence	отсутствие

■ Your Essential Assignments:

I. Quick check:

1. What is CRISPR?
2. How can we know whether we have a genetic mutation?
3. How will CRISPR be able to help families, who want to have a healthy baby?

II. Fill in the missing words:

Verb	Noun
define	
absent	
pregnant (become pregnant)	
intervene	

enhance	
tune	
chair	

III. Use a monolingual English dictionary and give the definitions of the words below:

guidance; intervention; enhancement; nonviable; heredity.

IV. Find English equivalents to the following word combinations:

№	Russian term	English equivalent
1	настройка генома человека с помощью дополнений, вычитаний и изменений	
2	относится к повышению мышечного тонуса	
3	меры, гарантирующие улучшение здоровья	
4	выдающийся исследователь	
5	бурные дискуссии	
6	разграничение, необходимое для улучшений	
7	разница между жизнью и смертью	
8	этические противоречия	
9	внесение изменений в зародышевую линию	
10	спаситель родного брата	
11	стволовые клетки	
12	руководство может оставаться более гибким	

V. Suggest Russian equivalents for the following word combinations:

№	English term	Russian equivalent
1	tweaking the human genome	
2	who is chairing the summit	
3	to drawing the line	
4	solving a life-threatening issue	
5	lower the risk of cardiovascular disease	
6	more efficient	
7	unexpected mutations to other parts of the genome	
8	transcription activator-like effector nucleases	
9	the former target	
10	inactivate HIV receptors	
11	to conceive healthy offspring	
12	more nimble	

VI. Find synonyms among the pool of words:

Pool of words	Synonyms
1) heredity\long-awaited\inheritance\much-anticipated	
2) prominent\thorny\known\acute\famous\sharp	
3) merely\current\simply\feature\actual\trait	
4) savior\stability\redeemer\former\resistance\ex	

VII. Answer the following questions. Use all information given before.

1. Why don't scientists want to introduce gene editing to the masses?
2. What "enhancements" of the human can you offer?
3. How can genes be edited?
4. What did researchers from China work with, applying CRISPR-system?
5. Is there an accumulation of mutations at generations because of big survival of the population of Earth nowadays?
6. What ethical problems can scientists face with?
7. Why do people, who have a child with the NEMO syndrome, want to have a second baby? What for?
8. What is better for scientists: guidance or law?

VIII. Match the sentence halves. Make complete sentences:

1.	Part of the problem for researchers, doctors and ethicists alike is defining	**A.**	with greater ease and precision than ever before is forcing researchers to consider these questions quickly.
2.	Baltimore, who is chairing the summit, believes that	**B.**	is different than helping parents who both carry genes for Huntington's Disease to have a child that is free of the disease (a change to the genome that would be passed on to future generations and would likely not be very commonly needed).
3.	Tweaking it could make a person function like	**C.**	to attack these issues is undeniable considering the range of opinions at the conference.
4.	Cheaper, and more efficient, gene editing technology that allows scientists to manipulate the human genome	**D.**	that when it comes to drawing the line for enhancement, the determining factor is whether the change would be optional versus therapeutic.

5.	The former target, say, using gene editing techniques to inactivate HIV receptors and achieve resistance of blood cells to the virus (which Sangamo BioSciences is working on in clinical trials)	**E.**	consider purely the statistics or the human, says Baltimore.
6.	The need for guidance on how	**F.**	– sometimes nicknamed a "savior sibling"—in the hopes that the second child's bone marrow can be employed to help the older sibling.
7.	To care for such patients, his team has seen families that try to have a second child	**G.**	very rare thanks to other already available reproductive technologies like in-vitro fertilization that could help most people.
8.	The question boils down to if one should	**H.**	"enhancement" and deciding if it would be a move in the right direction, as the word would suggest.
9.	For his part, Eric Lander from the Broad Institute said in a December 1 presentation at the summit that the need to employ germ line editing would remain very,	**K.**	the rare individuals who are born with a variant that allows them to function well with just a few hours of sleep.

IX. Read and translate the short text without any dictionary:

The ability to precisely and accurately change almost any part of any genome, even in complex species such as humans, may soon become a reality through genome editing. But with great power comes great responsibility – and few subjects elicit such heated debates about moral rights and wrongs. Although genetic engineering techniques have been around for some time, genome editing can achieve this with lower error rates, more simply and cheaply than ever – although the technology is certainly not yet perfect.

Genome editing offers a greater degree of control and precision in how specific DNA sequences are changed. It could be used in basic science, for human health, or improvements to crops. There are a variety of techniques but clustered regularly inter-spaced short palindromic repeats, or CRISPR, is perhaps the foremost.

X. Food for thought:

Genome editing techniques have so far been used to change genomes in individual cells and in entire (non-human) organisms. Benefits have included better targeted gene therapy in animal models of some diseases, such as Duchenne Muscular Dystrophy. It's also hoped that it will lead to a better understanding of the structure, function and regulation of genes. Genetic modification through genome editing of plants has already created herbicide- and infection-resistant crops.

But more contentious is how genome editing might be used to change traits in humans. While this has been the basis for many works of fiction, in real life our capacity to provide the sort of genetic engineering seen in films and books such as Gattaca and Brave New World has been substantially limited. Genome editing potentially changes this, presenting us with the very real possibility that any aspect of the human genome could be manipulated as we desire. This could mean eliminating harmful genetic conditions, or enhancing traits deemed advantageous, such as resistance to diseases. But this ability may also open the door to eugenics, where those with access to the technology could select for future generations based on traits considered merely desirable: eye, skin or hair color, or height.

Text 3. Where to Draw the Line on Gene-Editing Technology

Pre-reading

1. Have you ever heard about gene-editing technology?
2. Do you know what does CRISPR mean?

■ Read the given text and make your essential assignments:

The biologists have done it again. Not so long ago it was cloning and embryonic stem cells that challenged moral imagination. These days all eyes are on a powerful new technique for engineering or "editing" DNA. Relatively easy to learn and to use, CRISPR has forced scientists, ethicists and policymakers to reconsider one of the few seeming red lines in experimental biology: the difference between genetically modifying an individual's somatic cells and engineering the germ line that will be transmitted to future generations. Instead of genetic engineering for one person why not eliminate that disease trait from all of her or his descendants?

This week, the U.S. National Academy of Sciences, the Chinese Academy of Sciences, and the U.K. Royal Society are trying to find ways to redraw that red line. And redraw it in a way that allows the technology to help and not to hurt humanity. Perhaps the hardest but most critical part of the ethical challenge: doing that in a way that doesn't go down a dark path of "improvements" to the human race.

Compared to previous strategies, the technique known as CRISPR (clustered interspaced short palindromic repeats) is faster, more reliable and cheaper than previous methods for modifying the base pairs of genes. CRISPR is made up of scissors in the form of an enzyme that cuts DNA strands and an RNA guide that knows where to make the cut, so the traits expressed by the gene are changed. Already, labs are applying gene editing in pluripotent stem cells. Older methods are being used to help the human immune system's T cells resist HIV, which might be done better with CRISPR. Gene editing trials are also in the offing for diseases like leukemia. It looks very much like these genies are out of the bottle.

Since the 1960s, technical limitations, the prospect of unintended consequences, and the "eugenic" implications of deliberate alterations of future generations weighed heavily against germline engineering. Many countries, including many in Europe, have laws that forbid human germline modification. The National Institutes of Health won't pay for such research but there's no law against using private funds. China also doesn't prohibit it.

But over the past 20 years, advances in laboratory techniques, genetic screening for disease traits, and the prospective fruits of the Human Genome Project have smudged the red line. Gradually, the public health benefits of changing the human germline have gained as much emphasis as the risks. Some observers have noted that, aside from the efficiencies that could be realized with germline engineering, the ethical distinction between germline and somatic cell modification may already be moot, since even somatic cell modifications can

"leak" into effects on gametes. The emergence of CRISPR has made it impossible to delay more definitive guidance.

Even apart from risks and benefits, are we prepared to modify our genetic heritage with all the implications for humanity's relationship to the rest of the natural world? Following a wave of publicity about CRISPR, last spring a number of scientists and researchers called for a voluntary moratorium on its use. Their proposal was reminiscent of the Asilomar moratorium on recombinant DNA research in 1975. Asilomar is commonly (but not universally) thought to have been an effective response on the part of the scientific community to public fears about biohazards.

But the world of life sciences research is far different now than it was 40 years ago, when the community was much smaller and more intimate. Sophisticated experimental biology is now a globalized affair. Funding pressures, the virtually instantaneous availability of experimental procedures and results, and the fact that researchers may have limited face-to-face contact make self-policing far more challenging than it once was. Indeed, within weeks of the calls for a moratorium, a Chinese team performed a modification of non-viable embryos, a proof-of-concept experiment that fell smack into the ethical grey zone and further shook confidence in the prospects for an effective moratorium.

With events moving so quickly, the summit organized by the U.S. National Academies, along with its British and Chinese counterparts, will need to face a few key ethical issues. How can technical risks, like "off target" effects that change an important gene instead of the one intended, be avoided? Are there any diseases that could justify attempts to diffuse genetic changes in a human population? And who is to make such monumental decisions on behalf of unborn generations? Recommendations from the Academies aren't law but they can establish guiding principles for legitimate scientific practices.

Ten years ago a National Academy of Sciences committee that I co-chaired set rules for doing human embryonic stem cell research that were voluntarily adopted in many parts of the world. When it comes to the ethics of science, the scientific community needs to lead but also needs to listen to non-scientists. Especially in the case of the human germline, one principle worth defending is that between therapy and enhancement. Even if population-wide disease prevention is sometimes acceptable, attempts to otherwise "improve" the human race should be banned.

Other principles will apply mainly to agricultural research. Genetically modified plants and animals are the focus of a parallel National Academies study on the ecological risks of gene drive experiments that might someday lead to

deliberate changes of non-human populations in the wild. New techniques like CRISPR will make the recently approved fast growing salmon look old fashioned.

The experiments that are both the most promising and the most risky are that those that involve rapidly propagating species like insects, like eliminating the ability of mosquitoes to carry the malaria parasite. And accidents are always possible so best biosafety practices will have to be reviewed and strengthened, including perhaps inbred biological barriers like the “suicide genes” that will cause modified organisms to die if they escape the lab.

One thing is clear: CRISPR and its descendants will have lives beyond the laboratory.

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http://www.scientificamerican.com/article/where-to-draw-the-line-on-gene-editing-technology

- **Glossary of essential terms**

№	English term	Russian equivalent
1	experimental biology	эксперементальная биология
2	genetically modifying	генетически модифицированный
3	germline	зародышевая линия
4	cloning	клонироание
5	embryonic stem cells	эмбриональные стволовые клетки
6	human race	человеческая раса
7	enzyme	фермент
8	private funds	частные средства
9	distinction	различия
10	moot	спор
11	pluripotent	плюрипотентные
12	alteration	переделка
13	deliberate	преднамеренная
14	reminiscent	напоминает
15	genetic heritage	генетическое наследство
16	intimate	близкие, глубокие
17	biohazards	биологическая опасность
18	community	община

19	smudged	размазанный, нечеткий
20	funding	финансирование
21	affair	дело
22	self-policing	самоконтроль
23	non-viable	нежизнеспособный
24	enhancement	повышение
25	unborn	неродившийся
26	proof-of-concept	доказательство правильной концепции
27	legitimate	законные, допустимые
28	prevention	профилактика
29	agriculture	сельское хозяйство
30	promising	перспективный
31	propagating	распространяющиеся
32	accidents	несчастные случаи
33	biosafety	биобезопасность
34	suicide gene	гены-самоубийцы
35	descendants	потомки
36	imagination	воображение
37	powerful	мощный
38	forced	вынуждены
39	instead	вместо
40	redraw	перерисовать
41	improvements	улучшение
42	limitations	ограничения
43	consequences	последствия
44	benefit	выгода

- **Your Essential Assignments**

I. Quick check:

1. What stands for CRISPR?

2. Are there laws prohibiting the modification of the human germ-line?

3. What diseases can be treated with gene editing trials?

4. What does CRISPR technique do?

II. Suggest Russian equivalents for the following word combinations:

№	English term	Russian equivalent
1	embryonic stem cells	

2	genetically modifying	
3	somatic cell modification	
4	humanity's relationship	
5	scientific community	
6	pluripotent stem cells	
7	proof-of-concept	
8	key ethical issues	
9	unborn generations	
10	ecological risks	
11	suicide genes	

III. Fill in the gaps with the words and expressions from the text:

1. Not so long ago it was_____ and _____that challenged moral imagination.
2. Labs are already applying ____editing _____cells.
3. Other principles will apply mainly to _____ research.
4. The _____that are both the most promising and the most risky are that those that involve rapidly propagating species like_____, like eliminating the ability of mosquitoes to carry the _____parasite
5. But the _____sciences research is far different now than it was 40 years ago, when the _____was much smaller and more intimate.
6. Indeed, within weeks of the calls for a_____, a Chinese team performed a modification of_____, a proof-of-concept experiment that fell smack into the ethical grey zone and further shook confidence in the prospects for an effective moratorium.

IV. Use monolingual English dictionary and write down what could the words given below mean:

Germline, imitations, modifications, focus, experiments, descendants.

V. Find English equivalents for the following word combinations:

№	Russian term	English equivalent
1	"редактирование" ДНК	
2	генная инженерия	
3	базовая пара генов	
4	Т-клетки иммунной системы	
5	лейкемия	
6	лабораторные методы	
7	рекомбинанторные исследования	

8	нежизнеспособные эмбрионы	
9	сельскохозяйственные исследования	
10	генетически модифицированные растения и животные	
11	паразит малярии	
12	жизнь за пределами лаборатории	
13	"гены-самоубийцы"	

VI. Fill in the missing words:

Term (verb)	**Noun**	**Adjective**
experiment		
modify		
improve		
inherit		
risk		
imagine		

VII. Match the sentence halves. Make complete sentences.

1.	Not so long ago it was cloning and embryonic stem cells	A.	is faster, more reliable and cheaper than previous methods for modifying the base pairs of genes.
2.	Perhaps the hardest but most critical part of the ethical challenge:	B.	are trying to find ways to redraw that red line.
3.	Following a wave of publicity about CRISPR	C.	will have lives beyond the laboratory.
4.	When it comes to the ethics of science,	D.	attempts to otherwise “improve” the human race should be banned.
5.	Even if population-wide disease prevention is sometimes acceptable,	E.	the scientific community needs to lead but also needs to listen to non-scientists.
6.	One thing is clear: CRISPR and its descendants	F.	last spring a number of scientists and researchers called for a voluntary moratorium on its use.
7.	This week, the U.S. National Academy of Sciences, the Chinese Academy of Sciences, and the U.K.	G.	doing that in a way that doesn’t go down a dark path of “improvements” to the human race.

	Royal Society		
8.	Compared to previous strategies, the technique known as CRISPR (clustered interspaced short palindromic repeats)	H.	that challenged moral imagination.

VIII. Read and translate the short text without any dictionary:

Mankind has always dreamed of, to display a miracle cure, heal all diseases, or at least something similar. Cloning is one of the closest to this idea means.

Cloning allows you to grow parts of the human body, using the DNA of the host. These body parts can be used to replace the existing, but not useful. It is also possible to clone certain organs and replaced them sick bodies. Many people die from organ failure or loose bodies in accidents or born with imperfections. These people could be cured with the help of cloning.

IX. Match these words with their definitions:

1	enzyme	A	a result or effect of an action or condition.
2	agriculture	B	a series of germ cells each descended or developed from earlier cells in the series, regarded as continuing through successive generations of an organism.
3	benefits	C	property that is or may be inherited; an inheritance
4	diseases	D	human beings in general; humankind.
5	improve	E	the ability to do something or act in a particular way, especially as a faculty or quality.
6	clone	F	the faculty or action of forming new ideas, or images or concepts of external objects not present to the senses.
7	somatic cell	G	the action of stopping something from happening or arising.
8	prevention	H	any cell of a living organism other than the reproductive cells.
9	imagination	I	an organism or cell, or group of organisms or cells, produced asexually from one ancestor or stock, to which they are genetically identical.
10	power	J	make or become better.
11	human race	K	a disorder of structure or function in a human, animal, or plant, especially one that produces specific signs or symptoms or that affects a specific location and is not simply a direct result of physical injury.

12	heritage	L	a public performance or other entertainment of which the proceeds go to a particular charitable cause.
13	germ line	M	the science or practice of farming, including cultivation of the soil for the growing of crops and the rearing of animals to provide food, wool, and other products.
14	consequences	N	a substance produced by a living organism that acts as a catalyst to bring about a specific biochemical reaction.

X. Food for thought:

According to the supporters of eugenics, the society through the development of medical and social support for the disabled and other "artificial" measures to improve the quality of life has weakened the action of natural selection, whereby there was a danger of degeneration of nations. "Subnormal" individuals participating in reproduction, "clog up" the so-called "gene pool of the nation" substandard genes. Eugenic practices aimed at to stop the degeneration of the genetic population.

What do you think? Should people get involved in the genome or leave the matter to God?

Text 4. Childhood Cancer Risk Hides in Families

Genome study finds mutations passed on from parents are linked to at least one in 12 cancers in kids

Pre - reading.

■ **Work in pairs. Discuss the following questions and try to answer them.**

1. What are the causes of the cancer cells?
2. What has always characterized cancer?
3. What happens when a cancer cell is reborn?

■ Read the given text and make your essential assignments

A substantial number of children with cancer carry cancer-predisposing mutations inherited from a parent, according to a new study published Wednesday in The New England Journal of Medicine. Researchers examined the genes of more than a thousand children with cancer and found that 8.5 percent of them—most of whom had little family history of cancer—carry handed-down gene mutations that make them more susceptible to the disease. The figure might seem small, especially within a relatively rare diagnosis, but its implications are large—for the young patients and for their family members who might also be at risk—and could help doctors select more appropriate treatments.

Pediatric cancer is a mysterious case. Most cancers in the general population are caused by genetic mutations accumulated over a lifetime. But kids have not been around long enough to experience large amounts of UV rays, chemicals or other exposures that can lead to or exacerbate errors in DNA and spur cancer growth.

These findings "give us fundamental insights into the etiology of pediatric cancer—to help treat patients," says James Downing, chair of Childhood Cancer Treatment, president and CEO of St. Jude Children's Research Hospital and co-author of the new study, which is part of the Saint Jude–Washington University Pediatric Cancer Genome Project.

Predispositions

The researchers analyzed the genes of 1,120 pediatric cancer patients (median age 6.9), honing in on 60 genes that are associated with cancer predisposition to look for DNA errors passed down from a parent, known as germ-line mutations. These cancer-linked errors were present in 95 patients, most commonly in children with solid tumors outside of the central nervous system—and least frequently in those with leukemia. "This kind of work helps in understanding where the tumors are coming from—and also what are some of the underlying driving mutations," says Lisa Diller, chief medical officer of Dana–Farber/Boston Children's Cancer and Blood Disorders Center, who was not involved in the new work

For comparison, the team also analyzed these same genes in 966 (noncancerous patient) adults in the general population as represented by the 1,000 Genomes Project and found these germ-line mutations in only about 1.1 percent of people.

The researcher uncovered data from the pediatric cancer population that might alter long-standing traditions. "A big surprise is the family history," says Jinghui Zhang, chair of the Computational Biology department at Saint Jude's and

a co-author of the study. Or, more precisely, the lack thereof. Family histories have been a central tool for understanding the nature of a cancer and identifying persons who might be at risk. But Zhang and her colleagues found that of the patients with these germ-line mutations and with family cancer data, only 40 percent had a family history of cancer (which was a similar rate to the pediatric patients who did not have one of these flagged mutations). Furthermore, only 57 percent of the family cancers matched with the genetic predisposition.

Test and screen?

The heritable nature of these genetic mutations means that some in a patient's family—parents or siblings—might indeed harbor the same errors. But instituting testing and screening for all close relatives of a patient's is not an automatic next step. Although a family will likely want to know: "How likely is it that the germ-line mutation would progress to a harmful cancer?" Downing notes the answer will vary for different mutations. Heritable syndromes, including cancer predisposition, have a wide range of likelihoods of actually causing the disease, a concept known as penetrance. Many mutations—which exponentially raise risk for carriers against that of the general population—only go on to cause cancer in a relatively small fraction of carriers, Zhang explains. For most of these variants, however, "the penetrance is unknown," she says.

Some of the risk might become known when a parent is diagnosed with cancer. Interestingly, a handful of children with cancer harbored germ-line mutations on the BRACA1 or BRACA2 genes, which are typically associated with adult-onset cancers. In her hospital, Diller says, they reassure mothers with BRACA-associated breast cancer that any risk they might have passed down to their offspring is only for cancer later in life. Now, Diller says, "we'll have to look at if we need to change that policy—whether we should consider children of BRACA carriers at a higher risk" for cancers much earlier in life.

Treatment

In the meantime knowing that a patient has one of these mutations can help their doctors select treatment. "When a child has a mutation in these genes, it really tells us to stop and pause," Downing says. "We really need to approach that individual patient differently." Some gene signatures, for instance, raise the individual's susceptibility to the effects of radiation, a treatment that might otherwise be standard.

Additionally, Diller points out, having a germ-line mutation that is linked to a cancer in a paired organ, such as a kidney or retina, means that the other organ is also at higher risk. So instead of removing a kidney, as might be common in severe renal adenocarcinomas, doctors might prefer to do their best to save that organ, knowing that the other one might fall victim to cancer as well.

Bone marrow transplants, a common treatment for childhood cancers, might also need new considerations for these patients. Among the best marrow donors are matched siblings. But, as Diller notes, knowing these germ-line mutations' 50 percent probability of being present in a sibling, "you would want to make sure that the matched sibling is not also a carrier of the gene syndrome," she says.

Growing data, spreading syndromes

The findings provide the basis for a new program at Saint Jude's known as Genomes for Kids, or G4K, which will test all new admitted pediatric cancer patients for germ-line mutations. This will not only allow clinicians to use the results when deciding on treatment and family screening but help researchers pin down more links between heritable mutations and childhood cancer.

As more data come in, the frequency of these germ-line predisposition gene mutations will likely rise, Downing notes. "That frequency [of 8.5 percent] will probably be a lower limit," he says. "There will be more and more genes identified." And as the roster of potentially pathogenic gene mutations grows—and as more people have their genomes tested—Diller says we also need to be ready with better information about individual risk, along with helpful screening and lifestyle recommendations. For carriers of these mutations, Downing says, we still do not know if or when surveillance is appropriate. And that is no small issue, as Diller points out. Finding, for example, that a sibling of a cancer patient also carries the mutation, "you immediately make them into a patient rather than a healthy child—just because they carry a gene that we don't know how to interpret," she says.

Scientific American November 18, 2015

http://www.scientificamerican.com/article/childhood-cancer-risk-hides-in-families/

■ Glossary of essential terms for you to know

	English term	Russian equivalent
1	implication	значение, следствие
2	exacerbate	обострять, усиливать
3	spur	подгонять, толкать
4	substantial	существенный, значительный
5	predisposing	предрасположенный
6	susceptible	восприимчивый
7	screen	сортировать
8	rare	редкий
9	harbor	питать, затаить

10	surveillance	наблюдение
11	renal	почечный
12	mysterious	мистический
13	case	случай
14	to accumulate	накапливать
15	exposure	воздействие
16	to spur	побуждать
17	chair	председатель
18	predisposition	предрасположение, склонность
19	germ-line mutation	мутация зародышевой линии
20	tumor	опухоль
21	to alter	изменять
22	more precisely	точнее
23	sibling	родной брат, родная сестра
24	indeed	действительно
25	likelihood	вероятность
26	penetration	проникновение
27	breast	молочная железа
28	additionally	кроме того
29	kidney	почка
30	retina	сетчатка
31	severe	тяжелый, выраженный
32	marrow	костный мозг
33	roster	список
34	to alter	изменить
35	to approach	подходить, подступаться

I. Try to match the definition with the correct word.

1.mutation	**A.** medical care for an illness or injury
2.disease	**B.** the hereditary passing of biological attributes from ancestors to off-spring
3.cancer	**C**. disease of uncontrolled cellular proliferation
4.treatment	**D.** the state of being predisposed
5.predisposition	**E.** heritable change in genetic material
6.inheritance	**F.** the presiding officer of a meeting, organization, committee, or other deliberative body
7.chair	**G.** the proportion of individuals carrying a particular

	variation of a gene that also express an associated trait
8.penetrance	**H**. an abnormal condition of the body causing discomfort or dysfunction

II. Use monolingual English dictionary and write down what could the words given below mean:

Childhood; growth; susceptible; exposure; chemical; penetrance.

III. Find English equivalents to the following word combinations:

Russian term	**English equivalent**
1. известен как	
2. может помочь врачам выбрать наиболее подходящие процедуры	
3. наследуемая природа генетических мутаций	
4. предрасполагающие к раку мутации	
5. таинственный случай	
6. пересадка костного мозга	
7. немедленно сделать	
8. унаследовал от родителей	
9. парный орган	
10. экспоненциально повышают риск для носителей	
11. главный инструмент для понимания природы рака	
12.информация об индивидуальном риске	
13. семейная история рака	
14. центральная нервная система	
15. восприимчивы к болезни	

IV. Give Russian equivalents to the following English terms:

English term	**Russian equivalent**
1. family histories have been a central tool for understanding the nature of a cancer	
2. DNA errors passed down from a parent	
3. for comparison	
4. pediatric cancer is a mysterious case	
5. the researchers uncovered data from the pediatric cancer population	

6. an automatic next step	
7. could help doctors select more appropriate treatments	
8. in a relatively small fraction of carriers	
9. researchers examined the genes	
10. to give us fundamental insights into the etiology of pediatric cancer	
11. children with solid tumors outside of the central nervous system	
12. cancer-predisposing mutations	
13. known as germ-line mutations	
14. a big surprise is the family history	
15. genetic mutations accumulated over a lifetime	

V. Match the sentence halves. Make complete sentences:

1. This will not only allow clinicians to use the results when deciding on treatment and family screening but	A. the individual's susceptibility to the effects of radiation, a treatment that might otherwise be standard.
2. Some of the risk might become known	B. a central tool for understanding the nature of a cancer and identifying persons who might be at risk.
3. Among the best marrow donors	C. a patient has one of these mutations can help their doctors select treatment.
4. The heritable nature of these genetic mutations means	D. when a parent is diagnosed with cancer.
5. Some gene signatures, for instance, raise	E. help researchers pin down more links between heritable mutations and childhood cancer.
6.Family histories have been	F. that some in a patient's family—parents or siblings—might indeed harbor the same errors.
7. Most cancers in the general population are caused	G.are matched siblings.
8. In the meantime knowing that	H. by genetic mutations accumulated over a lifetime.

VI. Translate into English using all the active possible:

1. Семейные истории были главным инструментом для понимания природы рака и выявления лиц, которые могут оказаться под угрозой.

2. Это не только позволит врачам использовать результаты при принятии решения о лечении и семейном скрининге, но и поможет исследователям установить большую связь между наследственными мутациями и раком детей.

3. Детский рак - это таинственное явление.

4. Но назначение тестирования и скрининга всех близких родственников пациента не является автоматически следующим шагом.

5. Лучшие доноры костного мозга – братья и сестры.

6. Большинство случаев рака среди населения в целом вызвано генетическими мутациями, накопленными в течение жизни.

7. Более того, только 57% случаев рака в семье обусловлено генетической предрасположенностью.

VII. Fill in the missing words:

Verb	**Noun**
to examine	
	selection
to accumulate	
	association
to know	
	presentation
	involvement
to represent	
to understand	
	identification
	inclusion
to explain	
	consideration
to treat	
to remove	
	preference
	spread
	reassurance
to provide	
	admission
	allowance
to appropriate	

VIII. Write words for the definitions.

1. Someone who receives treatment from a doctor or other medically educated person.

p_____

2. Observation of individuals or groups of individuals.

s_____

3. The measure of how often a periodic event occurs.

f_____

4. Substance inside bones.

m_____

5. An organ in the body that produces urine.

k_____

6. A group of organisms of one species, occupying a defined area.

p_____

7. A value or condition that is not consistent with the true, specified, or expected value or condition.

e_____

8. Originator or creator of a work.

a_____

IX. Fill each gap with one word.

1. Pediatric cancer is a _____ case.
2. _____ histories have been a _____ tool for understanding the _____ of a cancer and identifying persons who might be at risk.
3. Or, more precisely, the _____ thereof.
4. The researches uncovered data from the _____ cancer population that might alter long-standing traditions.
5. In the meantime knowing that a patient has one of these mutations can _____ their doctors _____ treatment.
6. Most cancers in the general population by _____ mutations _____ over a lifetime.
7. Some of the _____ might become known when a parent is diagnosed with cancer.

8. But instituting _____ and _____ for all close relatives of a patient's is not an automatic _____ step.

X. Complete with the right preposition.

1. Genome study finds mutations passed on from parents are linked to _____ least one _____ 12 cancers in kids.
2. Most cancers _____ the general population are caused _____ genetic mutations accumulated over a lifetime.
3. _____ carriers_____ these mutations, Downing says, we still do not know if or when surveillance is appropriate.
4. This will not only allow clinicians to use the results when deciding _____ treatment and family screening but help researchers pin down more links between heritable mutations and childhood cancer.
5. As more data come in, the frequency _____ these germ-line predisposition gene mutations will likely rise, Downing notes.
6. Bone marrow transplants, a common treatment _____ childhood cancers, might also need new considerations _____ these patients.

Text 5. General Anesthesia Causes No Cognitive Deficit in Infants

Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them.**

- *What is anesthesia?*
- *Are there restrictions on the use of anesthesia?*

■ **Read the given text and make your essential assignments:**

Early-life exposure to anesthesia does not appear to lead to long-term cognitive problems, researchers announced today. New evidence from the first, randomized anesthesia trial in kids provides the strongest indication yet that exposing young children to anesthesia—at least for a brief time—will not saddle them with developmental deficits. The news comes just a couple of weeks after a

medical advisory group reiterated its concerns about such exposures among children younger than four years. Previously, multiple animal and human studies have linked such exposure with cognitive impairment, but none of the information on humans came from a gold-standard, randomized study design that could help eliminate other reasons to explain such a connection.

This is a "reassuring finding, but it is not the final answer," says Dean Andropoulos, anesthesiologist in chief at Texas Children's Hospital and an expert who was not involved in the work. The new study assesses only what happens to youngsters after a relatively brief bout with anesthetics, so it is possible that longer or repeated exposures to such chemicals may still cause neurodevelopmental issues. There may also be deficits in anesthesia-exposed children that are not measurable until later in life.

The study followed more than 500 infants undergoing hernia repair across the U.S., Australia, the U.K., Canada, the Netherlands, New Zealand and Italy. The surgeries lasted an average of roughly an hour. About half of the children were randomly selected to be put under with general anesthesia, and the other half stayed awake during the surgery and received targeted anesthetic in a specific body region. The kids in the study were all younger than 60 weeks and were matched by where they had the surgery and whether they were born prematurely.

At age two, the children in both groups completed a battery of neurocognitive tests that examined how they thought and reacted to the world around them. The researchers found that the kids in both groups performed similarly. "This is the strongest evidence we have to date that a brief anesthetic exposure likely isn't a problem," says Andrew Davidson, leader of the investigation and director of the Melbourne Children's Trials Center at the Murdoch Children's Research Institute.

The findings were published October 24 in the *Lancet* and will be presented Sunday at the American Society of Anesthesiologists annual meeting in San Diego.

In recent weeks a medical group concerned about exposing young children to anesthesia, which includes the American Academy of Pediatrics and the U.S. Food and Drug Administration, cited the growing body of literature linking anesthesia exposure and neurodevelopmental issues to emphasize that although parents should not hold off on medically necessary surgery, they should always weigh the pros and cons of exposing a young child to anesthesia or sedatives. Numerous animal studies have indicated that anesthesia exposure early in life, when the brain is exceptionally sensitive, can lead to brain cell death and altered connections between neurons. The group also urged medical providers and parents to try to avoid using anesthetics during diagnostic procedures such as MRIs whenever possible.

Many of the surgeries performed on young children are short, similar to the ones in the experiment, but there are still unanswered questions about how these brief exposures to anesthesia may influence brain function later in life. To test if there may be harm from short exposures, those in the current study will also be reassessed at age five with a new spate of memory and cognitive tests that could pick up subtler differences that may not have been apparent at a young age, Davidson says. “There are some aspects of development—like high executive function, reasoning skills and memory—that you don’t actually acquire until you are older.”

Although most kids are generally healthy and do not require early-life surgeries or diagnostic procedures, in total at least half a million children younger than three years are exposed to anesthetic agents annually. There are several ongoing studies examining the long-term neurocognitive effects of such experiences. Meanwhile researchers are also looking into possible alternative anesthetics and ways to mitigate any anesthesia-related neurological damage.

Scientists are also trying to determine how to assess any real-world implications of a child’s potential anesthesia-linked neurological deficits. Testing a few points worse on IQ tests or other cognitive measurements may not substantially change a child’s daily life. But developmental experts have worried about how those deficits could stack up if they are common among many kids exposed to early anesthesia.

Until now, findings from observational studies of kids who had early-life surgeries have been mixed. They could not definitively answer if the anesthesia itself harms the brain or if some other underlying issues were at work—such as sicker kids need surgery and go on to have cognitive issues fueled by their illness. This new study, at least, helps to answer that question.

Scientific American October 24, 2015

http://www.scientificamerican.com/article/general-anesthesia-causes-no-cognitive-deficit-in-infants/

■ Glossary of essential terms for you to know

	English term	Russian equivalent
1	anesthesia	наркоз
2	infant	младенец
3	hernia	грыжа
4	announce	объявлять
5	evidence	подтверждение
6	indication	доказательство
7	a brief time	короткое время

8	a medical advisory group	врачебно- консультативная группа
9	concern	обеспокоенность
10	previously	ранее
11	impairment	нарушение
12	study design	исследование
13	reassure	обнадеживает
14	brief bout	непродолжительная схватка
15	exposures	воздействие
16	exposed children	облученные дети
17	undergoing	перенесших
18	surgery	операция
19	targeted anesthetic	местная анестезия
20	react	реагировать
21	similarly	аналогично
22	society	общество
23	drug	сильнодействующие лекарства
24	necessary	причины
25	weigh	взвешивать
26	sedatives	седативные средства
27	altered connections	измененные соединения
28	avoid	избежать
29	diagnostic procedures	диагностические процедуры
30	brief exposures	краткие воздействия
31	harm	вред
32	spate	всплеск
33	subtler differences	тонкие различия
34	the current study	текущее исследование
35	annually	ежегодно
36	ongoing	продолжающееся
37	experience	переживание
38	meanwhile	между тем
39	mitigate	смягчать
40	assess	оценить
41	implication	последствия
42	measurement	измерение
43	not substantially	не существенно
44	until	до сих пор

45	definitively	окончательно
46	harms the brain	вредит мозгу

■ Your Essential Assignments

I. Quick check

1. What is anesthesia?
2. What is the effect of anesthesia on the infants' health?
3. Does anesthesia influence on children's development?

II. Suggest Russian equivalents for the following word combinations:

English term	Russian equivalent
cognitive deficit	
reiterated its concerns	
help eliminate other reasons	
brief bout with anesthetics	
awake during the surgery	
reacted to the world around them	
weigh the pros and cons	
exceptionally sensitive	
a new spate of memory	
ways to mitigate any anesthesia	
change a child's daily life	

III. Find synonyms among the pool of words:

Pool of words	Synonyms
1 announce 2 assesses 3 evaluate 4 declare	
1 respond 2react 3 explore4 research	
1 impairment 2 necessary3 violations 4 reasons	

IV. Pick antonyms to the words

Words	Antonyms (opposites)
evidence	
harm	
healthy	
react	
mitigate	

V. Use a monolingual English dictionary and write down what could the words given below mean:

Infant, concern, impairment, surgery, harm, implication

VI. Find English equivalents to the following word combinations:

Russian term	**English equivalent**
Обеспечивается сильнейшим доказательством	
Продолжительное или многократное воздействие такими химическими веществами	
Подвергнуты общему наркозу	
Руководитель исследования	
Не стоит откладывать операцию по медицинским показаниям	
Оставшиеся без ответа вопросы	
Умение аргументировать свою точку зрения	
Подвергаются воздействию анестетиков ежегодно	
Ученые изучают возможности альтернативных анестетиков	

VII Fill in the gaps with the words and expressions from the text:

1. This is a…… says Dean Andropoulos, anesthesiologist in chief at Texas Children's Hospital and an expert who was not involved in the work.

2. The surgeries lasted an average….

3. In recent weeks a medical group concerned about exposing young children to anesthesia, which includes……., cited the growing body of literature linking anesthesia exposure and neurodevelopmental issues to emphasize that although parents should not hold off on medically necessary surgery, they should always weigh the pros and cons of exposing a young child to anesthesia or sedatives

4. Although most kids are……. in total at least half a million children younger than three years are exposed to anesthetic agents annually

5. Scientists are also trying to determine…… of a child's potential anesthesia-linked neurological deficits.

VIII. Match the sentence halves. Make complete sentences:

1. Early-life exposure to anesthesia does	**A** Sunday at the American Society of Anesthesiologists annual meeting in San

	Diego
2. The kids in the study were	**B** on IQ tests or other cognitive measurements may not substantially change a child's daily life.
3. The findings were published October 24 in the *Lancet* and will be presented	**C** to avoid using anesthetics during diagnostic procedures such as MRIs whenever possible.
4. The group also urged medical providers and parents to try	**D** not appear to lead to long-term cognitive problems, researchers announced today
5. Although most kids are generally healthy and do not require early-life surgeries or diagnostic procedures,	**E** have been mixed
6. Meanwhile researchers are also looking into	**F** possible alternative anesthetics and ways to mitigate any anesthesia-related neurological damage
7. Testing a few points	**G** helps to answer that question.
8. Until now, findings from observational studies of kids who had early-life surgeries	**H** in total at least half a million children younger than three years are exposed to anesthetic agents annually
9. This new study, at least,	**I** all younger than 60 weeks and were matched by where they had the surgery and whether they were born prematurely.

IX. Translate into English using the text

1. Эта новость появилась буквально через пару недель после того, как врачебно-консультативная группа вновь выразила свою обеспокоенность по поводу таких случаев среди детей моложе 4 лет.

2. Изучению подверглись более 500 новорожденных, перенесших грыжесечение по всей территории США, Австралии, Великобритании, Канады, Нидерландов, Новой Зеландии и Италии.

3. В возрасте 2 лет дети в обеих группах завершили комплекс нейрокогнитивных тестов, было проверено, как они думают и реагируют на окружающий их мир.

4. Результаты были опубликованы 24 октября в Ланцет и будут представлены в воскресенье на ежегодном совещании американского общества анестезиологов в Сан-Диего.

5. Многочисленные исследования на животных показали, что воздействие наркоза на ранних этапах жизни, когда мозг исключительно чувствительный, может привести к смерти клеток мозга и изменению соединений между нейронами.

X. Read and translate the short text without any dictionary:

From the moment of birth in the human brain is, there are 14 billion cells, and the number is until death does not increase. On the contrary, after 25 years it decreases by 100 thousand a day. A minute spent on reading pages, dies about 70 cells. After 40 years of degradation of the brain is sharply accelerated, and after 50 neurons (nerve cells) dry out and reduces the volume of the brain.

XI. Answer the following questions. Use all information given before.

1. What did scientists do according to the text?
2. How were the children chosen for the study?
3. What does leader Andrew Davidson say about the study?
4. What are the challenges associated with anesthetics studied by scientists?

Text 6. Bee Symbiosis Reveals Life's Deepest Partnerships: Q&A

Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them.**

- *What is bee symbiosis?*
- *What other kinds of symbiosis can you name?*

■ **Read the given text and make your essential assignments:**

The biologist Nancy Moran has spent a career investigating the surprising nature of symbiosis, a phenomenon in which two species can appear to merge into one. Two years ago, Nancy Moran moved from Yale University to the University

of Texas, Austin, along with 120,000 bees. Bees are famous for living in large social groups, but Moran was interested in more than just the hive: She's delving into the diverse ecosystem of bacteria that evolved along with the bees, a group that contributes to the health of hives and their resilience to infection.

Bees and their microbiota are just one example of symbiosis — a close relationship between two species that typically helps both. Symbiosis can take a variety of forms. Cleaner fish scour dead skin from other fish and gain a meal in the process. The myriad microbes that live within our guts help us digest certain foods.

But much of Moran's work has focused on a deeper kind of partnership, one involving microbes known as endosymbionts that are passed from their host to its offspring. With her collaborator Paul Baumann, a bacteriologist at the University of California, Davis, Moran uncovered the tightly interwoven nature of these host-microbe relationships. Many pairs have become completely dependent on each other, some even swapping genes. Her research centered largely on aphids, sap-feeding insects that infest plants. The aphids can't survive without a microbe called *Buchnera aphidicola*, which lives within the insect and provides it with essential nutrients.

Moran is now doing similar work with bees, and her research could eventually help scientists understand colony collapse disorder, the mysterious plague devastating honeybee populations. Moran hopes an evolutionary biologist's perspective will also provide important insight into the workings of the human microbiome — the collection of microbes that live on and inside us — which has become a hot topic in human health.

Quanta Magazine spoke with Moran in July at the Society for Molecular Biology and Evolution conference in Vienna, where she presented her latest research on bees. An edited and condensed version of the interview follows.

QUANTA MAGAZINE: What was the field of symbiosis like when you started in the 1980s?

NANCY MORAN: It was a fringe topic in biology at the time. Most symbionts can't be studied outside their host, and everyone was ignoring them. There were very limited tools for studying them. Then in the 1980s, new molecular methods became available to amplify DNA and then sequence it.

How did you use those tools to study symbiosis?

One of the first things we did was create an evolutionary tree for aphids and their resident bacteria, *Buchnera*. We figured out they had coevolved for more than 100 million years, since aphids first appeared. Aphids basically wouldn't exist without symbionts. We found that pattern to be quite widespread. Major groups of insects evolved because they had symbionts.

How do symbionts help their insect hosts?

They often provide nutrition. Aphids live on plant sap, which is unsuitable for animals because it doesn't have all the essential amino acids *Buchnera* makes essential amino acids for them. The tsetse fly, which feeds on blood, has a symbiont that makes B vitamins. Using genomics, we have been able to find the exact genes that are required for making nutrients such as amino acids or vitamins.

How do symbionts change in response to their hosts?

When we started to sequence whole genomes of symbiotic bacteria, we realized they had unusual patterns of gene evolution. Endosymbionts often lose lots of genes. In fact, the extreme tininess of some of the endosymbiont genomes was a surprise when we started, so much so that we didn't believe our initial results. For the aphid-*Buchnera* case, the genome size of the *Buchnera* symbiont had been predicted to be seven times the size of the *E. coli* genome, when in fact it was only one-seventh the size!

When a symbiont becomes integrated with a host so closely, the result is something like a totally new creature, a fusion of the host and symbiont, that now has capabilities not present in either one alone.

You've discovered that sometimes symbiotic relationships are more complex than simply host and symbiont. Can you give us an example?

Aphids have symbiotic bacteria called *Hamiltonella defensa* that protect them against wasps. But the bacteria are only protective if they're infected by a certain virus. So the virus, within the symbiont, within the aphid, is protecting all of them. It's a multilevel system; all three benefit if the aphid is resistant to the wasp.

I think I am drawn to the complexity of these things that come about during evolution. They look so complicated and elaborate, but you can understand them if you think about how selection acted at different levels.

The term symbiosis generally refers to a helpful relationship between two species. But you caution that it can have a downside.

Symbionts aren't always good. The evolutionary interests of bacteria can be in agreement with those of the host, or against them. Symbiotic bacteria might evolve to benefit the host so that the host will live longer (and continue providing a home for the bacteria). Or the bacteria might kill the host, so that the host corpse will disperse microbes. For example,*Photorhabdus luminescens* is a gut symbiont of nematode worms. The nematodes colonize an insect, and the *P. luminescens* exits the nematodes, infects the insect, and kills it. Then the nematodes take up the bacteria and travel to a new host.

In the last few years, you've moved from studying aphids and endosymbionts to bees and their microbes. What inspired the change?

It's very difficult to perform experiments on endosymbionts because the organisms need them to survive. You can't remove the symbiont and see what happens.

What drew you to bees?

Bees are social insects, which gives microbes the opportunity to be transferred from animal to animal. In this way, the bee microbiome is a lot like the human microbiome.

How can the bee microbiome help us understand the human version?

Different bee colonies have different strains with different gene collections, just as people have their own unique collection of microbes.

In human microbiome studies, the links between the microbiome and health are correlative. We rarely have causative data. In bees, we can do more direct experiments. We can do something to the colony and see if it thrives or fails. For example, we isolate pupae in the lab and inoculate the emerging adult bees with specific bacteria. It's a simpler system but still complex.

What do you hope to learn about bee health?

Clean bees, those with no microbes, may be worse at dealing with environmental challenges, such as food shortages, stress and pathogens. There's some evidence that certain bacterial strains can protect honeybees against an RNA virus that is the species' most common and deadly pathogen. The virus is widespread in bees, and it kills some colonies but seems innocuous in others. Why? It probably has to do with the microbiome and how resilient the colony is.

Will your work identify potential causes of colony collapse disorder?

It's only speculation at this point. But you can imagine that a naturally occurring bee colony has little exposure to other colonies. A microbe will survive only if its host colony survives. But commercial bees are raised closer together than in the wild, so there's more opportunity for microbes to spread among colonies. If you take a lot of colonies and put them a few feet apart, you could create conditions where there's greater advantage [from the microbe's perspective] to invading other colonies rather than relying on a single host. That could select for bacteria that are harmful to the colony — for example those that cause the bees to develop diarrhea and spread the microbe. Modeling studies based on human pathogens suggest that lots of social contact could create more-harmful microbes.

Over the last 30 years, symbiosis has transformed from an unpopular area of research to a trendy topic — the human microbiome is the subject of thousands of publications. What's missing from the latest studies?

Nowadays, we see a lot of headlines that assume a diverse microbiota is good. The general tone, even among biologists — except for evolutionary biologists — is that it's all beneficial. But diversity can be good or bad. An

evolutionary perspective might provide some balance. We need to understand evolutionary processes at different levels, such as between a host and its microbes, and among different kinds of bacteria living inside the host.

Scientific American October 5, 2015

http://www.scientificamerican.com/article/bee-symbiosis-reveals-life-s-deepest-partnerships-q-a/

■ Glossary of essential terms for you to know

№	English term	Russian equivalent
1.	bee	пчела
2.	evolve	развиваться, эволюционировать
3.	hive	улей
4.	resilience	устойчивость
5.	symbiosis	симбиоз
6.	myriad	мириады, бесчисленное количество
7.	host	хозяин
8.	eventually	в конечном счете
9.	gut	кишка, внутренности
10.	fringe	край, грань
11.	aphid	тля
12.	wasp	оса
13.	pattern	узор, структура, модель
14.	plague	чума
15.	amplify	усилить
16.	widespread	распространенный
17.	digest	переваривать, усваивать
18.	endosymbiont	эндосимбионт
19.	response	ответ, реакция
20.	innocuous	безобидный, безвредный
21.	tsetse fly	муха цеце
22.	sap	сок
23.	essential	незаменимые
24.	genomics	геномика
25.	tininess	миниатюрность
26.	creature	творение, создание
27.	fusion	слияние
28.	infect	заражать

29.	multilevel	многоуровневый
30.	elaborate	сложный
31.	selection	выбор, отбор
32.	act	действовать
33.	downside	обратная сторона
34.	strain	штамм
35.	pathogen	патогенный микроорганизм
36.	innocuous	безобидный, безвредный
37.	pupae	куколки
38.	thrive	процветать
40.	delve	вникать, копать

■ Your Essential Assignments

I. Quick check :

1. What is symbiosis?
2. What is the name of a microbe, which lives within aphids?

II. Fill in the gaps with the words and expression from the text:

1. Bees and their microbiota are just one example of symbiosis — a close …between two species that typically helps both.
2. The myriad microbes that live within our …help us …certain foods.
3. Many …have become completely dependent on each other, some even …genes.
4. Aphids basically wouldn't exist …symbionts.
5. The …fly, which feeds on blood, has a symbiont that makes B vitamins.
6. Endosymbionts often …lots of genes.
7. The evolutionary interests of …can be in agreement with those of the host, or …them.
8. Bees are …insects, which gives microbes the opportunity to be …from animal to animal. In this way, the bee microbiome is a lot like the human microbiome.

III. Suggest Russian equivalents for the following word combinations:

№	English term	Russian equivalent
1.	large social groups	
2.	resilience to infection	
3.	the myriad microbes	

4.	deeper kind of partnership	
5.	completely dependent on each other	
6.	mysterious plague	
7.	most symbionts	
8.	limited tools	
9.	evolutionary tree	
10.	quite widespread	
11.	multilevel system	
12.	different strains	

IV. Answer the following questions. Use all information given before:

1. What forms of symbiosis can you name?
2. Why do microbes live in the humans' guts?
3. What is Moran's research centered on?
4. What lice cannot survive without a microbe called Bunchera aphidicola?
5. What similar work is Moran doing at the moment?
6. Whom her research could help to?
7. What does Moran hope for in the future?
8. Why do major groups of insects have evolved?

V. Find English equivalents for the following word combinations:

№	Russian term	English equivalent
1.	Тесная взаимосвязь	
2.	Определенные продукты	
3.	Различные формы	
4.	Аналогичная работа	
5.	Таинственная чума	
6.	Эволюционный биолог	
7.	Горячая тема	
8.	Коллекция микробов	
9.	Основные группы	
10.	Сок растения	
11.	Необычные закономерности	
12.	Незаменимые аминокислоты	
13.	Первоначальные результаты	
14.	Новое творение	

15.	Полезные отношения	
16.	Симбиотические бактерии	
17.	Прямые эксперименты	

VI. Translate into English using all the active possible:

1. Пчелы и их микрофлора являются лишь одним из примеров симбиоза — тесной взаимосвязи между двумя видами, которая обычно помогает обоим.
2. Мириады микробов, которые живут в наших кишках, помогают нам переваривать определенные продукты.
3. Тля в принципе не может существовать без симбионтов.
4. Основные группы насекомых эволюционировали, потому что они были симбионтами.
5. С помощью геномики, мы нашли точные гены, необходимые для принятия питательных веществ, таких как аминокислоты или витамины.
6. Тля имеет симбиотические бактерии, которые называются Hamiltonella- защитник, они защищают их от осы.
7. Термин симбиоз в целом относится к полезным отношениям между двумя видами.

VII. Use a monolingual English dictionary and write down what could the words given below mean:

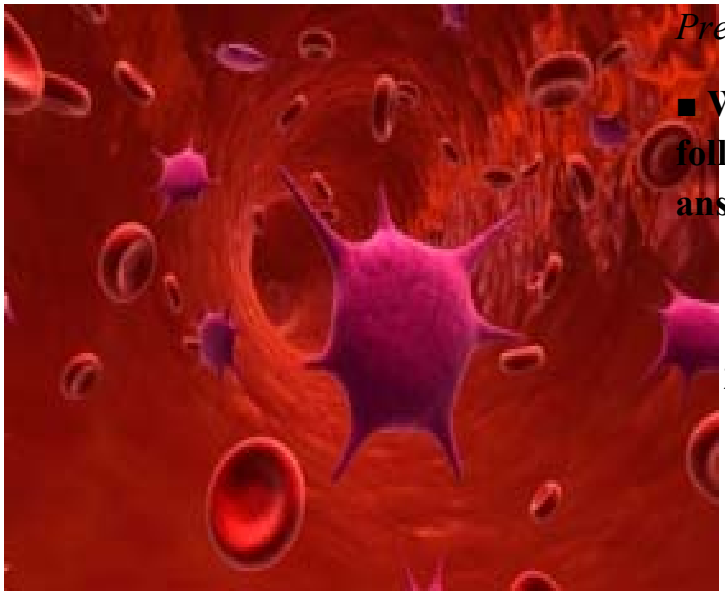
Symbiosis, endosymbionts, bee , tininess, creature, strain, research, evolution.

VIII. Food for thought:

Imagine that scientists have learned to instill any bacteria to man. How do you think can we improve health and human's resistance to infections?

Text 7. Disguised Nanoparticles Slip Past Body's Immune Defense

Drug-delivery systems coated in platelets repair damaged blood vessels



Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them.**

1. What do you know about immune system?
2. What is the role of the immune particles?

■ **Read the given text and make your essential assignments:**

Researchers say that they have found a way to smuggle drug-carrying nanoparticles past the body's immune system: by camouflaging them to look like cell fragments found in human blood.

Man-made nanoparticles — created from plastic or metal — can be designed to deliver a cargo of drugs to specific areas of the body. But they are often attacked and swallowed up by the body's natural defence system, which sees them as foreign invaders.

The disguised particles are not only able to evade detection, but also exploit the natural properties of platelets to treat bacterial infections and to repair damaged blood vessels more effectively than conventional ways of delivering drugs, report the team. The researchers were led by Liangfang Zhang at the University of California, San Diego, and published their work in *Nature* on September 16.

Zhang's team began with 100-nanometer-wide particles made of the biodegradable polymer PLGA, and coated them in membranes taken from human platelets — cell fragments found in the blood that accumulate at sites of tissue damage and begin the clotting process. This helps the particles to evade the immune system, the authors say.

Researchers have previously tried to attach key parts of platelet membranes onto nanoparticles to avoid immune attack; in particular, the platelet's CD47 protein. That protein sends out a 'don't eat me' signal to the body's immune system, says Dennis Discher, a nanoengineer at the University of Pennsylvania in Philadelphia. But Zhang's nanoparticles boast the most complete set of membrane proteins yet, says OmidFarokhzad, a physician and nanotechnologist at the Brigham and Women's Hospital in Boston, Massachusetts, who wrote a News & Views article that accompanied the paper.

Cloaked assassins

The platelet-coated nanoparticles have other advantages. Bacteria such as methicillin-resistant*Staphylococcus aureus* (MRSA), for instance, can stick to platelets — a feature they exploit to protect themselves from the immune system. This makes them naturally more likely to interact with coated nanoparticles. Platelets are also attracted to specific areas of the body where tissue damage is occurring.

The particles harness platelets' unique natural abilities, says Samir Mitragotri, a chemical engineer at the University of California, Santa Barbara, who was not involved in the work. "This a highly innovative approach," he adds.

Zhang's team injected cloaked nanoparticles — with antibiotics inside — into mice infected with MRSA. This reduced MRSA bacteria populations in the liver and spleen by 1,000 times compared to when mice were given conventional antibiotics, and required just one-sixth of the conventional drug dose. (In other organs nanoparticles were also more effective than conventional drug delivery, but the difference was less pronounced).

The team also exploited the fact that platelets tend to migrate to damaged blood vessels. They loaded camouflaged nanoparticles with a drug called docetaxel, to see if it could prevent the excess thickening of damaged artery walls (an effect that can cause problems after surgery). When these nanoparticles were injected into rats that had damaged blood vessels, the particles clustered in larger concentrations at the damaged sites than in the rats' healthy tissue. And the docetaxel treatment was more effective when delivered this way than when it was delivered into the blood stream without using nanoparticles, the team showed.

The ability to deliver high drug doses to these sites while avoiding immune-system cells called macrophages, which usually destroy most nanoparticles even at disease sites, is impressive, says Discher.

Question marks

But not everyone is convinced about the particles' cloaking ability. Although a small fraction of the particles clustered at sites of disease, the vast majority of them quickly ended up in the animals' liver and spleen — suggesting that the

majority of particles were still being caught by immune defenses in those locations, says MoeinMoghimi, a specialist in nanotechnology pharmaceuticals at the University of Copenhagen. Moghimi thinks that a much more stringent examination of the body's immune response to the particles is needed.

Zhang says that his team next plans to make larger amounts of the cloaked nanoparticles, and to test their use in larger animals before therapies could begin trials in humans. Because platelets tend to cluster around cancer cells in the blood, as well as around bacteria, the team will next see whether cloaked nanoparticles could be used to target cancer, he adds.

Developing therapies from hybrid nanoparticles that combine synthetic and biological components will be a long and bumpy road, says Farokhzad. "But is this a technology I would bet on? Absolutely. I think the promise is huge."

Scientific American September 16, 2015
http://www.scientificamerican.com/article/disguised-nanoparticles-slip-past-body-s-immune-defense/

■ Glossary of essential terms

№	English term	Russian equivalent
1	nanoparticles	наночастицы
2	smuggle	переправить
3	to camouflage	маскировать, скрывать
4	natural properties	натуральные свойства
5	body's natural defense system	естественная система защиты организма
6	unique natural abilities	уникальные природные способности
7	swallow	проглотить
8	invader	захватчик
9	blood vessels	кровеносные сосуды
10	exploit	использовать
11	the biodegradable polymer	биоразлагаемый полимер
12	hybrid nanoparticles	гибридные наночастицы
13	tissue	ткань,
14	can stick to platelets	может присоединяться к тромбоцитам
15	conventional drug delivery	обычная доставка лекарств
16	to coat	покрывать
17	platelets	тромбоциты
18	boast	хвалиться

19	resistant	стойкий, сопротивляющийся
20	occur	происходят
22	harness	жгут
23	conventional	обычный
24	spleen	селезенка
25	liver	печень
26	thickening	утолщение, уплотнение
27	cluster	кластер
28	vast	обширные
29	tend	как правило
30	bumpy	ухабистая

■ Your Essential Assignments

I. Use a monolingual English dictionary and give the definitions of the words below:

Antibiotic, engineer, harness, abilities, chemical, nanoparticles

II. Fill in the gaps with the words and expressions from the text:

1. The platelet-coated nanoparticles ……
2. Platelets are ……..of the body where tissue damage is occurring.
3. The team also exploited the fact ……to damaged blood vessels
4. Moghimi thinks ……to the particles is needed.

III. Suggest Russian equivalents for the following word combinations:

1	created from plastic or metal	
2	unique natural abilities	
3	damaged blood vessels	
4	to treat bacterial infections	
5	specialist in nanotechnology pharmaceuticals	
6	to test their use in larger animals	
7	combine synthetic and biological components	
8	destroy most nanoparticles	

IV. Make a sentence with the words:

Paper, membrane, proteins, nanotechnologist, antibiotics.

V. Find English equivalents for the following word combinations:

	Russian terms	English equivalents
1	замаскированные частицы	
2	поврежденные кровеносные сосуды	
3	избежать частиц иммунной системы	
4	взятых из человеческих тромбоцитов	
5	процесс свертывания	
6	инновационный подход	
7	утолщение стенок артерий	
8	наночастицы замаскированные лекарством	
9	техногенные наночастицы	

VI.Find synonyms among the pool of words

Pool of words	Synonyms
1Natural/2alone / 3only/ 4native	
1Supply, /2 process/ 3operation /4deliver	
1Effective/ 2vessels /3 efficient / 4water-craft	
1Large/2 big/3 commonly/4 usually	

VII. Match the sentence halves. Make complete sentences:

1	Researchers have previously tried to attach key parts of platelet membranes onto nanoparticles	A	which usually destroy most nanoparticles even at disease sites
2	The ability to deliver high drug doses to these sites while avoiding immune-system cells called macrophages	B	to evade the immune system, the authors say.
3	The disguised particles are not only able to evade detection, but also exploit the natural properties of	C	to avoid immune attack; in particular, the platelet’s CD47 protein
4	This helps the particles	D	in larger concentrations at the damaged sites than in the rats' healthy tissue.
5	The team also exploited the fact that platelets	E	that his team next plans to make larger amounts of the cloaked nanoparticles,

			and to test their use in larger animals before therapies could begin trials in humans
6	When these nanoparticles were injected into rats that had damaged blood vessels, the particles clustered	F	tend to migrate to damaged blood vessels.
7	Zhang says	G	to treat bacterial infections and to repair damaged blood vessels more effectively than conventional ways of delivering drugs, report the team.

VIII. Answer the following questions.

1) What are the advantages of nanoparticles coated platelets?

2) How did Zhang's team use the ability of platelets migrate to the damaged blood vessels?

3) What man-made nanoparticle materials are made?

4) Why did the researchers tryto attach a key part of the platelet membrane to nanoparticles?

5) What are the plans of the Zhang's team?

IX.Fill in the missing words:

Term(verb)	Noun
attack	
swallow	
evade	
exploit	
repair	
attach	
deliver	

Text 8. Where Could the First CRISPR Baby Be Born?

A look at the legal landscape suggests where human genome editing might be used in research or reproduction

Pre – reading

■ **Work in pairs. Discuss the following questions and try to answer them. Then quickly scan the text to check your answers.**

1. What is genetic engineering?
2. What is an embryo?
3. Which country is already doing experiments with editing the genome of human embryos?

■ **Read the given text and make your essential assignments:**

CRISPR/Cas9 (Clustered Regularly Interspaced Short Palindromic Repeats / CRISPR associated protein 9), which has brought unprecedented ease and precision to genetic engineering, could be used to manipulate the DNA of embryos in a dish to learn about the earliest stages of human development.

They are meeting in China; they are meeting in the United Kingdom; and they met in the United States last week. Around the world, scientists are gathering to discuss the promise and perils of editing the genome of a human embryo. Should it be allowed—and if so, under what circumstances?

The meetings have been prompted by an explosion of interest in the powerful technology known as CRISPR/Cas9, which has brought unprecedented ease and precision to genetic engineering. This tool, and others like it, could be used to manipulate the DNA of embryos in a dish to learn about the earliest stages of human development. In theory, genome editing could also be used to 'fix' the mutations responsible for heritable human diseases. If done in embryos, this could prevent such diseases from being passed on.

The prospects have prompted widespread concern and discussion among scientists, ethicists and patients. Fears loom that if genome editing becomes acceptable in the clinic to stave off disease, it will inevitably come to be used to

introduce, enhance or eliminate traits for non-medical reasons. Ethicists are concerned that unequal access to such technologies could lead to genetic classism. And targeted changes to a person's genome would be passed on for generations, through the germ line (sperm and eggs), fuelling fears that embryo editing could have lasting, unintended consequences.

Adding to these concerns, the regulations in many countries have not kept pace with the science.

Nature has tried to capture a snapshot of the legal landscape by querying experts and government agencies in 12 countries with histories of well-funded biological research. The responses reveal a wide range of approaches. In some countries, experimenting with human embryos at all would be a criminal offence, whereas in others, almost anything would be permissible.

Concerns over the manipulation of human embryos are nothing new. Rosario Isasi, a legal scholar at McGill University in Montreal, Canada, points to two key waves of legislation over the years: one sparked by concerns about the derivation of embryonic stem cells, which was largely deemed acceptable; the other about reproductive cloning, which was largely prohibited for safety reasons.

The current regulatory mosaic is their legacy. Tetsuya Ishii, a bioethicist at Hokkaido University in Sapporo, Japan, spent nearly a year analysing relevant legislation and guidelines in 39 countries, and found that 29 have rules that could be interpreted as restricting genome editing for clinical use (M. Araki and T. Ishii Reprod. Biol. Endocrinol. 12, 108; 2014). But the 'bans' in several of these countries — including Japan, China and India—are not legally binding. “The truth is, we have guidelines but some people never follow them,” said Qi Zhou, a developmental biologist at the Chinese Academy of Sciences Institute of Zoology in Beijing, at a meeting hosted by the US National Academy of Sciences in Washington DC last week. Ishii considers the rules in nine other countries—among them Russia and Argentina—to be “ambiguous”. The United States, he notes, prohibits federal funding for research involving human embryos, and would probably require regulatory approval for human gene editing, but does not officially ban the use of the technique in the clinic. In countries where clinical use is banned, such as France and Australia, research is usually allowed as long as it meets certain restrictions and does not attempt to generate a live birth (see 'CRISPR embryos and the law').

Many researchers long for international guidelines that, even if not enforceable, could guide national lawmakers. Developing such a framework is one of the aims of ongoing discussions; the US National Academy, for example, plans to hold an international summit in December and then produce recommendations for responsible use of the technique in 2016.

But the research has already begun, and more is coming. Scientists in China announced in April that they had used CRISPR to alter the genomes of human embryos, albeit ones incapable of producing a live baby (P. Liang et al. Protein Cell 6, 363–372; 2015). Xiao-Jiang Li, a neuroscientist at Emory University in Atlanta, Georgia, who has used the technique in monkeys, says he has heard rumours that several other Chinese laboratories are already doing such experiments. And in September, developmental biologist Kathy Niakan of the Francis Crick Institute in London applied to the UK Human Fertilisation and Embryology Authority for permission to use the technique to study errors in embryo development that can contribute to infertility and miscarriage. No one so far has declared an interest in producing live babies with edited genomes, and initial experiments would suggest that it is not yet safe. But some suspect that it is only a matter of time.

Ishii predicts that countries with high rates of in vitro fertilization will be the first to attempt clinical applications. Japan, he says, has one of the highest numbers of fertility clinics in the world, and has no enforceable rules on germline modification. The same is true for India.

Guoping Feng, a neuroscientist at the Massachusetts Institute of Technology in Cambridge, hopes that with improvement, the technique could eventually be used to prevent genetic disease. But he argues that it is much too soon to be trying it in the clinic. “Now is not the time to do human-embryo manipulation,” he says. “If we do the wrong thing, we can send the wrong message to the public—and then the public will not support scientific research anymore.”

Scientific American October 14, 2015

http://www.scientificamerican.com/article/where-could-the-first-crispr-baby-be-born/?

■ Glossary of essential terms

№	English term	Russian equivalent
1	ease	легкость, простота
2	precision	точность
3	guidelines	принципы
4	promise	перспектива
5	ambiguous	неоднозначно
6	to prohibit	запрещать
7	dish	пробирка
8	reveal	раскрывать

9	manipulate	управлять
10	circumstances	обстоятельства
11	unintented	непреднамеренный, непредусмотренный
12	prompt	побуждать, подсказывать, толкать
13	concern	беспокойство
14	stave off	предотвратить
15	ethicist	специалист по этике
16	targeted	целенаправленный
17	neuroscientist	нейробиолог
18	cloning	клонирование
19	inevitably	неизбежно
20	pass on	передавать, проходить
21	fertilization	оплодотворение
22	infertility	бесплодие
23	miscarriage	выкидыш
24	improvement	улучшение
25	permissible	допустимый
26	offence	правонарушение
27	legislation	законодательство
28	point	указывать, направлять
29	legacy	наследие
30	binding	связующий, обязательный
31	ambiguous	неоднозначно
32	attempt	попытка
33	framework	структура, рамки
34	albeit	хотя и
35	incapable	недееспособный
36	developmental	эволюционный
37	suspect	подозрение
38	permission	разрешение
39	matter	дело, вопрос
40	germline	зародышевая

■ Your Essential Assignments

I. Fill in the missing words:

Term (verb)	Noun	Adjective
edit	.....	.....
gather	.....	.....
allow	.....	.....
prevent	.....	.....
predict	.....	.....
publish	.....	.....
reproduce	…..	……

II. Match these words with their definitions:

1.	embryo	**A.**	change
2.	mutation	**B.**	action outside the law
3.	editing	**C.**	a method which allows to solve the required task
4.	genome	**D.**	the initial stage of development of the animal
5.	offence	**E.**	guardian of hereditary information
6.	generation	**F.**	a place where research is carried out
7.	DNA	**G.**	a sudden change in the DNA
8.	technology	**H.**	a place where health services have
9.	recommendation	**I.**	all children
10.	institute	**J.**	the set of all genes
11.	clinic	**K.**	the pathological process in the organism
12.	disease	**L.**	helpful information

III. Find English equivalents to the following word combinations:

№	Russian term	English equivalent
1.	редактирование генома	
2.	генная инженерия	
3.	управление ДНК	
4.	человеческий эмбрион	
5.	мощная технология	
6.	наследственные заболевания человека	
7.	взрыв интереса	
8.	уголовное преступление	
9.	китайская лаборатория	

10	научные исследования	

IV. Give Russian equivalents to the following English terms:

№	English term	Russian equivalent
1	around the world	
2	stages of development	
3	passed on for generations	
4	study errors	
5	initial experiments	
6	funding for research	
7	enforceable rules	
8	genetic disease	
9	international summit	
10	criminal offence	

V. Find synonyms among the pool of words:

Pool of words	Synonyms
1) 1. editing /2. precision /3. redaction/ 4. accuracy	
2) 1. powerful /2. stage/3. lap /4. strong **3)** 1. target /2. introduce / 3. goal /4. enter	
4) 1. reliable/2. largely /3. responsible/4. mostly	

VI. Answer the following questions. Use all information given before:

1. What is the editing of the genome?
2. What are the experts concerned about?
3. What will you need to edit the genome?
4. Are there countries, where clinical trials are forbidden?
5. When will CRISPR / Cas9 be available to use?
6. What is the human genome editing technology?
7. What is the purpose of using CRISPR/Cas9 technology?
8. What biological object are scientists experimenting on?

VII. Use monolingual English dictionary and write down what could the words given below mean:

Reproduction, offence, mutation, embryo, engineering.

VIII. Read and translate the short text without any dictionary

The functions of CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats) and CRISPR-associated (Cas) genes are essential in adaptive immunity in select bacteria and archaea, enabling the organisms to respond to and eliminate invading genetic material. These repeats were initially discovered in the 1980s in *E. coli* but their function wasn't confirmed until 2007 by Barrangou and colleagues, who demonstrated that *S. thermophilus* can acquire resistance against a bacteriophage by integrating a genome fragment of an infectious virus into its CRISPR locus.

IX. Read and translate the short text without any dictionary:

Американским ученым удалось модифицировать систему редактирования генома CRISPR/Cas9, снизив количество ошибок практически до нуля. Низкая точность была основной преградой к использованию системы на человеке, сообщает сайт N+1 со ссылкой на журнал Science.

Система CRISPR/Cas9 состоит из эндонуклеазы Cas9, которая способна разрезать двухцепочечную молекулу ДНК, и связанной с ней молекулы РНК, которая по принципу комплементарности (*форма взаимодействия в генотипе, приводящая к новому признаку*) позволяет белку найти нужный участок в геноме. Такая система позволяет редактировать определенные участки ДНК, нацеливаясь на них последовательностью направляющей РНК.

Text 9. The Woman Who Stared at Wasps

The biologist Joan Strassmann discusses the evolution of cooperation, how amoebas can teach us about competition, and why the definition of "organism" needs an overhaul

Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them.**

1. Should people be afraid of wasps?

2. What ways do wasps help to improve human's life?

■ **Read the given text and make your essential assignments:**

As an undergraduate in the 1970s, Joan Strassmann split her time between writing short stories and laying siege to the office of her mentor, the sociobiologist Dick Alexander. For two years, she insisted on meeting with him every Friday to discuss research, a schedule that she now thinks was probably quite an imposition. "He would give me so much reading," she recalled, "it would take me forever. I would work night and day to finish it, and maybe this was his strategy—maybe he was hoping I'd cancel or something." But that intense focus became one of the hallmarks of her pioneering research on social insects.

In graduate school at the University of Texas, Austin, Strassmann began to study wasps that live in hierarchical colonies, starting with a nest that was thriving in a tractor shed near the main campus. "I was really planning to work on something else, some other social organism, like ground squirrels," said Strassmann, now at Washington University in St. Louis. "Everyone knew that I was actually terrified of wasps." But on a dare, she and a friend ventured into the shed and painted each wasp with a different identifying dot of paint, the standard preparation for studying the social dynamics of an insect colony. Then she just started watching them. And watching them.

She was in good company. Insects that live in cooperative colonies—ants, termites, and some wasps and bees—have fascinated scientists for more than a century because they pose an evolutionary conundrum. Darwin himself saw their way of life as a challenge to his ideas. The theory of evolution seems to predict that each individual will fight to pass on its traits, but in a colony, only a very small number of insects actually get to reproduce: the queens and their mates. The rest

give up their chance to contribute to the gene pool, caring for the offspring of others instead. How could this lifestyle, known as eusociality, have evolved? How could it make sense for the ancestors of modern worker bees or wasps to give up their autonomy? It seems biologically implausible.

Right around the time Strassmann was in college, however, biologists began to understand how social insects could fit into the framework of evolutionary theory. In a seminal paper, W.D. Hamilton proposed that cooperation might make sense in closely related individuals that share enough genes. If a bee with a maiden-aunt helper produces twice as many offspring as it might have otherwise, that arrangement makes evolutionary sense for the non-reproducing assistant, which is indirectly passing on its genes. But that benefit is reduced as relatedness declines, so eusociality would only arise among close relatives.

Strassmann found clear evidence of Hamilton's idea in her tractor-shed wasps. When a nest is destroyed, its members will disperse to sister nests in a pattern that reflects their level of relationship to the queens. Each wasp serves only the leaders it is most closely related to. Strassmann continued to study wasps over the next 20 years with her husband and collaborator, David Queller, and the two have uncovered many other details of how relatedness shapes the behavior of social insects, including how colonies keep relatedness high when multiple queens reign, and what it takes to turn a worker into a queen.

About 17 years ago, however, the pair began to shift to a new model organism, the amoeba Dictyostelium discoideum. They suspected that this unusual creature could offer new insights into the dynamics of cooperation. In moments of starvation, these soil-dwelling amoebas crowd together and build a tower rising above the ground from which they disperse their spores to other, more hospitable places. Some 20 percent of the group will sacrifice themselves to build the tower with their bodies, while the rest take advantage of it to spread their genes.

Quanta Magazine spoke with Strassmann about the evolution of social insects, the secret lives of queen wasps, and what she's learned about cooperation from a single-celled creature. An edited and condensed version of the interview follows.

QUANTA MAGAZINE: You began to study social wasps just as scientists were debating the origins of eusociality. What was that like?

JOAN STRASSMANN: It was a really exciting time. W.D. Hamilton's paper came out in 1964, but it wasn't really appreciated until the early '70s, when I was in college. He outlined the framework of something called inclusive fitness, which is the sum of the effects of an individual's actions on its own and others' reproduction. Ultimately, it is the measure of an individual's actions on the representation of its genes in the next generation.

You gave up wasps for a single-celled creature. That's a huge jump. Most people don't change model organisms midcareer.

It was kind of an insane thing to do. Especially since we had been studying wasps in Tuscany. We know Italian, we've lectured in Italian, we have a lot of really good friends there … yeah. It's insane!

The amoebas must have been doing something really amazing for you to change.

All social-insect people know about Dictyostelium because it has a solitary stage and a social stage. The single cells eat bacteria by engulfment, they live in soil, and so on. But when they starve, they aggregate and form a multicellular body that crawls toward light and ultimately coalesces into a structure called a fruiting body. About 20 percent of the cells form a stalk. The others flow up to the top and form spores, which are then dispersed. Conceptually, this can be viewed as similar to a social-insect colony. The stalk is the workers—they don't get to reproduce, but we think they enhance dispersal of the spores.

But if the Dictyostelium cells in that multicellular body aren't all genetically identical, are some sacrificing themselves for others with whom they have nothing in common? You know how I told you the only way groups evolve eusociality is by daughters' staying with their mothers? The idea that you would get eusociality from aggregation seems really wrong. There should be too many conflicts there. Who's going to die, who's going to become the spores, and so on. There were some obvious questions to ask.

In 1998, scientists had sequenced the Dictyostelium genome but hadn't published it yet. We knew we'd be able to plunder it for relatedness analysis. Dave and I just reached the point where we felt, "Wow, we've thought of this—we are going to feel like damn fools if somebody does this instead of us."

So what did you do?

I got on the Internet and discovered there was a Dictyostelium email list. I just started posting questions. I wanted to know whether two different strains would still form a fruiting body together.

The first study we did, we found that different strains do mix, and they do cheat. Two different strains will not contribute equally to spore and stalks. One of them is taking advantage of the other. So this is a good way to look at social organisms.

Why does that make them a good model?

Because there's conflict—social competition.

How conflicts are controlled in social organisms is a major topic of evolution research. In eusocial organisms, controlling conflict—through castes, for

instance, which make it very clear who gives way to whom—allows them to live together.

In Dicty, where some take advantage of others, we can ask questions about how they manage the conflict so as to allow this form of sociality to persist.

What made Dicty really attractive was that we could ask "why?" on a mechanistic level, because we could knock out genes and do all those things that cell biologists do. That was super-exciting to us. It's just been fabulous. We've found that there are over 100 genes that, if inactivated, will cause a Dicty strain to begin to cheat, for instance. That suggests that the genetics of sociality in Dicty is complex.

And we're moving into bacteria now. We found some insane relationships between bacteria and Dicty: For instance, Dictyostelium actually appear to farm bacteria for their own consumption! So we're not working on wasps at all. It's sad but true.

Now you're trying to define what it means to be an organism?

We feel that defining the actual key characteristics that make an organism has never been properly done. People have proposed requirements "genetic uniformity," "all connected together" and "single-cell bottlenecks"—that means that there's a phase where the organism is just a single cell.

None of them really hold up—plants don't have single-cell bottlenecks, for instance. Working on Dicty, you realize how narrow-minded most biologists are. They are so blinded by a very few organisms. If you want to think about what an organism is, you can't start with a mouse. You can't! You have to start with, well, is a biofilm an organism? If it is, why? If not, why not?

What Dave and I came up with—and this is mostly Dave—is that what makes an organism is the level at which there is the highest level of cooperation and the lowest level of conflict. If the parts, whatever they are, are largely cooperative and whatever conflict there was has been controlled, that's an organism. Our plan is to write a book on this.

My favorite example is the Portuguese man-of-war. They say it's not an organism, that it's a colony of lots of little organisms. No—it's an organism! I'm sorry!

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http://www.scientificamerican.com/article/the-woman-who-stared-at-wasps/

■ Glossary of essential terms

№	English term	Russian equivalent
1.	laying siege	осаждать, отправлять
2.	mentor	наставник
3.	overhaul	капитальный ремонт
4.	hallmark	фирменная марка, проба, отличительный признак
5.	consumption	потребление
6.	nest	гнездо
7.	squirrel	белка
8.	a dare	спор, вызов
9.	fascinate	очаровывать
10.	implausible	неправдоподобный
11.	framework	рамки, структура
12.	benefit	выгода
13.	destroy	разрушать
14.	disperse	рассеиваться
15.	uncovered	раскрывать, снимать
16.	relatedness	связанность
17.	starvation	голодание
18.	sacrifice	жертвовать
19.	engulfment	поглощение
20.	colundrum	загадка, головоломка
21.	advantage	преимущество
22.	fabulous	невероятный, сказочный
23.	trait	черта, особенность
24.	appear	появиться
25.	venture	предприятие
26.	Portuguese man-of-war	Португальский кораблик
27.	wasp	оса
28.	arrangement	договоренность
29.	venture	предок
30.	maiden-aunt	девичья бабка
31.	shed	сарай
32.	decline	снижение
33.	collaborator	сотрудник, соратник

34.	reign	царствовать
35.	soil-dwelling	почвенные
36.	appreciate	ценить
37.	midcareer	в середине карьеры
38.	insane	ненормальный
39.	solitary	одинокий
40.	coalesces	срастается
41.	crawl	ползать
42.	fruiting body	плодовые тела
43.	plunder	грабеж
44.	strain	напряжение
45.	persist	сохраняться

■ Your Essential Assignments

I. Fill in the missing words:

Term (verb)	Noun
employ	.....
exist	.....
prevent	.....
evolve	.....
support	.....
separate	.....

II. Use monolingual English dictionary and write down what could the words given below mean:

Benefit; bacteria; evolved; termites, wasp, advantage.

III. Read and translate the short text without any dictionary.

It is unlikely there will be a being more simply than an amoeba, after all it only the thin external cover filled with watery liquid, and in it — the kernel containing genetic material. Amoebas have no constant form, but is before and the back, and they move, contracting and pushing out itself in the direction of food. They eat, enveloping smaller parts of seaweed and bacteria and absorbing them, and breed divison.

IV. Match the sentence halves. Make complete sentences:

1.	I was really planning to work on something else, some other social organism,	**A.**	have fascinated scientists for more than a century because they pose an evolutionary conundrum.
2.	Insects that live in cooperative colonies—ants, termites, and some wasps and bees	**B.**	offer new insights into the dynamics of cooperation.
3.	Strassmann found clear evidence	**C.**	but it wasn't really appreciated until the early '70s, when I was in college.
4.	Each wasp serves only the leaders	**D.**	like ground squirrels
5.	They suspected that this unusual creature could	**E.**	that it's a colony of lots of little organisms.
6.	W.D. Hamilton's paper came out in 1964,	**F.**	it is most closely related to.
7.	The first study we did, we found that different strains do mix,	**G.**	and they do cheat.
8.	They say it's not an organism,	**H.**	of Hamilton's idea in her tractor-shed wasps.

V. Find Russian equivalents for the following word combinations

№	English term	Russian equivalent
1.	evolutionary conundrum	
2.	biologically implausible	
3.	multiple queens	
4.	a single-celled creature	
5.	multicellular body	
6.	in addition	
7.	just a single cell	
8.	fruiting body	
9.	social competition	

VI. Answer the following questions. Use all information given before:

1. What insects live in cooperative colonies?
2. Why do some social amoebas sacrifice themselves?
3. What is the inclusive fitness?
4. What stages does Oictyostelium have?
5. What is the fruiting body?

VII. Match words with their definitions.

1.	Schedule	**A.**	To continue to do smth. although it is annoying other people.
2.	Venture	**B.**	When smth. becomes less in amount, importance, quality
3.	Offspring	**C.**	A system of rules, ideas, that used to plan or decide smth.
4.	Framework	**D.**	The child of a person or animal.
5.	Decline	**E.**	A flying insect with thin, black and yellow body.
6.	Wasp	**F.**	To leave a safe place and go somewhere, that may evolve risks.
7.	Persist	**G.**	A list of planned activities or thought to be done.

VIII. Read and translate the short text without any dictionary.

В аспирантуре в Университете Техаса, Остин, Штрассман начала изучать ос, которые живут в иерархических колониях, начиная с гнезд, которые процветали в сарае, недалеко от главного университетского городка. "Я на самом деле планировала работать над чем-то еще, каким-то другим социальным организмом, как белки," сказала Штрассман, теперь уже в Университете Вашингтона в Сент-Луисе. "Все знали, что я была в ужасе от ос." Но на спор, она и ее друг рискнули пойти в сарай и окрасить каждую осу различной выделяющейся краской. Так и началась эта история…

Text 10. Dinosaurs Evolved in a Startlingly Short Time

New fossil dates show beasts arose from their ancestors in half the time researchers previously thought

Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them.**

1. What do you know about dinosaurs?
2. What ages have dinosaurs existed?

■ **Read the given text and make your essential assignments:**

Animals escaping from an erupting volcano 235 million years ago in northwestern Argentina. These species, found as fossils in the Chanares Formation, include early mammal relatives (the dicynodont Dinodontosaurus in the left background, and the cynodont Massetognathus in the left foreground) and early dinosaur precursors (Lewisuchus in the right background, and Lagerpeton in the right foreground). By measuring radioactive isotopes in zircons crystals from the volcanic ash, scientists were able to determine the precise age of this fossil assemblage.

Victor Leshyk

Dinosaurs took less than 5 million years to evolve from their reptile predecessors, the early dinosauromorphs, a new study finds.

The finding revamps the time line between the dinosaurs and early dinosauromorphs. Until now, researchers thought that it took at least 10 million to 15 million years for the early dinosauromorphs to evolve into dinosaurs.

"It really narrows the amount of time between the appearance of these early dinosauromorphs and the first dinosaurs," said study co-researcher Randall Irmis, a paleontologist at the University of Utah and a curator of paleontology at the Natural History Museum of Utah. "Rather than there being 10 [million] or 15 million years between when the first dinosauromorphs show up and the first dinosaurs, now it's just 5 million years."

Early dinosauromorphs were just like dinosaurs, except for a few key features. For instance, dinosaurs had a ball-and-socket hip that could rotate easily, and additional sacral vertebrae (a vertebra at the end of the spine), which helped strengthened the hips. This allowed dinosaurs to develop stronger leg muscles, which, along with their forward-hinging feet, helped them run faster than their competitors. They also developed an extra hole in their skulls, which let them cool off after vigorous activity.

Even though paleontologists had studied these predecessors previously, they still haven't been certain about the age of the rocks containing early dinosauromorph fossils, Irmis said. He and his colleagues gave the matter a closer look, investigating the Chanares Formation in northwestern Argentina, a site known for containing the fossils of early dinosauromorphs and early dinosaurs.

The researchers relied on a handy mineral called zircon to help them date the early dinosauromorph-containing rock layer. When zircon crystals form, they trap the radioactive element uranium within them. Over time, uranium decays into lead.

"We know the exact rate at which uranium decays into lead," Irmis told Live Science. By measuring the ratio of uranium to lead, researchers can determine how long ago the zircon crystal formed.

However, zircon isn't present in all rocks. So the researchers looked for volcanic ash, where the mineral is more commonly found. Luckily, they found zircon crystals in a rock layer that contained early dinosauromorphs. The scientists took a sample from that layer, as well as from the younger layer above it, so they could bookend the finding.

Dating the rock

The researchers crushed the rock samples so they could isolate the zircon crystals, which are as small as grains of sand, Irmis said. Then, the scientists analyzed about 20 zircon crystals from each sample, using a mass spectrometer, an instrument that separates elements and isotopes (a variation of an element) by mass and concentration, the researchers said.

What's more, the zircon crystals contained a helpful cross-check: They have different uranium isotopes that decay at different rates, and "we're fairly confident we've got the right age if they all agree with each other," Irmis said.

The results show that the rock layer is between 234 million and 236 million years old, from the Late Triassic period, he said, meaning the early dinosauromorphs within the layer are the same age. This new date is 5 million to 10 million years younger than previously thought, Irmis said.

Dinosaurs may have evolved rapidly (geologically speaking), but it appears they came to dominate paleo-Earth in a smooth and gradual manner, Irmis said.

That is, they didn't suddenly wrest power from their early dinosauromorph relatives.

"When we look at the ecosystems of [the] first dinosauromorphs and the ecosystems with the first dinosaurs, it's interesting that we don't see much difference in how the ecosystems are put together," Irmis said. "You don't seem to see dinosaurs showing up and immediately taking over."

He added, "it really emphasizes that there wasn't much special about the first dinosaurs. They were pretty similar to their early dinosauromorph relatives and probably doing very similar things."

Dinosaurs move forward

Some dinosauromorphs persisted for another 20 million years after dinosaurs emerged, Irmis said. But the dinosaurs' adaptations appear to have been advantageous in the long run, Irmis said. These changes helped dinosaurs prosper until the 6-mile-long (10 kilometers) asteroid wiped them out 66 million years ago, Irmis said.

But dinosaurs took a while to spread throughout the world, the researchers note. The dinosaurs didn't dominate the mid to high latitudes—such as present-day Argentina, Brazil and South Africa—until the late Triassic, about 215 million years ago. It took dinosaurseven longer to dominate the lower latitudes, such as present-day western and eastern North America, areas that were closer to the equator at that time, Irmis said.

The new research is a "solid study," said Kenneth Lacovara, a professor of paleontology and geology and the dean of the School of Earth & Environment at Rowan University in New Jersey, who wasn't involved with the study.

"The story is that there was a very rapid evolution and a very rapid achievement of dominance in the fauna as they go from [early] dinosauromorphs to dinosaurs," Lacovara said. It shows that "being a dinosaur is a really good idea. It really works. It allows them to outcompete things that aren't like dinosaurs. And if you include birds, being a dinosaur is still a pretty good thing."

The findings were published online today (Dec. 7) in the journal Proceedings of the National Academy of Sciences.

Scientific American December 8, 2015

http://www.scientificamerican.com/article/dinosaurs-evolved-in-a-startlingly-short-time/

■ Glossary of essential terms for you to know

	English term	Russian equivalent
1.	escape	побег

2.	erupt	прорезываться
3.	startlingly	поразительно
4.	fossil	ископаемое
5.	precursor	предвестник
6.	ash	пепел
7.	precise	точный
8.	assemblage	сборище
9.	revamp	реконструкция
10.	ball-and-socket hip	шаровидное бедро
11.	rotate	поворачиваться
12.	sacral	крестцовый
13.	forward-hinging feet	вперед – закрученные ноги
14.	skull	череп
15.	vigorous	бодрый
16.	site	место
17.	layer	слой
18.	amass	копить
19.	persist	упорствовать
20.	emerge	появляться
21.	prosper	преуспевать
22.	latitude	широта
23.	dean	декан
24.	outcompete	вытеснять

■ Your Essential Assignments

I. Fill in the missing words:

Term	Noun
Adapt	
Inherit	
Form	
Differ	
Discover	
Describe	

II. Use monolingual English dictionary and write down what could the words given below mean:

Sample, evolve, similar, research, vigotous.

III. Find synonyms among the pool of words:

Pool of words	Synonyms
1) evolve, develop, narrow, progress, reduce.	
2) finding, competitors, rival, discovery	
3) after, smooth, post, sleek	
4) movement, accept, motion, agree	

IV. Find English equivalents to the following word combinations:

English term	Russian equivalent
To develop into dinosaurs	
The finding revamps the time line	
A handy mineral	
Radioactive isotopes	
Researchers can determine	
The zircon crystal	
Adaptation appears	
Dominate the mid to high latitudes	

V. Find opposites to the given words

Word	Opposite
Evolution	
Future	
Accept	
To survive	
To fail	
Gradually	
To escape	

VI. Give Russian equivalents to the following English terms:

English term	Russian equivalent
Удобный минерал	

Обнаружение обновляет временную линию	
Чтобы превратиться в динозавров	
Доминируют в средних и высоких широтах	
Исследователи могут определить	
Адаптация появляется	
Кристалл циркона	
Радиоактивные изотопы	

VII. Match these words with their definitions

Word	Definition
1. Identical	A. The act or process of producing young animals or plants
2. Evolution	B. To find something that was hidden or that people did not know about before
3. Feature	C. Exactly the same
4. Reproduction	D. Someone's child or children, animal's baby or babies.
5. Existence	E. Something important or typical of a place or thing
6. To discover	F. A substance in general that everything in the world consist of
7. Matter	G. The gradual change and development
8. Offspring	H. The state of existing
9. Evidence	I. Fact that makes you believe that smth. exists or is true.

VIII. Translate into English using all active possible.

1. Динозаврам требовалось время, чтобы распространиться по всему миру, отмечают исследователи.

2. Это действительно сужает количество времени между появлением ранних динозавроморфов и первых динозавров.

3. Это позволило динозаврам развивать сильные мышцы ног.

4. Даже если палеонтологи ранее изучали этих предшественников, они до сих пор не были уверены о возрасте пород, содержащих окаменелости ранних динозавроморфов.

Text 11. Beyond Resveratrol: The Anti-Aging NAD Fad

Pre-reading

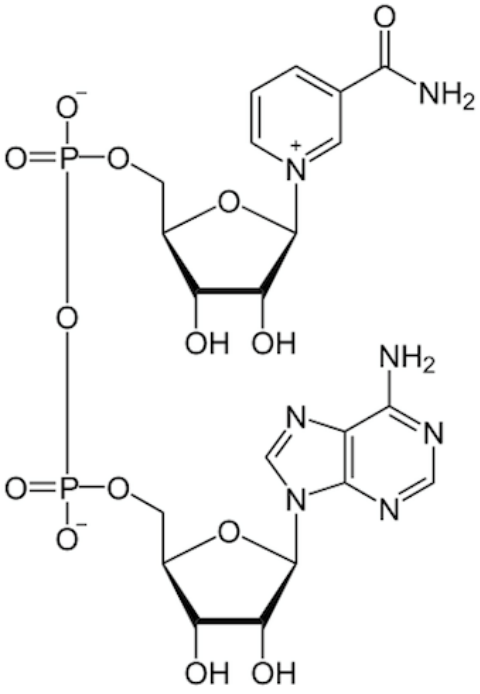
■ **Work in pairs. Discuss the following questions and try to answer them.**

1. What is NAD?
2. What do you know about mitochondria?
3. What causes heart failure?
4. What leads to mitochondrial dysfunction?

■**Read the given text and make your essential assignments:**

Mitochondria are our cells' energy dynamos. Descended from bacteria that colonized other cells about 2 billion years, they get flaky as we age. A prominent theory of aging holds that decaying of mitochondria is a key driver of aging. While it's not clear why our mitochondria fade as we age, evidence suggests that it leads to everything from heart failure to neurodegeneration, as well as the complete absence of zipping around the supper table.

Recent research suggests it may be possible to reverse mitochondrial decay with dietary supplements that increase cellular levels of a molecule called NAD (nicotinamide adenine dinucleotide). But caution is due: While there's promising test-tube data and animal research regarding NAD boosters, no human clinical results on them have been published.



STRUCTURE OF NICOTINAMIDE ADENINE DINUCLEOTIDE, OXIDIZED (NAD+).

NAD is a linchpin of energy metabolism, among other roles, and its diminishing level with age has been implicated in mitochondrial deterioration. Supplements containing nicotinamide riboside, or NR, a precursor to NAD that's found in trace amounts in milk, might be able to boost NAD levels. In support of that idea, half a dozen Nobel laureates and other prominent scientists are working with two small companies offering NR supplements.

The NAD story took off toward the end of 2013 with a high-profile paperby Harvard's David Sinclair and colleagues. Sinclair, recall, achieved fame in the mid-2000s for research on yeast and mice that suggested the red wine ingredient resveratrol mimics anti-aging effects of calorie restriction. This time his lab made headlines by reporting that the mitochondria in muscles of elderly mice were restored to a youthful state after just a week of injections with NMN (nicotinamide mononucleotide), a molecule that naturally occurs in cells.

It should be noted, however, that c strength was not improved in the NMN-treated mice the researchers speculated that one week of treatment wasn't enough to do that despite signs that their age-related mitochondrial deterioration was reversed.

NMN isn't available as a consumer product. But Sinclair's report sparked excitement about NR, which was already on the market as a supplement called Niagen. Niagen's maker, ChromaDex, a publicly traded Irvine, Calif., company, sells it to various retailers, which market it under their own brand names. In the wake of Sinclair's paper, Niagen was hailed in the media as a potential blockbuster.

In early February, Elysium Health, a startup cofounded by Sinclair's former mentor, MIT biologist Lenny Guarente, jumped into the NAD game by unveiling another supplement with NR. Dubbed Basis, it's only offered online by the company. Elysium is taking no chances when it comes to scientific credibility. Its website lists a dream team of advising scientists, including five Nobel laureates and other big names such as the Mayo Clinic's Jim Kirkland, a leader in geroscience, and biotech pioneer Lee Hood. I can't remember a startup with more stars in its firmament.

A few days later, ChromaDex reasserted its first-comer status in the NAD game by announcing that it had conducted a clinical trial demonstrating that a single dose of NR resulted in statistically significant increases in NAD in humans the first evidence that supplements could really boost NAD levels in people. Details of the study won't be out until it's reported in a peer-reviewed journal, the company said. (ChromaDex also brandishes Nobel credentials: Roger Kornberg, a Stanford professor who won the Chemistry prize in 2006, chairs its scientific advisory board. He’s the son of Nobel laureate Arthur Kornberg, who, ChromaDex proudly notes, was among the first scientists to study NR some 60 years ago.)

SIRT1 PROTEIN, RED, CIRCLES THE CELL'S CHROMOSOMES, BLUE.

The NAD findings tie into the ongoing story about enzymes called sirtuins, which Guarente, Sinclair and other researchers have implicated as key players in conferring the longevity and health benefits of calorie restriction. Resveratrol, the

wine ingredient, is thought to rev up one of the sirtuins, SIRT1, which appears to help protect mice on high doses of resveratrol from the ill effects of high-fat diets.

A slew of other health benefits have been attributed to SIRT1 activation in hundreds of studies, including several small human trials.

Here's the NAD connection: In 2000, Guarente's lab reported that NAD fuels the activity of sirtuins, including SIRT1the more NAD there is in cells, the more SIRT1 does beneficial things. One of those things is to induce formation of new mitochondria. NAD can also activate another sirtuin, SIRT3, which is thought to keep mitochondria running smoothly.

The Sinclair group's NAD paper drew attention partly because it showed a novel way that NAD and sirtuins work together. The researchers discovered that cells' nuclei send signals to mitochondria that are needed to maintain their normal operation. SIRT1 helps insure the signals get through. When NAD levels drop, as they do with aging, SIRT1 activity falls off, which in turn makes the crucial signals fade, leading to mitochondrial dysfunction and all the ill effects that go with it.

NAD boosters might work synergistically with supplements like resveratrol to help reinvigorate mitochondria and ward off diseases of aging. Elysium is banking on this potential synergyits NR-containing supplement includes a resveratrol-like substance called pterostilbene (pronounced tero-STILL-bean), which is found in blueberries and grapes.

Why pterostilbene instead of resveratrol?

While resveratrol has hogged the anti-aging spotlight over the past decade, unsung researchers in places like Oxford, Miss., have quietly shown that pterostilbene is a kind of extra-potent version of resveratrol. The pterostilbene molecule is nearly identical to resveratrol's except for a couple of differences that make it more "bioavailable" (animal studies indicate that about four times as much ingested pterostilbene gets into the bloodstream as resveratrol). Test-tube and rodent studies also suggest that pterostilbene is more potent than resveratrol when it comes to improving brain function, warding off various kinds of cancer and preventing heart disease.

Scientific American March 11, 2015

http://blogs.scientificamerican.com/guest-blog/beyond-resveratrol-the-anti-aging-nad-fad/

■ Glossary of essential terms

	English term	Russian term
1	flake	расслаиваться, шелушиться
2	dietary supplements	пищевые добавки

3	boosters	ускорители
4	diminishing	уменьшающийся
5	deterioration	ухудшение
6	precursor	предшественник
7	yeast	дрожжи
8	decay	распад
9	fade	увядать
10	to zip	сжимать
11	to induce	побудить
12	to rev up	для увеличения скорости
13	synergistically	синергично, автономно
14	reinvigorate	активизировать
15	ward off	отвратить
16	speculate	предполагать
17	extra-potent version	очень мощная версия
18	ingested	поглощенный
19	test-tube	пробирка
20	nickels and dimes	червонцы и пятаки
21	drag off	утащить
22	astonishing	удивительный
23	to be hailed	быть расценена
24	credibility	правдоподобие
25	oxidized	окисленный
26	prominent	видный
27	biotech	биотехнологии
28	blueberries	черника
29	grapes	виноград
30	to protect	защищать
31	longevity	долговечность
32	peer-reviewed	рецензируемые
33	brain	головной мозг
34	buzz about	шуметь по поводу
35	sidewalk	тротуар
36	mentor	наставник
37	preventing	предотвращение
38	evidence	доказательства

■ Your Essential Assignments

I. Quick check

1. What is the difference between a NAD and a NR?
2. What is the core energy of metabolism?

II. Find synonyms among the pool of words

Pool of words	Synonyms
1.protect/2.mentor/3.defend/4. tutor/5. coacher	
1.formation/2.deterioration/3.degradation/4.organization	
1. announce2 opportunity /3. .report /4.chance	
1. publicly/2 openly/3.researcher/4.explorer	

III. Fill in the gaps with the words and expressions from the text

1. The researchers discovered that __ __ send signals to mitochondria that are needed to maintain their normal operation.
2. A slew of other __ __ have been attributed to SIRT1 activation in hundreds of studies, including several small human trials.
3. A prominent theory of aging holds that __ __ __ is a key driver of aging.
4. Elysium isn't the only __ vendor.
5. The scientists reportedly characterized NR's __ __ __ as "nothing short of astonishing."
6. Details of the study won't be out until it's reported in __ __ __, the company said.
7. Test-tube and __ __ also suggest that pterostilbene is more potent than resveratrol when it comes to improving __ __, warding off various kinds __ __ and preventing heart disease.
8. Even before Sinclair's paper, researchers had shown in 2012 that when given doses of NR, __ on high-fat diets gained 60 percent __ __ than they did on the same diets without NR.
9. When NAD levels drop, as they do with aging, SIRT1 __ __ off, which in turn makes the __ __ fade, leading to __ __ and all the ill effects that go with it.
10. Besides, it probably won't be __ __ more data come out given the growing buzz __ __.

IV. Fill in the missing words:

Term	Noun	Adjective
connect		

find		
colonize		
report		

V. Use monolingual English dictionary and write down what could words given bellow mean:

diet, supplement, booster, ward, to hail, injection.

VI. Suggest Russian equivalents for the following word combinations

№	English term	Russian equivalent
1	Mitochondria are our cells' energy dynamos	
2	Human clinical results on them have been published	
3	NAD can also activate another sirtuin	
4	SIRT1 activity falls off	
5	SIRT1 helps insure the signals get through	
6	Resveratrol, the wine ingredient, is thought to rev up one of the sirtuins	
7	Further, none of the mice on NR showed signs of diabetes	
8	Ward off diseases of aging	

VII. Read and translate the short text without any dictionary:

Elysium isn't the only pterostilbene vendor. In fact, ChromaDex also offers pterostilbene for supplements separately from Niagen.

How excited should we be about all this? If I were a middle-aged mouse, I'd be ready to spend some of the nickels and dimes I'd dragged off the sidewalk to try NR supplements. Even before Sinclair's paper, researchers had shown in 2012 that when given doses of NR, mice on high-fat diets gained 60 percent less weight than they did on the same diets without NR. Further, none of the mice on NR showed signs of diabetes, and their energy levels improved. The scientists reportedly characterized NR's effects on metabolism as "nothing short of astonishing."

But the paucity of human data gives me pause. Nobel laureates notwithstanding, I plan to wait until more is known before jumping up from the supper table to run out for some NR. Besides, it probably won’t be long before more data come out given the growing buzz about NAD.

Text 12. CRISPR tweak may help gene-edited crops by pass biosafety regulation

Pre-reading

■ **Work in pairs. Discuss the following questions and try to answer them.**

1. What do you know about biosafety regulation?
2. Do you think that CRISPR can help gene-editing crops?

Read the given text and make your essential assignments:

A twist on a revolutionary gene-editing technique may make it possible to modify plant genomes while sidestepping national biosafety regulations, South Korean researchers say.Plant scientists have been quick to experiment with the popular CRISPR/Cas9 technique, which uses an enzyme called Cas9, guided by two RNA strands, to precisely cut segments of DNA in a genome. By disabling specific genes in wheat and rice, for example, researchers hope to make disease-resistant strains of the crops. But the process can introduce bits of foreign DNA into plant genomes. And some jurisdictions, such as the European Union, could decide to classify such plants as genetically modified organisms (GMOs)1 — making their acceptance by regulatory bodies contentious, says geneticist Jin-Soo Kim of Seoul National University.

Kim and his team tweaked the technique so that it can delete specific plant genes without introducing foreign DNA, creating plants that he and his colleagues think “might be exempt from current GMO regulations”.

“In terms of science, our approach is just another improvement in the field of genome editing. However, in terms of regulations and public acceptance, our method could be path-breaking,” says Kim.

DNA-free CRISPR

Conventionally, researchers get CRISPR/Cas9 working in a plant cell by first shuttling in the gene that codes for the Cas9 enzyme. The gene is introduced on a plasmid — a circular packet of DNA — which is usually carried into a plant by the bacterial pest Agrobacterium tumefaciens. As a result, Agrobacterium DNA can end up in the plant’s genome. Even if the pest is not used, fragments of the Cas9 gene may themselves be incorporated into the plant's genome.

To get around this problem, Kim and his colleagues avoid gene-shuttling altogether. They report a recipe to assemble the Cas9 enzyme together with its guide RNA sequences (which the enzyme requires to find its target) outside the plant, and use solvents to get the resulting protein complex into the plant. The technique works efficiently to knock out selected genes in tobacco plants, rice, lettuce and thale cress, they say, reporting their results in Nature Biotechnology2.

“I think this is a milestone work for plant science,’ says bioethicist Tetsuya Ishii at Hokkaido University in Sapporo, Japan, who has extensively studied the framework of regulation surrounding genetic engineering in plants.

Kim wants to use the technique to edit the banana; the crop's most popular cultivar, the Cavendish variety, is struggling to combat a devastating soil fungus and may go extinct. Gene editing could, for example, be used to knock out the receptor that the fungus uses to invade cells, without any need, in Kim’s view, to classify the resulting banana as a GMO. “We will save the banana so that our children and grandchildren can still enjoy the fruit,” he says.

Skirting regulations

Other scientists have recently achieved similar results with different genome-editing techniques. Jeffrey Wolt, a specialist in risk analysis of plant biotechnology at Iowa State University in Ames, points out that some researchers have introduced gene-editing protein complexes called TALENs directly into plants, for example3; others have used nanoparticles to usher in different gene-editing proteins4. To his mind, Kim’s paper is just one more tool in plant breeders’ arsenals — although many researchers say that CRISPR is cheaper and easier to use than other tools.

Jens Boch, a plant geneticist at Martin Luther University of Halle-Wittenberg in Germany who helped to develop TALEN, says that he hopes that workarounds that avoid Agrobacterium will not be necessary. When plants reproduce sexually, their genes are remixed, so they produce some offspring that do not have the offending bacterial DNA; breeding these Agrobacterium-free plants should appease regulators, he hopes. Agrobacterium “is just too easy to use, and this is going to be the method of choice”, he says. “I don't believe that plant breeders will use Kim’s method.” (Still, Kim points out that some plants, such as the banana, do not reproduce sexually, so would not lose an Agrobacterium gene if it were lodged in their genome.)

It is unclear what stance regulatory authorities will take on CRISPR-edited plants. The European Commission is currently debating regulations to take into account the latest techniques, and it is conceivable that it will still classify plants as GMOs even if they lack foreign DNA.

In the United States, editing plants with Agrobacterium is currently a trigger for regulation by the Animal and Plant Health Inspection Service, yet plants edited in other ways have bypassed regulations. But rules may change there too: in July, the White House launched a multiyear initiative to review federal regulations on agricultural biotechnology.

If regulations on CRISPR plants do turn out to be severe, Boch says, "the method proposed by Kim is a very good one to circumvent some of the possible criticisms".

Nature October 19, 2015

http://www.nature.com/news/crispr-tweak-may-help-gene-edited-crops-bypass-biosafety-regulation-1.18590

■ Glossary of essential terms

№	**English term**	**Russian equivalent**
1	biosafety	биобезопасность
2	gene-editing	генное редактирование
3	sidestepping	обходя
4	disease-resistant	устойчив к болезням
5	researchers	исследователи
6	jurisdictions	юрисдикция
7	avoid	избегать
8	fungus	грибок
9	altogether	в целом
10	milestone	веха
11	surrounding	окружающий
12	engineering	инжиниринг, проектирование
13	authorities	власти
14	agricultural biotechnology	сельскохозяйственная биотехнология
15	similar	подобное
16	launched	запущенный
17	circumvent	обходить
18	plant breeders	селекционеры
19	current	современный

I. Fill in the missing words:

1. A twist on a revolutionary ……may make it possible to modify plant genomes while sidestepping national biosafety regulations.

2. By disabling specific genes in wheat and rice, for example, researchers hope to make…….

3. And some jurisdictions, such as the European Union, could decide to classify such plants as…..— making their acceptance by regulatory bodies contentious

4. The gene is introduced on a plasmid —…..— which is usually carried into a plant by the bacterial pest…...

5. It is unclear what stance regulatory authorities will take…...

6. Conventionally, researchers ……in a plant cell by first shuttling in the gene that codes for the Cas9 enzyme.

II. Find English equivalents to the following word combinations:

№	**English term**	**Russian equivalent**
1	Genetically modified organisms	
2	Introducing foreign DNA	
3	Without any need	
4	The technique works efficiently	
5	To take into account the latest techniques	
6	Agricultural biotechnology	
7	They produce some offspring	
8	The offending bacterial DNA	
9	Stance regulatory authorities	

III. Find Russian equivalents to the following word combinations:

1	Чужеродная ДНК	
2	Национальные правила безопасности	
3	Редактирования генома	
4	Техника работает эффективно	
5	Популярный сорт урожая	
6	Белковые комплексы	
7	Другие использовали наночастицы	
8	Обходные пути	
9	Использовать растворители	
10	Изменить геномы растения	

IV. Use monolingual English dictionary and write down what could the words given below mean:

Plant, stance, agricultural, strains, fungus.

V. Find synonyms among the pool of words:

Pool of words	**Synonyms**
1)work/2)method/3)be employed/ 4)technique	
1)hope/2)plant/3)expectation4)/grower	
1)account/2)report/3)conceivable/4)thinkable	
1)specialist/2)provide food/3)feed/4)expert	
1)introduce/2)obligatory3)present/4)necessary	

VI. Fill in the missing words:

Term(Noun)	**Adjective**
Prevention	
Bacteria	
Technique	
Risk	
Diversity	

VIII. Match the sentence halves. Make complete sentences:

1	A twist on a revolutionary gene-editing technique may make	A	called Cas9, guided by two RNA strands, to precisely cut segments of DNA in a genome.
2	the process can introduce bits of foreign DNA	B	to edit the banana
3	Kim and his team tweaked the technique so	C	regulation by the Animal and Plant Health Inspection Service, yet plants edited in other ways have bypassed regulations
4	Even if the pest is not used, fragments of the Cas9 gene may themselves be incorporated	D	it possible to modify plant genomes while sidestepping national biosafety regulations
5	Kim wants to use the technique	E	into the plant's genome.
6	In the United States, editing plants	F	that it can delete specific plant genes

	with Agrobacterium is currently a trigger for		without introducing foreign DNA
7	Plant scientists have been quick to experiment with the popular CRISPR/Cas9 technique, which uses an enzyme	G	into plant genomes

IX. Fill in the missing words:

Term(verb)	Noun
Classify	
Control	
Discuss	
Appear	
Change	
Determinate	
Use	

X. Make sentences with the given words:

GMOs, Agrobacterium tumefaciens, gene-editing, fungus, scientists, enzyme.

ADDITIONAL TEXTS

Text 1. What Sparked the Cambrian Explosion?

An evolutionary burst 540 million years ago filled the seas with an astonishing diversity of animals. The trigger behind that revolution is finally coming into focus

Given the importance of oxygen for animals, researchers suspected that a sudden increase in the gas to near-modern levels in the ocean could have spurred the Cambrian explosion. To test that idea, they have studied ancient ocean sediments laid down during the Ediacaran and Cambrian periods, which together ran from about 635 million to 485 million years ago.

A series of dark, craggy pinnacles rises 80 meters above the grassy plains of Namibia. The peaks call to mind something ancient — the burial mounds of past civilizations or the tips of vast pyramids buried by the ages.

The stone formations are indeed monuments of a faded empire, but not from anything hewn by human hands. They are pinnacle reefs, built by cyanobacteria on the shallow sea floor 543 million years ago, during a time known as the Ediacaran period. The ancient world occupied by these reefs was truly alien. The oceans held so little oxygen that modern fish would quickly founder and die there. A gooey mat of microbes covered the sea floor at the time, and on that blanket lived a variety of enigmatic animals whose bodies resembled thin, quilted pillows. Most were stationary, but a few meandered blindly over the slime, grazing on the microbes. Animal life at this point was simple, and there were no predators. But an evolutionary storm would soon upend this quiet world.

Within several million years, this simple ecosystem would disappear, and give way to a world ruled by highly mobile animals that sported modern anatomical features. The Cambrian explosion, as it is called, produced arthropods

with legs and compound eyes, worms with feathery gills and swift predators that could crush prey in tooth-rimmed jaws. Biologists have argued for decades over what ignited this evolutionary burst. Some think that a steep rise in oxygen sparked the change, whereas others say that it sprang from the development of some key evolutionary innovation, such as vision. The precise cause has remained elusive, in part because so little is known about the physical and chemical environment at that time.

But over the past several years, discoveries have begun to yield some tantalizing clues about the end of the Ediacaran. Evidence gathered from the Namibian reefs and other sites suggests that earlier theories were overly simplistic — that the Cambrian explosion actually emerged out of a complex interplay between small environmental changes that triggered major evolutionary developments.

Some scientists now think that a small, perhaps temporary, increase in oxygen suddenly crossed an ecological threshold, enabling the emergence of predators. The rise of carnivory would have set off an evolutionary arms race that led to the burst of complex body types and behaviours that fill the oceans today. “This is the most significant event in Earth evolution,” says Guy Narbonne, a palaeobiologist at Queen's University in Kingston, Canada. “The advent of pervasive carnivory, made possible by oxygenation, is likely to have been a major trigger.”

ENERGY TO BURN

In the modern world, it's easy to forget that complex animals are relative newcomers to Earth. Since life first emerged more than 3 billion years ago, single-celled organisms have dominated the planet for most of its history. Thriving in environments that lacked oxygen, they relied on compounds such as carbon dioxide, sulfur-containing molecules or iron minerals that act as oxidizing agents to break down food. Much of Earth's microbial biosphere still survives on these anaerobic pathways.

Animals, however, depend on oxygen — a much richer way to make a living. The process of metabolizing food in the presence of oxygen releases much more energy than most anaerobic pathways. Animals rely on this potent, controlled combustion to drive such energy-hungry innovations as muscles, nervous systems and the tools of defence and carnivory — mineralized shells, exoskeletons and teeth.

Given the importance of oxygen for animals, researchers suspected that a sudden increase in the gas to near-modern levels in the ocean could have spurred the Cambrian explosion. To test that idea, they have studied ancient ocean sediments laid down during the Ediacaran and Cambrian periods, which together

ran from about 635 million to 485 million years ago.

In Namibia, China and other spots around the world, researchers have collected rocks that were once ancient seabeds, and analysed the amounts of iron, molybdenum and other metals in them. The metals' solubility depends strongly on the amount of oxygen present, so the amount and type of those metals in ancient sedimentary rocks reflect how much oxygen was in the water long ago, when the sediments formed.

These proxies seemed to indicate that oxygen concentrations in the oceans rose in several steps, approaching today's sea-surface concentrations at the start of the Cambrian, around 541 million years ago — just before more-modern animals suddenly appeared and diversified. This supported the idea of oxygen as a key trigger for the evolutionary explosion.

But last year, a major study of ancient sea-floor sediments challenged that view. Erik Sperling, a palaeontologist at Stanford University in California, compiled a database of 4,700 iron measurements taken from rocks around the world, spanning the Ediacaran and Cambrian periods. He and his colleagues did not find a statistically significant increase in the proportion of oxic to anoxic water at the boundary between the Ediacaran and the Cambrian.

“Any oxygenation event must have been far, far smaller than what people normally considered,” concludes Sperling. Most people assume “that the oxygenation event essentially raised oxygen to essentially modern-day levels. And that probably wasn't the case”, he says.

The latest results come at a time when scientists are already reconsidering what was happening to ocean oxygen levels during this crucial period. Donald Canfield, a geobiologist at the University of Southern Denmark in Odense, doubts that oxygen was a limiting factor for early animals. In a study published last month, he and his colleagues suggest that oxygen levels were already high enough to support simple animals, such as sponges, hundreds of millions of years before they actually appeared. Cambrian animals would have needed more oxygen than early sponges, concedes Canfield. “But you don't need an increase in oxygen across the Ediacaran/Cambrian boundary,” he says; oxygen could already have been abundant enough “for a long, long time before”.

“The role of oxygen in the origins of animals has been heavily debated,” says Timothy Lyons, a geobiologist at the University of California, Riverside. “In fact, it's never been more debated than it is now.” Lyons sees a role for oxygen in evolutionary changes, but his own work with molybdenum and other trace metals suggests that the increases in oxygen just before the Cambrian were mostly temporary peaks that lasted a few million years and gradually stepped upward (see 'When life sped up').

MODERN MIRRORS

Sperling has looked for insights into Ediacaran oceans by studying oxygen-depleted regions in modern seas around the globe. He suggests that biologists have conventionally taken the wrong approach to thinking about how oxygen shaped animal evolution. By pooling and analysing previously published data with some of his own, he found that tiny worms survive in areas of the sea floor where oxygen levels are incredibly low — less than 0.5% of average global sea-surface concentrations. Food webs in these oxygen-poor environments are simple, and the animals feed directly on microbes. In places where sea-floor oxygen levels are a bit higher — about 0.5–3% of concentrations at the sea surface — animals are more abundant but their food webs remain limited: the animals still feed on microbes rather than on each other. But around somewhere between 3% and 10% oxygen levels, predators emerge and start to consume other animals.

The implications of this finding for evolution are profound, Sperling says.The modest oxygen rise that he thinks may have occurred just before the Cambrian would have been enough to trigger a big change. “If oxygen levels were 3% and they rose past that 10% threshold, that would have had a huge influence on early animal evolution,” he says. “There's just so much in animal ecology, lifestyle and body size that seems to change so dramatically through those levels.”

The gradual emergence of predators, driven by a small rise in oxygen, would have meant trouble for Ediacaran animals that lacked obvious defences. “You're looking at soft-bodied, mostly immobile forms that probably lived their lives by absorbing nutrients through their skin,” says Narbonne.

Studies of those ancient Namibian reefs suggest that animals were indeed starting to fall prey to predators by the end of the Ediacaran. When palaeobiologist Rachel Wood from the University of Edinburgh, UK, examined the rock formations, she found spots where a primitive animal called C*loudina* had taken over parts of the microbial reef. Rather than spreading out over the ocean floor, these cone-shaped creatures lived in crowded colonies, which hid their vulnerable body parts from predators — an ecological dynamic that occurs in modern reefs.

C*loudina* were among the earliest animals known to have grown hard, mineralized exoskeletons. But they were not alone. Two other types of animal in those reefs also had mineralized parts, which suggests that multiple, unrelated groups evolved skeletal shells around the same time. “Skeletons are quite costly to produce,” says Wood. “It's very difficult to come up with a reason other than defence for why an animal should bother to create a skeleton for itself.” Wood thinks that the skeletons provided protection against newly evolved predators. Some C*loudina* fossils from that period even have holes in their sides, which scientists interpret as the marks of attackers that bore into the creatures' shells.

Palaeontologists have found other hints that animals had begun to eat each other by the late Ediacaran. In Namibia, Australia and Newfoundland in Canada, some sea-floor sediments have preserved an unusual type of tunnel made by an unknown, wormlike creature. Called *Treptichnus* burrows, these warrens branch again and again, as if a predator just below the microbial mat had systematically probed for prey animals on top. The *Treptichnus* burrows resemble those of modern priapulid, or 'penis', worms — voracious predators that hunt in a remarkably similar way on modern sea floors.

The rise of predation at this time put large, sedentary Ediacaran animals at a big disadvantage. “Sitting around doing nothing becomes a liability,” says Narbonne.

THE WORLD IN 3D

The moment of transition from the Ediacaran to the Cambrian world is recorded in a series of stone outcrops rounded by ancient glaciers on the south edge of Newfoundland. Below that boundary are impressions left by quilted Ediacaran animals, the last such fossils recorded on Earth. And just 1.2 meters above them, the grey siltstone holds trails of scratch marks, thought to have been made by animals with exoskeletons, walking on jointed legs — the earliest evidence of arthropods in Earth's history.

No one knows how much time passed in that intervening rock — maybe as little as a few centuries or millennia, says Narbonne. But during that short span, the soft-bodied, stationary Ediacaran fauna suddenly disappeared, driven to extinction by predators, he suggests.

Narbonne has closely studied the few fauna that survived this transition, and his findings suggest that some of them had acquired new, more complex types of behaviour. The best clues come from traces left by peaceful, wormlike animals that grazed on the microbial mat. Early trails from about 555 million years ago meander and criss-cross haphazardly, indicating a poorly developed nervous system that was unable to sense or react to other grazers nearby — let alone predators. But at the end of the Ediacaran and into the early Cambrian, the trails become more sophisticated: creatures carved tighter turns and ploughed closely spaced, parallel lines through the sediments. In some cases, a curvy feeding trail abruptly transitions into a straight line, which Narbonne interprets as potential evidence of the grazer evading a predator.

This change in grazing style may have contributed to the fragmentation of the microbial mat, which began early in the Cambrian. And the transformation of the sea floor, says Narbonne, “may have been the most profound change in the history of life on Earth”, . The mat had previously covered the seabed like a coating of plastic wrap, leaving the underlying sediments largely anoxic and off

limits to animals. Because animals could not burrow deeply in the Ediacaran, he says, “the mat meant that life was two-dimensional”. When grazing capabilities improved, animals penetrated the mat and made the sediments habitable for the first time, which opened up a 3D world.

Tracks from the early Cambrian show that animals started to burrow several centimeters into the sediments beneath the mat, which provided access to previously untapped nutrients — as well as a refuge from predators. It's also possible that animals went in the opposite direction. Sperling says that the need to avoid predators (and pursue prey) may have driven animals into the water column above the seabed, where enhanced oxygen levels enabled them to expend energy through swimming.

The emerging evidence about oxygen thresholds and ecology could also shed light on another major evolutionary question: when did animals originate? The first undisputed fossils of animals appear only 580 million years ago, but genetic evidence indicates that basic animal groups originated as far back as 700 million to 800 million years ago. According to Lyons, the solution may be that oxygen levels rose to perhaps 2% or 3% of modern levels around 800 million years ago. These concentrations could have sustained small, simple animals, just as they do today in the ocean's oxygen-poor zones. But animals with large bodies could not have evolved until oxygen levels climbed higher in the Ediacaran.

Understanding how oxygen influenced the appearance of complex animals will require scientists to tease more-subtle clues out of the rocks. “We've been challenging people working on fossils to tie their fossils more closely to our oxygen proxies,” says Lyons. It will mean deciphering what oxygen levels were in different ancient environments, and connecting those values with the kinds of traits exhibited by the animal fossils found in the same locations.

This past autumn, Woods visited Siberia with that goal in mind. She collected fossils of *Cloudina* and another skeletonized animal, *Suvorovella*, from the waning days of the Ediacaran. Those sites gave her the chance to gather fossils from many different depths in the ancient ocean, from the more oxygen-rich surface waters to deeper zones. Wood plans to look for patterns in where animals were growing tougher skeletons, whether they were under attack by predators and whether any of this had a clear link with oxygen levels, she says. “Only then can you pick out the story.”

Scientific American June 29, 2015

http://www.scientificamerican.com/article/a-battle-of-the-sexes-is-waged-in-the-genes-of-humans-bulls-and-more/

Text 2. Biotech Interest in Mini Organs Booms

Biologists are building banks of 'organoids', and learning a lot about human development on the way

It was an otherwise normal day in November when Madeline Lancaster realized that she had accidentally grown a brain. For weeks, she had been trying to get human embryonic stem cells to form neural rosettes, clusters of cells that can become many different types of neuron. But for some reason her cells refused to stick to the bottom of the culture plate. Instead they floated, forming strange, milky-looking spheres.

"I didn't really know what they were," says Lancaster, who was then a postdoc at the Institute of Molecular Biotechnology in Vienna. That day in 2011, however, she spotted an odd dot of pigment in one of her spheres. Looking under the microscope, she realized that it was the dark cells of a developing retina, an outgrowth of the developing brain. And when she sliced one of the balls open, she could pick out a variety of neurons. Lancaster realized that the cells had assembled themselves into something unmistakably like an embryonic brain, and she went straight to her adviser, stem-cell biologist Jürgen Knoblich, with the news. "I've got something amazing," she told him. "You've got to see it."

Lancaster and her colleagues were not the first to grow a brain in a dish. In 2008, researchers in Japan reported that they had prompted embryonic stem cells from mice and humans to form layered balls reminiscent of a cerebral cortex. Since then, efforts to grow stem cells into rudimentary organs have taken off. Using carefully timed chemical cues, researchers around the world have produced three-dimensional structures that resemble tissue from the eye, gut, liver, kidney, pancreas, prostate, lung, stomach and breast. These bits of tissue, called organoids because they mimic some of the structure and function of real organs, are furthering knowledge of human development, serving as disease models and drug-screening platforms, and might eventually be used to rescue damaged organs (see 'The organoid bank'). "It's probably the most significant development in the

stem-cell field in the last five or six years," says Austin Smith, director of the Wellcome Trust/MRC Stem Cell Institute at the University of Cambridge, UK.

The current crop of organoids isn't perfect. Some lack key cell types; others imitate only the earliest stages of organ development or vary from batch to batch. So researchers are toiling to refine their organoids—to make them more complex, more mature and more reproducible. Still, biologists have been amazed at how little encouragement cells need to self-assemble into elaborate structures. "It doesn't require any super-sophisticated bioengineering," says Knoblich. "We just let the cells do what they want to do, and they make a brain."

Growing a gut

This shouldn't come as a major surprise, says molecular biologist Melissa Little at the University of Queensland, Australia. "The embryo itself is incredibly able to self-organize; it doesn't need a template or a map." That has been known since the early 1900s, when embryologists showed that sponges that had been broken up into single cells could reassemble themselves. But such work fell out of fashion, and modern biologists have focused their attention on purifying cells and growing them in culture—often in flat layers that do little to mimic normal human tissue.

Studying these cells to understand how an organ functions is like studying a pile of bricks to understand the function of a house, says Mina Bissell, a cancer researcher at the Lawrence Berkeley National Laboratory in California. "We should just begin to make the house," she says. Bissell's work on cultures of breast cells helped to propagate the idea that cells behave differently in 3D cultures than in conventional flat ones. By the mid-2000s, the idea was catching on. The burst of enthusiasm was fuelled by Yoshiki Sasai, a stem-cell biologist at the RIKEN Center for Developmental Biology in Kobe, Japan, who turned heads when he grew a cerebral cortex, followed by a rudimentary optic cup and pituitary gland.

Just a year after Sasai announced his layered cortex, Hans Clevers, a stem-cell researcher at the Hubrecht Institute in Utrecht, the Netherlands, reported the creation of a mini-gut. The breakthrough stemmed from a discovery in 2007, when Clevers and his colleagues had identified intestinal stem cells in mice. In the body, these cells seemed to have an unlimited capacity to divide and replenish the intestinal lining, and one of Clevers' postdocs, Toshiro Sato, was tasked with culturing them in the lab.

Rather than growing the cells flat, the pair decided to embed them in matrigel, a soft jelly that resembles the extracellular matrix, the mesh of molecules that surrounds cells. "We were just trying things," Clevers says. "We hoped that we would make maybe a sphere or a blob of cells." Several months later, when

Clevers put his eye to Sato's microscope, he saw more than blobs. The cells had divided, differentiated into multiple types, and formed hollow spheres that were dotted with knobby protrusions. Inside, the team found structures that resembled the intestine's nutrient-absorbing villi as well as the deep valleys between them called crypts. "The structures, to our total astonishment, looked like real guts," Clevers says. "They were beautiful."

The mini-guts, reported in 2009, may prove to be a powerful tool in personalized medicine. Clevers and his team are using them to study the effectiveness of drugs in people with cystic fibrosis, who have genetic defects that affect ion channels and disrupt the movement of water in and out of the cells lining the lungs and intestine. The researchers take rectal biopsies from people with the disease, use the cells to create personalized gut organoids and then apply a potential drug. If the treatment opens the ion channels, then water can flow inwards and the gut organoids swell up. "It's a black-and-white assay," Clevers says, one that could prove quicker and cheaper than trying drugs in people to see whether they work.

He has already used the system to assess whether a drug called Kalydeco (ivacaftor), and 5 other cystic-fibrosis drugs, will work in about 100 patients; at least 2 of them are now taking Kalydeco as a result.

Organoids may also help physicians to choose the best therapies for people with cancer. Earlier this year, Clevers revealed that he had grown a bank of organoids from cells extracted from colorectal tumours, and David Tuveson, a cancer researcher at Cold Spring Harbor Laboratory in New York, worked with Clevers to generate pancreas organoids using biopsies taken from people with pancreatic cancer. In both cases, the organoids could be used to find drugs that work best on particular tumours. "What patients are looking for is a logical approach to their cancer," Tuveson says. "I'm very excited about what we're learning."

The small-scale stomach

That excitement is shared by developmental biologist James Wells, who last year reported that he and his team had created an organoid that resembled part of a human stomach.

Wells started with a different raw material to Clevers, whose organoids arise from adult stem cells that can generate only a limited number of cell types. Wells, who is at the Cincinnati Children's Hospital Medical Center in Ohio, and his colleagues craft organoids from embryonic stem cells, which have the ability to become almost any type of cell. As a result, they have been able to create mini-organs that are more complex.

A decade ago, Wells and his colleagues began trying to coax human embryonic stem cells to form intestinal cells. When the team manipulated two key signalling pathways, the layer of cells produced tiny round buds. Wells noticed that these 'spheroids' mimicked sections of the primitive gut tube, which forms four weeks after conception. This was thrilling, because he realized that he now had a starting point from which to develop a variety of organoids. "Every organ from your mouth down to your anus—oesophagus, lungs, trachea, stomach, pancreas, liver, intestine, bladder—all of them come from this very primitive tube," he says.

Wells and his colleagues mined the literature and their own experience to determine what chemical cues might send these gut tubes down the developmental path toward a specific organ. Using this strategy, in 2011 the team developed its first human organoid, an intestine about the size of a sesame seed. But growing a stomach was a bigger challenge. In humans, the organ has two key areas: the fundus at the top, which churns out acid, and the antrum towards the base, which produces many key digestive hormones—and the signalling pathways that lead to one versus the other were unknown. What is more, "the human stomach is different from the stomachs of most animals that we use in the lab", so there is no good animal model, says Kyle McCracken, a former graduate student of Wells and now a medical student at the center.

The researchers went for a trial-and-error approach: they made some educated guesses and painstakingly tested different combinations of growth factors. Eventually, the effort paid off. In a 2014 paper, Wells and his team revealed that they had created organoids that resembled the antrum. Using these as a model system, the team says that it has figured out the chemical trigger that prompts the development of a fundus. Now the researchers are working to answer other basic questions about stomach development and physiology, such as which factors regulate acid secretion, and they are trying to generate other mini-organs from their primitive gut tubes.

This newfound ability to examine human development excites Daniel St Johnston, a developmental geneticist at the University of Cambridge's Gurdon Institute. "You can actually watch how the cells organize themselves to make complicated structures," he says—something that is impossible in a human embryo. But most organoids are still single tissues, which limits what developmental biologists can learn, he says. "There are certain questions you can't really address because they depend upon the physiology of the whole organism."

The baby kidney

Melissa Little has spent more than a decade marvelling at the complexity of the kidney. "It has, in an adult, probably 25–30 different cell types, each doing

different jobs," she says. Tubular structures called nephrons filter fluid from the blood and produce urine. The surrounding space, called the interstitium, holds an intricate network of blood vessels and the plumbing that carries urine away.

In 2010, Little and her colleagues started trying to turn embryonic stem cells into a progenitor cell that gives rise to nephrons. For three years, they tried various combinations and timings of growth factors. "It really took a lot of mucking around to make progress," she says. But finally, in 2013, the team landed on just the right mixture. Little had been aiming to produce just the progenitor cells. But when she looked in the dish she saw two cell types spontaneously patterning themselves as they would in an embryo. "There was a moment of, 'Oh wow. Isn't that amazing'," she says.

This organoid resembles an embryonic kidney rather than an adult one: it has a mix of nephron progenitors and the cells that give rise to urine-collecting ducts. "If you want to get them to mature further, that's where the challenge really lies," Little says. So her team has been working to grow a more-sophisticated version—with blood vessels and interstitium. The hope then is to transplant the mini-organs into mice to see if they will mature and produce urine. "I'm pretty excited about what we can build," Little says.

Because the kidney plays a key part in drug metabolism and excretion, Little thinks that her mini-kidneys could be useful for testing drug candidates for toxicity before they reach clinical trials. And researchers say that other human organoids, such as heart and liver, could similarly be used to screen drug candidates for toxic effects—offering a better read-out on the response of an organ than is possible with standard tissue culture or animal testing.

But Michael Shen, a stem-cell researcher at Columbia University in New York who has created a prostate organoid, is sceptical that these model systems could completely replace lab animals. Animals can show how a therapy affects the immune system, for example, something that organoid systems cannot currently do. "You want to be able to validate your experimental findings in an *in vivo* system," he says. "I view that as a rigorous test."

Little livers

Takanori Takebe was inspired to grow a liver after a chilling spell in New York. While working in the organ-transplantation division at Columbia University in 2010, Takebe saw people die from liver failure owing to a lack of organs. "That was a sad situation," he says. When he looked into tissue engineering, he thought that the usual methods—seeding cells onto an artificial scaffold—seemed destined to fail. Part of the problem, he says, is that adult liver cells are very difficult to grow. "We cannot maintain it in culture for even a couple of hours."

Takebe, who took up a research position at Yokohama City University in Japan, decided to work on induced pluripotent stem (iPS) cells, adult cells that have been reprogrammed to behave like embryonic stem cells. He coaxed human iPS cells into forming liver-cell precursors, or hepatoblasts. In the embryo, hepatoblasts rely on a complex symphony of signals from other nearby cells to mature, and Takebe suspected that these support cells would also be necessary to develop a liver in a dish. He and his colleagues mixed hepatoblasts with such cells—called mesenchymal and endothelial cells—and it worked. The team managed to create 'liver buds', structures no bigger than a lentil that resemble the liver of a six-week-old human embryo. The researchers went on to find that, unlike mature liver cells, such structures can survive in culture for as long as two months.

A liver bud is still a far cry from an entire liver—a hefty, multi-lobed organ composed of tens of billions of hepatocytes. But Takebe hopes that if he can infuse many thousands of buds into a failing organ, he might be able to rescue enough of its function to make a transplant unnecessary. The process seems to work in mice. When Takebe and his group transplanted a dozen of the buds into mouse abdomens, they saw dramatic effects. Within two days, the buds had connected up with the mouse's blood supply, and the cells went on to develop into mature liver cells that were able to make liver-specific proteins and to metabolize drugs. To mimic liver failure, the team wiped out the animals' natural liver function with a toxic drug. After a month, most of the control mice had died, but most of those that received liver bud transplants had survived.

Takebe and his team hope to start human trials in four years. "We will target the children that critically need a liver transplant," he says. He and his colleagues are currently working to make the liver buds smaller and produce them in huge quantities that they can infuse through the large portal vein that feeds the liver. Takebe thinks that the timeline is "doable". But Smith says that the process seems rushed, and that the basic biology of these organs needs to be well understood before they are used in the clinic. "It's like running before you can walk," he says.

Biologists know that their mini-organs are still a crude mimic of their life-sized counterparts. But that gives them something to aim for, says Anthony Atala, director of the Wake Forest Institute for Regenerative Medicine in Winston-Salem, North Carolina. "The long-term goal is that you will be able to replicate more and more of the functionality of a human organ." Already, the field has brought together developmental biologists, stem-cell biologists and clinical scientists. Now the aim is to build more-elaborate organs—ones that are larger and that integrate more cell types.

And Wells says that even today's rudimentary organoids are facilitating discoveries that would have been difficult to make in an animal model, in which

the molecular signals are hard to manipulate. “In a Petri dish it’s easy,” he says. “We have chemicals and proteins that we can just dump onto these cells.”

Scientific American July 29, 2015
http://www.scientificamerican.com/article/biotech-interest-in-mini-organs-booms/

Text 3. Synthetic Biology's First Malaria Drug Meets Market Resistance

Commercial use of genetically engineered yeast to make medicine has modest impact

By Mark Peplow, Nature magazine on February 25, 2016

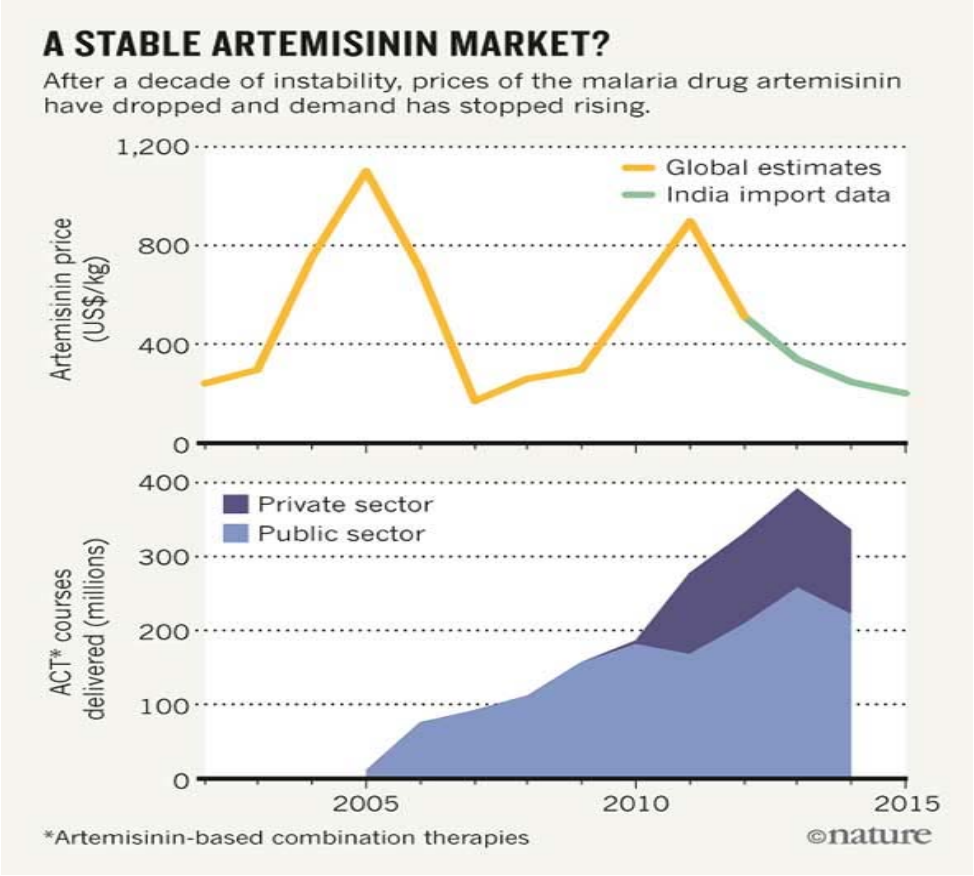
When Paris-based pharmaceutical giant Sanofi started to sell malaria drugs made with the help of genetically engineered yeast in 2014, the move was hailed as a triumph for synthetic biology. The yeast was fermented in a vat to produce a chemical that Sanofi converted into artemisinin, which is used to make leading malaria treatments called artemisinin-based combination therapies (ACTs). Many hoped that the process would offer a cheap and plentiful supply of drugs to tackle a disease that claims almost half a million lives worldwide every year.

Yet Sanofi produced no ‘semi-synthetic’ artemisinin (SSA) at all in 2015, *Nature* has learned. And the company is now selling the manufacturing site in Garessio, Italy, where it made its SSA.

That such celebrated drugmaking technology—developed with the help of US$64 million from the Bill & Melinda Gates Foundation—stands idle illustrates the complicated web of economic forces that affects the market for malaria drugs.

“This is a perfect example of how a new manufacturing process becomes extremely hard to scale up when there is a complex ecosystem of players,” says Prashant Yadav, a health-policy researcher at the William Davidson Institute at the University of Michigan, Ann Arbor, who studies the ACT market.

Before the advent of SSA, the only source of artemisinin was the sweet wormwood plant (*Artemisia annua*), the discovery of which won Chinese scientist Youyou Tu a share of the 2015 Nobel Prize in Physiology or Medicine. But the agricultural supply has been erratic. Shortages of*A. annua* send prices soaring, which attracts more farmers to plant it; their produce then swamps the market, depressing prices and triggering fresh shortages (see ‘A stable artemisinin market?’).



SUPPLEMENT OR SAVIOUR?

The synthetic-biology route promised to end this rollercoaster by providing a stable and reliable source of artemisinin. Sanofi developed the capacity to produce almost 60 tonnes of the chemical per year—about one-third of global need—and the company hoped to supply other ACT manufacturers with the raw materials.

“In reality, that has not happened,” says Yadav. Sanofi has so far used its SSA to make more than 39 million treatments of its own version of ACT—representing about 10% of global ACT demand—but has not sold the chemical to other drugmakers.

That is partly because of a glut in agricultural artemisinin. For the past two years, the naturally derived chemical has sold for less than $250 per kilogram—below Sanofi's 'no profit–no loss' margin of around $350–400 per kilogram. "If that price is already very low and there's a bumper crop, there's no reason to fire up a fermenter," says Jay Keasling of the University of California, Berkeley, who led the team that first developed the yeast strain.

But ACT manufacturers such as China's Guilin Pharma and India's Cipla are also reluctant to buy their drug ingredients from Sanofi, says Yadav, because the company is a direct competitor in the ACT market.

And Sanofi has not found it worthwhile to increase production of its own ACT because demand has plateaued. This is in part the result of growing efforts to diagnose malaria before doling out medicine: malaria treatments are often taken by people with fevers who do not actually have malaria, so more-accurate diagnoses help to reduce the number of treatments needed. Whether demand will rise again will depend on how international efforts to tackle malaria develop in the future, and how much funding will be available to purchase ACTs.

By July, Sanofi will complete the sale of its Garessio manufacturing plant to Bulgarian company Huvepharma, a contract manufacturer responsible for fermenting the engineered yeast in vats to make artemisinic acid—the precursor to artemisinin—for Sanofi.

Nicola de Risi, a manager for Huvepharma in Rome who will head the firm's Italian division, hopes that by gaining control of the entire SSA production process (from yeast to final product), the company will be able to lower costs and make sales to other ACT manufacturers. But Huvepharma will switch to using plant-derived artemisinin if it cannot make SSA cost-competitive, de Risi says.

PATH, a global-health organization based in Seattle, Washington, which coordinated the development of SSA, says that it still considers the project a success. "Since SSA entered the market, we have observed better price stability, and there has been adequate supply of artemisinin," it said in a statement.

"There is merit to the argument that SSA has contributed somewhat to stabilizing prices," says Yadav. But the main causes of price stability, he adds, are the recent steady demand for ACTs and long-term purchasing contracts with ACT manufacturers, set up by the Global Fund to Fight AIDS, Tuberculosis and Malaria.

PATH and Keasling say that SSA was always intended to be a supplemental source to fill gaps in agricultural production, or to cope with spikes in demand. But Claire Marris, a sociologist of science at City University London who previously worked at the Centre for Synthetic Biology and Innovation at Imperial College London, says that in her experience, SSA is often portrayed by those working in

the field as simply a low-cost, high-volume substitute for agricultural artemisinin. “It was constantly talked about,” she says. Now, Marris worries that unrealistic expectations for SSA’s achievements could damage public trust in synthetic biology.

When the Gates Foundation awarded the first of its grants for the SSA project in 2004, it explicitly aimed to lower the cost of each ACT treatment from $2.40 to “well under a dollar”. But the median price of Sanofi’s ACT had already dipped to $0.92 per adult treatment by 2012, well before the introduction of SSA, and it has changed little since then.

De Risi says that SSA production will restart later this year so that Sanofi can produce its own ACT treatment. “I think it’s good for synthetic artemisinin,” says Yadav, who points out that other ACT producers may be more willing to buy artemisinin from Huvepharma because it is not an ACT producer itself—and therefore not a direct competitor.

Meanwhile, Guilin Pharma and Cipla are making plans to develop their own SSA, and Keasling hopes that more research and development work could make the synthetic process cheaper in the long term. “I’d like to see SSA take over as the dominant form, and some day I think it will,” says Keasling. “But we have to be patient.”

Scientific American February 25, 2016
http://www.scientificamerican.com/article/synthetic-biology-s-first-malaria-drug-meets-market-resistance/

Text 4. CRISPR/Cas9 and Targeted Genome Editing: A New Era in Molecular Biology

The development of efficient and reliable ways to make precise, targeted changes to the genome of living cells is a long-standing goal for biomedical researchers. Recently, a new tool based on a bacterial CRISPR-associated protein-9 nuclease (Cas9) from *Streptococcus pyogenes* has generated considerable excitement. This follows several attempts over the years to manipulate gene function, including homologous recombination and RNA interference (RNAi). RNAi, in particular, became a laboratory staple enabling inexpensive and high-throughput interrogation of gene function, but it is hampered by providing only temporary inhibition of gene function and unpredictable off-target effects. Other recent approaches to targeted genome modification – zinc-finger nucleases [ZFNs,] and transcription-activator like effector nucleases [TALENs]– enable researchers

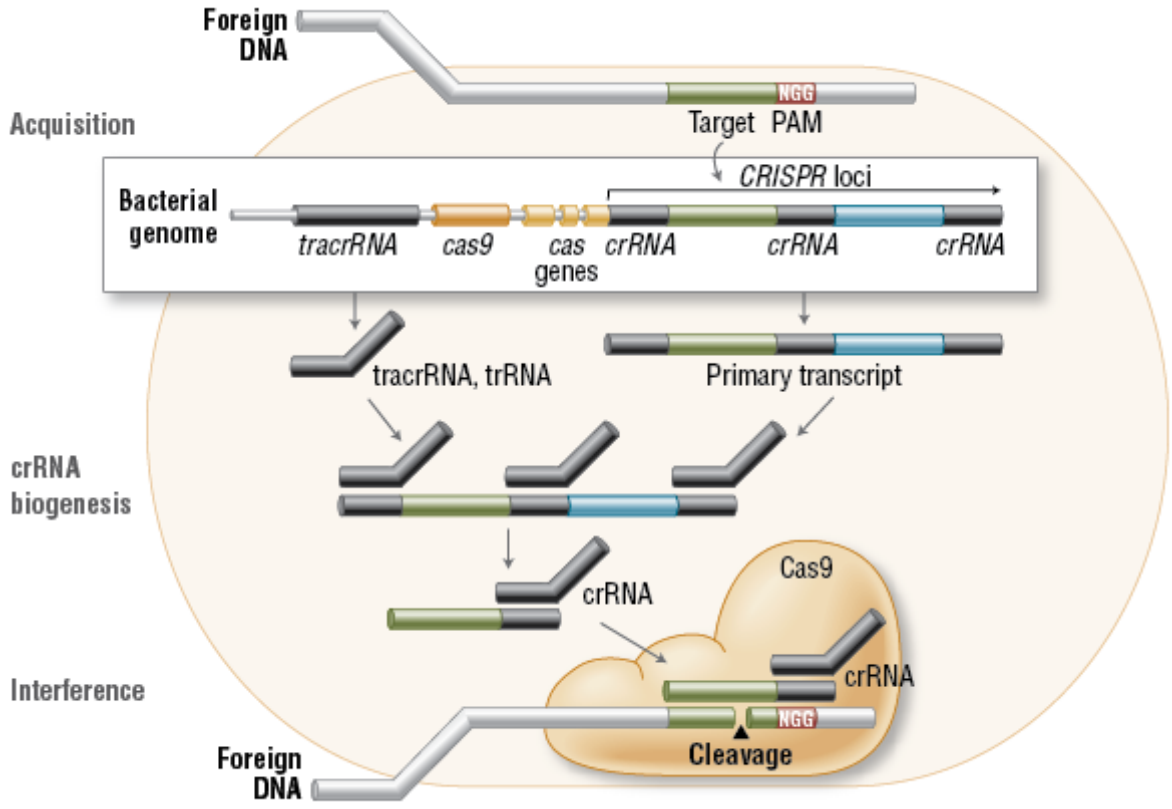
to generate permanent mutations by introducing doublestranded breaks to activate repair pathways. These approaches are costly and time-consuming to engineer, limiting their widespread use, particularly for large scale, high-throughput studies.

The Biology of Cas9

The functions of CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats) and CRISPR-associated (Cas) genes are essential in adaptive immunity in select bacteria and archaea, enabling the organisms to respond to and eliminate invading genetic material. These repeats were initially discovered in the 1980s in *E. coli* , but their function wasn't confirmed until 2007 by Barrangou and colleagues, who demonstrated that *S. thermophilus*can acquire resistance against a bacteriophage by integrating a genome fragment of an infectious virus into its CRISPR locus .

Three types of CRISPR mechanisms have been identified, of which type II is the most studied. In this case, invading DNA from viruses or plasmids is cut into small fragments and incorporated into a CRISPR locus amidst a series of short repeats (around 20 bps). The loci are transcribed, and transcripts are then processed to generate small RNAs (crRNA – CRISPR RNA), which are used to guide effector endonucleases that target invading DNA based on sequence complementarity

(Figure1)



Genome Editing Glossary

Cas = CRISPR-associated genes

Cas9, Csn1 = a CRISPR-associated protein containing two nuclease domains, that is programmed by small RNAs to cleave DNA

crRNA = CRISPR RNA

dCAS9 = nuclease-deficient Cas9

DSB = Double-Stranded Break

gRNA = guide RNA

HDR = Homology-Directed Repair

HNH = an endonuclease domain named for characteristic histidine and asparagine residues

Indel = insertion and/or deletion

NHEJ = Non-Homologous End Joining

PAM = Protospacer-Adjacent Motif

RuvC = an endonuclease domain named for an *E. coli* protein involved in DNA repair

sgRNA = single guide RNA

tracrRNA, trRNA = trans-activating crRNA

TALEN = Transcription-Activator Like Effector Nuclease

ZFN = Zinc-Finger Nuclease

One Cas protein, Cas9 (also known as Csn1), has been shown, through knockdown and rescue experiments to be a key player in certain CRISPR mechanisms (specifically type II CRISPR systems). The type II CRISPR mechanism is unique compared to other CRISPR systems, as only one Cas protein (Cas9) is required for gene silencing . In type II systems, Cas9 participates in the processing of crRNAs , and is responsible for the destruction of the target DNA . Cas9's function in both of these steps relies on the presence of two nuclease domains, a RuvC-like nuclease domain located at the amino terminus and a HNH-like nuclease domain that resides in the mid-region of the protein .

To achieve site-specific DNA recognition and cleavage, Cas9 must be complexed with both a crRNA and a separate trans-activating crRNA (tracrRNA or trRNA), that is partially complementary to the crRNA . The tracrRNA is required for crRNA maturation from a primary transcript encoding multiple pre-crRNAs. This occurs in the presence of RNase III and Cas9 .

During the destruction of target DNA, the HNH and RuvC-like nuclease domains cut both DNA strands, generating double-stranded breaks (DSBs) at sites defined by a 20-nucleotide target sequence within an associated crRNA transcript (11, 14). The HNH domain cleaves the complementary strand, while the RuvC domain cleaves the noncomplementary strand.

The double-stranded endonuclease activity of Cas9 also requires that a short conserved sequence, (2–5 nts) known as protospacer-associated motif (PAM), follows immediately 3′- of the crRNA complementary sequence. In fact, even fully

complementary sequences are ignored by Cas9-RNA in the absence of a PAM sequence .

Cas9 and CRISPR as a New Tool in Molecular Biology

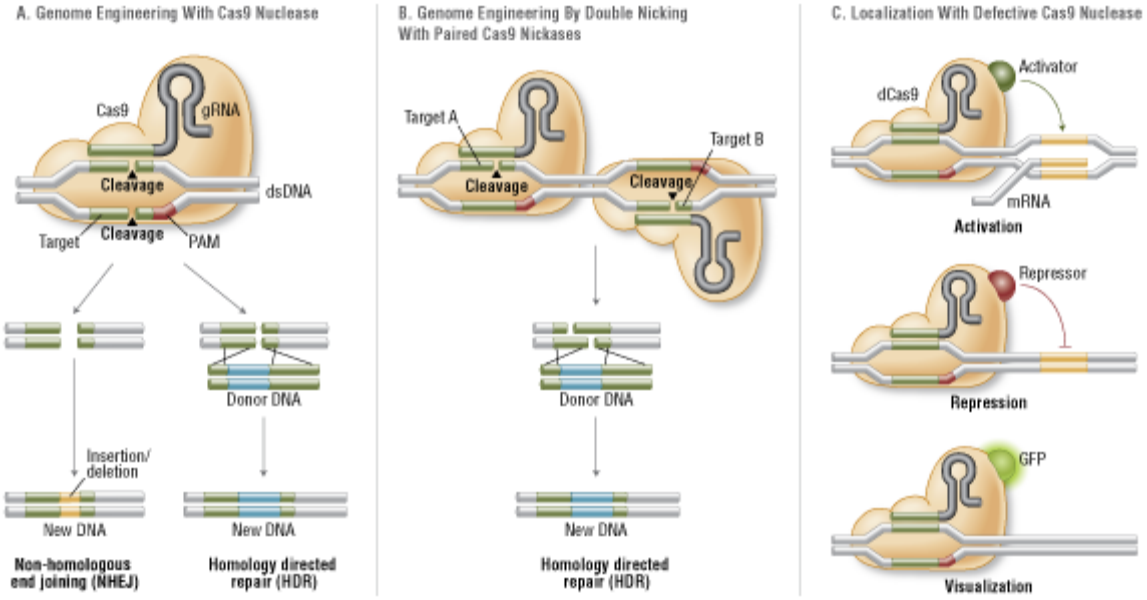
The simplicity of the type II CRISPR nuclease, with only three required components (Cas9 along with the crRNA and trRNA) makes this system amenable to adaptation for genome editing. This potential was realized in 2012 by the Doudna and Charpentier labs. Based on the type II CRISPR system described previously, the authors developed a simplified two-component system by combining trRNA and crRNA into a single synthetic single guide RNA (sgRNA). sgRNAprogrammed Cas9 was shown to be as effective as Cas9 programmed with separate trRNA and crRNA in guiding targeted gene alterations (Figure 2A).

To date, three different variants of the Cas9 nuclease have been adopted in genome-editing protocols. The first is wild-type Cas9, which can site-specifically cleave double-stranded DNA, resulting in the activation of the doublestrand break (DSB) repair machinery. DSBs can be repaired by the cellular Non-Homologous End Joining (NHEJ) pathway , resulting in insertions and/or deletions (indels) which disrupt the targeted locus. Alternatively, if a donor template with homology to the targeted locus is supplied, the DSB may be repaired by the homology-directed repair (HDR) pathway allowing for precise replacement mutations to be made (Figure 2A) .

Cong and colleagues took the Cas9 system a step further towards increased precision by developing a mutant form, known as Cas9D10A, with only nickase activity. This means it cleaves only one DNA strand, and does not activate NHEJ. Instead, when provided with a homologous repair template, DNA repairs are conducted via the high-fidelity HDR pathway only, resulting in reduced indel mutations . Cas9D10A is even more appealing in terms of target specificity when loci are targeted by paired Cas9 complexes designed to generate adjacent DNA nicks (see further details about “paired nickases” in Figure 2B).

The third variant is a nuclease-deficient Cas9 (dCas9, Figure 2C) . Mutations H840A in the HNH domain and D10A in the RuvC domain inactivate cleavage activity, but do not prevent DNA binding . Therefore, this variant can be used to sequence-specifically target any region of the genome without cleavage. Instead, by fusing with various effector domains, dCas9 can be used either as a gene silencing or activation tool .Furthermore, it can be used as a visualization tool. For instance, Chen and colleagues used dCas9 fused to Enhanced Green Fluorescent Protein (EGFP) to visualize repetitive DNA sequences with a single sgRNA or nonrepetitive loci using multiple sgRNAs.

Figure 2. CRISPR/Cas9 System Applications



A. Wild-type Cas9 nuclease site specifically cleaves double-stranded DNA activating double-strand break repair machinery. In the absence of a homologous repair template non-homologous end joining can result in indels disrupting the target sequence. Alternatively, precise mutations and knock-ins can be made by providing a homologous repair template and exploiting the homology directed repair pathway.

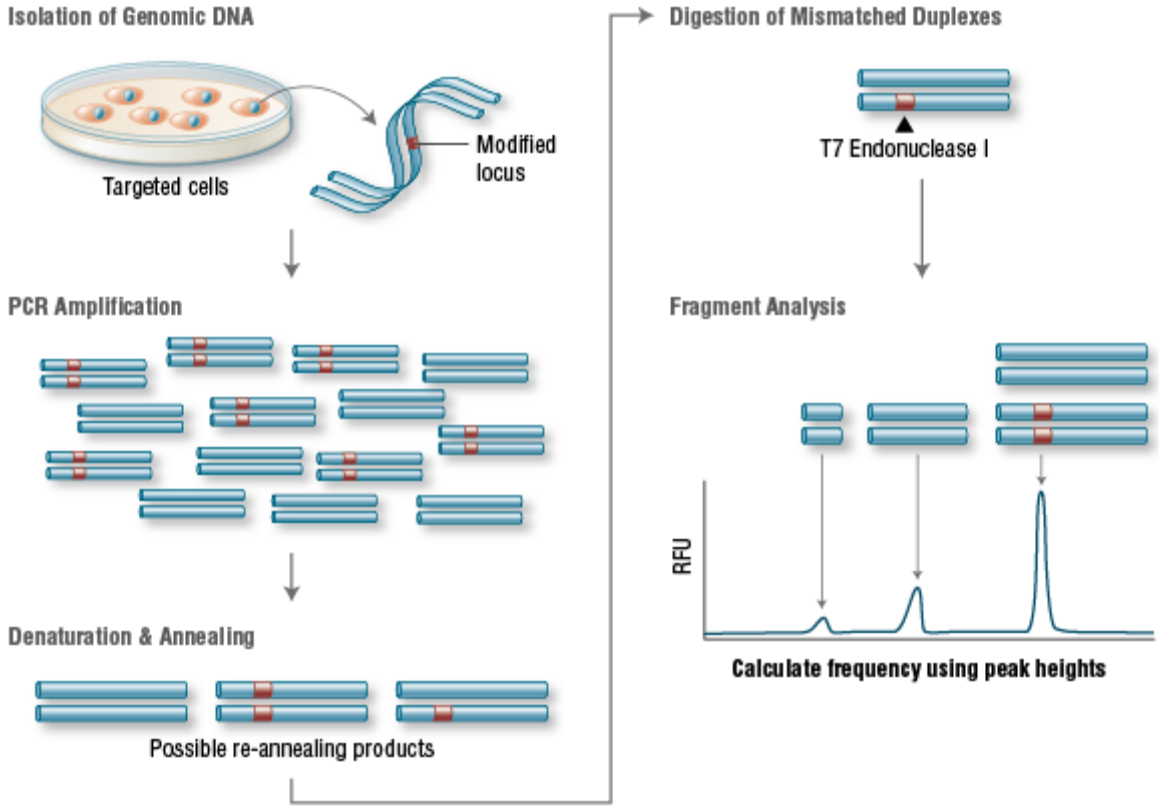
B. Mutated Cas9 makes a site specific single-strand nick. Two sgRNA can be used to introduce a staggered double-stranded break which can then undergo homology directed repair.

C. Nuclease-deficient Cas9 can be fused with various effector domains allowing specific localization. For example, transcriptional activators, repressors, and fluorescent proteins.

Targeting Efficiency and Off-target Mutations

Targeting efficiency, or the percentage of desired mutation achieved, is one of the most important parameters by which to assess a genome-editing tool. The targeting efficiency of Cas9 compares favorably with more established methods, such as TALENs or ZFNs . For example, in human cells, custom-designed ZFNs and TALENs could only achieve efficiencies ranging from 1% to 50% . In contrast, the Cas9 system has been reported to have efficiencies up to >70% in zebrfish and plants,and ranging from 2–5% in induced pluripotent stem cells . In addition, Zhou and colleagues were able to improve genome targeting up to 78% in one-cell mouse embryos, and achieved effective germline transmission through the use of dual sgRNAs to simultaneously target an individual gene .

A widely used method to identify mutations is the T7 Endonuclease I mutation detection assay (36, 37) (Figure 3). This assay detects heteroduplex DNA that results from the annealing of a DNA strand, including desired mutations, with a wildtype DNA strand (37).



Genomic DNA is amplified with primers bracketing the modified locus. PCR products are then denatured and re-annealed yielding 3 possible structures. Duplexes containing a mismatch are digested by T7 Endonuclease I. The DNA is then electrophoretically separated and fragment analysis is used to calculate targeting efficiency.

Another important parameter is the incidence of off-target mutations. Such mutations are likely to appear in sites that have differences of only a few nucleotides compared to the original sequence, as long as they are adjacent to a PAM sequence. This occurs as Cas9 can tolerate up to 5 base mismatches within the protospacer region or a single base difference in the PAM sequence . Off-target mutations are generally more difficult to detect, requiring whole-genome sequencing to rule them out completely.

Recent improvements to the CRISPR system for reducing off-target mutations have been made through the use of truncated gRNA (truncated within the crRNA-derived sequence) or by adding two extra guanine (G) nucleotides to the 5′ end. Another way researchers have attempted to minimize off-target effects is with the use of “paired nickases”. This strategy uses D10A Cas9 and two

sgRNAs complementary to the adjacent area on opposite strands of the target site (Figure 2B). While this induces DSBs in the target DNA, it is expected to create only single nicks in off-target locations and, therefore, result in minimal off-target mutations.

By leveraging computation to reduce off-target mutations, several groups have developed webbased tools to facilitate the identification of potential CRISPR target sites and assess their potential for off-target cleavage. Examples include the CRISPR Design Tool and the ZiFiT Targeter, Version 4.2 .

Applications as a Genome-editing and Genome Targeting Tool

Following its initial demonstration in 2012, the CRISPR/Cas9 system has been widely adopted. This has already been successfully used to target important genes in many cell lines and organisms, including human, bacteria, zebrafish, *C. elegans*, plants, *Xenopus tropicalis*, yeast, *Drosophila*, monkeys, rabbits, pigs, rats and mice. Several groups have now taken advantage of this method to introduce single point mutations (deletions or insertions) in a particular target gene, via a single gRNA. Using a pair of gRNA-directed Cas9 nucleases instead, it is also possible to induce large deletions or genomic rearrangements, such as inversions or translocations. A recent exciting development is the use of the dCas9 version of the CRISPR/Cas9 system to target protein domains for transcriptional regulation, epigenetic modification , and microscopic visualization of specific genome loci.

The CRISPR/Cas9 system requires only the redesign of the crRNA to change target specificity. This contrasts with other genome editing tools, including zinc finger and TALENs, where redesign of the protein-DNA interface is required. Furthermore, CRISPR/Cas9 enables rapid genome-wide interrogation of gene function by generating large gRNA libraries for genomic screening.

The future of CRISPR/Cas9

The rapid progress in developing Cas9 into a set of tools for cell and molecular biology research has been remarkable, likely due to the simplicity, high efficiency and versatility of the system. Of the designer nuclease systems currently available for precision genome engineering, the CRISPR/Cas system is by far the most user friendly. It is now also clear that Cas9's potential reaches beyond DNA cleavage, and its usefulness for genome locus-specific recruitment of proteins will likely only be limited by our imagination.

NEB expressions Issue I, 2014

https://www.neb.com/tools-and-resources/feature-articles/crispr-cas9-and-targeted-genome-editing-a-new-era-in-molecular-biology

Text 5. Scientists Discover Children's Cells Living in Mothers' Brains

The connection between mother and child is ever deeper than thought

The link between a mother and child is profound, and new research suggests a physical connection even deeper than anyone thought. The profound psychological and physical bonds shared by the mother and her child begin during gestation when the mother is everything for the developing fetus, supplying warmth and sustenance, while her heartbeat provides a soothing constant rhythm.

The physical connection between mother and fetus is provided by the placenta, an organ, built of cells from both the mother and fetus, which serves as a conduit for the exchange of nutrients, gasses, and wastes. Cells may migrate through the placenta between the mother and the fetus, taking up residence in many organs of the body including the lung, thyroid, muscle, liver, heart, kidney and skin. These may have a broad range of impacts, from tissue repair and cancer prevention to sparking immune disorders.

It is remarkable that it is so common for cells from one individual to integrate into the tissues of another distinct person. We are accustomed to thinking of ourselves as singular autonomous individuals, and these foreign cells seem to belie that notion, and suggest that most people carry remnants of other individuals. As remarkable as this may be, stunning results from a new study show that cells from other individuals are also found in the brain. In this study, male cells were found in the brains of women and had been living there, in some cases, for several decades. What impact they may have had is now only a guess, but this study revealed that these cells were less common in the brains of women who had Alzheimer's disease, suggesting they may be related to the health of the brain.

We all consider our bodies to be our own unique being, so the notion that we may harbor cells from other people in our bodies seems strange. Even stranger is the thought that, although we certainly consider our actions and decisions as originating in the activity of our own individual brains, cells from other individuals

are living and functioning in that complex structure. However, the mixing of cells from genetically distinct individuals is not at all uncommon. This condition is called chimerism after the fire-breathing Chimera from Greek mythology, a creature that was part serpent part lion and part goat. Naturally occurring chimeras are far less ominous though, and include such creatures as the slime mold and corals.

Microchimerism is the persistent presence of a few genetically distinct cells in an organism. This was first noticed in humans many years ago when cells containing the male "Y" chromosome were found circulating in the blood of women after pregnancy. Since these cells are genetically male, they could not have been the women's own, but most likely came from their babies during gestation.

In this new study, scientists observed that microchimeric cells are not only found circulating in the blood, they are also embedded in the brain. They examined the brains of deceased women for the presence of cells containing the male "Y" chromosome. They found such cells in more than 60 percent of the brains and in multiple brain regions. Since Alzheimer's disease is more common in women who have hadmultiple pregnancies, they suspected that the number of fetal cells would be greater in women with AD compared to those who had no evidence for neurological disease. The results were precisely the opposite: there were fewer fetal-derived cells in women with Alzheimer's. The reasons are unclear.

Microchimerism most commonly results from the exchange of cells across the placenta during pregnancy, however there is also evidence that cells may be transferred from mother to infant through nursing. In addition to exchange between mother and fetus, there may be exchange of cells between twins *in utero*, and there is also the possibility that cells from an older sibling residing in the mother may find their way back across the placenta to a younger sibling during the latter's gestation. Women may have microchimeric cells both from their mother as well as from their own pregnancies, and there is evenevidence for competition between cells from grandmother and infant within the mother.

What it is that fetal microchimeric cells do in the mother's body is unclear, although there are some intriguing possibilities. For example, fetal microchimeric cells are similar to stem cells in that they are able to become a variety of different tissues and may aid in tissue repair. One research group investigating this possibility followed the activity of fetal microchimeric cells in a mother rat after the maternal heart was injured: they discovered that the fetal cells migrated to the maternal heart and differentiated into heart cells helping to repair the damage. In animal studies, microchimeric cells were found inmaternal brains where they became nerve cells, suggesting they might be functionally integrated in the brain. It is possible that the same may be true of such cells in the human brain.

These microchimeric cells may also influence the immune system. A fetal microchimeric cell from a pregnancy is recognized by the mother's immune system partly as belonging to the mother, since the fetus is genetically half identical to the mother, but partly foreign, due to the father's genetic contribution. This may "prime" the immune system to be alert for cells that are similar to the self, but with some genetic differences. Cancer cells which arise due to genetic mutations are just such cells, and there are studies which suggest that microchimeric cells may stimulate the immune system to stem the growth of tumors. Many more microchimeric cells are found in the blood of healthy women compared to those with breast cancer, for example, suggesting that microchimeric cells can somehow prevent tumor formation. In other circumstances, the immune system turns against the self, causing significant damage. Microchimerism is more common in patients suffering from Multiple Sclerosis than in their healthy siblings, suggesting chimeric cells may have a detrimental role in this disease, perhaps by setting off an autoimmune attack.

This is a burgeoning new field of inquiry with tremendous potential for novel findings as well as for practical applications. But it is also a reminder of our interconnectedness.

Scientific American December 4, 2012
http://www.scientificamerican.com/article/scientists-discover-childrens-cells-living-in-mothers-brain/

Text 6. We Need More Proof That Prenatal Gene Screens Are Beneficial

Blood tests are safer for pregnant women but do not tell the whole truth

Expecting a baby often provokes mixed emotions—wonder and amazement but also concern. Will the child be healthy? Happy? Find his or her spot in the world? Several prenatal blood tests are now available that attempt to ease some of

the anxiety—at least about health. By analyzing trace amounts of fetal DNA in a pregnant woman's bloodstream, these tests (which go by such names as Harmony, MateriT21 PLUS and verifi) can identify various genetic anomalies up to six months before birth. Whether or not parents to be take advantage of these laboratory measures is, of course, up to them. But results from screening tests can be misleading, and industry and federal regulators are not doing enough to ensure that people get all the information they need.

At present, the tests detect major abnormalities—such as three copies of the 21st, 18th or 13th chromosome, which lead, respectively, to Down, Edwards and Patau syndrome. These measures are a definite safety improvement over earlier procedures to check the genes of the unborn. Previously such chromosomal abnormalities could be detected prenatally only by invasive tests, such as amniocentesis, which carry a small risk of triggering a miscarriage.

The new screens were originally offered to women older than 35 years, who are at a higher risk of delivering babies with Down syndrome or other genetic maladies. But now companies are marketing such tests to women with low-risk pregnancies as well. Last November a market research firm reported that the tests accounted for $563.4 million worth of sales in 2014—a figure that is expected to quadruple by 2022.

Federal regulations have not caught up with the advancing technology, however. Under current rules, such gene screens are considered lab tests, which, unlike new drugs, do not have to show they offer clinically meaningful benefits. Instead manufacturers only need to demonstrate that their tests generate results within certain statistically acceptable limits of error.

This standard made more sense in the days when blood tests looked primarily for individual compounds, such as sugar molecules or hormones. Gene tests are different: they take a lot more interpretation and analysis to determine if a suspicious result indicates a true genetic aberration or merely a benign variation.

For one thing, the amount of fetal DNA found in the maternal bloodstream is minute and must be copied many times to generate enough material to test. The amplification process, among other things, may lead to double counting mistakes that give the false impression of an extra 21st chromosome, for example, where none exists. A second source of uncertainty stems from the fact that the new genetic tests are actually screening tests, which, by definition, cast a broad net that includes many more false-positive results than more specialized diagnostic procedures that are typically more accurate.

Ignoring this distinction can lead to serious problems. If a screening test on 1,000 people correctly identified 19 out of 20 true cases of a genetic problem, it would have what statisticians call a sensitivity rate of 95 percent. That sounds

pretty good, but that same test might also yield 10 false positives—10 other people in the group for whom the test incorrectly suggested a problem. The sensitivity rate would still be 95 percent because the test caught most of the true positives. But just over 65 percent of all the positive results—19 out of 29—were actually correct.

It is this last ratio—what statisticians called the positive predictive value—that tells you how much faith you should really have in a particular test result. And yet most gene-screening companies do not provide the positive predictive values for their tests. Instead they tout their tests' sensitivity rate, which can mislead patients and even their physicians. Problems with prediction are why anyone who receives a positive result from a screening test should follow it up with a more precise diagnostic exam.

The U.S. Food and Drug Administration also needs to follow up on these tests. It should accelerate efforts to change the rules so that the makers of gene screens give more clinically relevant information, such as predictive values. The companies that offer noninvasive prenatal screening should do more to educate all of us about a test's potential drawbacks. And expectant parents should think carefully about whether they want to undergo these screenings in the first place, particularly because new blood tests that supposedly provide a glimpse into the entire fetal genome—including possible predispositions to heart disease, cancer or diabetes—are just around the corner.

Scientific American February 1, 2016
http://www.scientificamerican.com/article/we-need-more-proof-that-prenatal-gene-screens-are-beneficial/

Text 7. Monkeys Are Genetically Modified to Show Autism Symptoms

But it is unclear how well the results match the condition in humans

The laboratory monkeys run obsessively in circles, largely ignore their peers and grunt anxiously when stared at. Engineered to have a gene that is related to

autism spectrum disorder in people, the monkeys are the most realistic animal model of the condition yet, say their creators. Researchers hope that the animals will open up new ways to test treatments and investigate the biology of autism. But the jury is still out on how well the monkeys' condition matches that of people with autism.

Autism has a vast array of symptoms and types, but researchers think that at least 100 genes play a part. The scientists who led the latest work, which is published on 25 January in *Nature*, turned to the autism-related gene *MECP2*: both people who have extra copies of the gene (*MECP2*-duplication syndrome) and people who have certain mutations in this gene (Rett's syndrome) share many of the symptoms of autism. Previously researchers have engineered monkeys to have autism-related genes, but this is the first published demonstration of a link between those genes and the animals' behavior.

Back in 2010, the team, led by researchers at the Chinese Academy of Sciences' Institute of Neuroscience in Shanghai attached human *MECP2* genes to a harmless virus, which they injected into the eggs of crab-eating macaque monkeys (*Macaca fascicularis*) before fertilizing them. The developing embryos were then implanted into female monkeys. The result was eight genetically manipulated newborns, who had between one and seven extra copies of *MECP2*. Examinations of other, stillborn monkeys revealed that the extra copies were being expressed in the brain. "That was the first exciting moment," says Zilong Qiu, a molecular biologist at the Institute of Neuroscience and a co-author on the paper.

The next breakthrough came about a year later when the monkeys showed behaviors that hinted at autism: running around in tight circles in a strange manner. "If another monkey is in its way, it will either jump over the monkey, or go around it, but then it would return to its original circular path," says co-author Sun Qiang, a reproductive biologist at the institute.

The team launched a battery of behavioral tests, which showed that all of the monkeys had at least one autism-like symptom, such as repetitive or asocial behavior, and that the symptoms were more severe in males, as seen in people with the *MECP2* duplications. But this still wasn't enough to be sure that the monkeys were a sound model of autism — and a paper that the team submitted for publication in 2013 was rejected. Among other things, reviewers wanted to know whether the unusual behavior was just the result of fiddling around with the genome. "We needed to show where the gene makes a difference," says Qiu.

That opportunity came with the next generation of macaques, which the team created with unprecedented speed. When the monkeys were 27 months old and not yet sexually mature, Sun's team took testes from the males, matured the tissue artificially by grafting it under the skin on the backs of castrated mice, and

used the resulting sperm to fertilize eggs from non-engineered macaques. The offspring showed asocial behavior at about 11 months. That both gene and symptoms seemed to be passed on to a second generation was finally enough to convince reviewers, says Qiu.

The macaque model is "superior" to existing mouse models of autism because "it actually shows more clearly some of the autism-like behaviors", says Alysson Muotri, who researches stem cells, autism and Rett's syndrome at the University of California, San Diego. But he adds that the symptoms in both mice and monkeys still seem less severe than "what we actually observe in human patients". "It remains to be seen if the model can actually generate novel insights into the human condition," he says.

Huda Zoghbi, a pioneer of *MECP2* studies in mice at Baylor College of Medicine in Houston, Texas, is even more cautious. The monkeys do not mimic some of the human *MECP2*-duplication symptoms, such as seizures and severe cognitive problems, she notes. This could be because the expression of the gene in the monkey model is triggered by a different mechanism from that in humans — a limitation that the authors recognize — and she advises caution in using the model to make assumptions about human autism.

Qiu, meanwhile, is excited by the prospect of using the model to identify exactly where in the brain the *MECP2* overexpression causes trouble. His team is already using brain-imaging technology on the monkeys to pinpoint such areas. Next, the researchers plan to use the CRISPR gene-editing technique to knock out the extra *MECP2* copies in cells in those regions and then check whether the autisim-like symptoms stop.

It is unlikely that such a technique would be approved for use in people any time soon. But the regions identified in the monkey study could be targeted with other, existing treatments — such as deep brain stimulation, which has had success for Parkinson's disease and depression. Because the structure of the mouse brain is so different from that of human brains, Qiu says that the monkey imaging will allow more parallels to be drawn with humans than mice studies could.

Working with a mental-health hospital, the team is also trying to identify the autism-linked genes that are most common in the Chinese population.

If non-human primates prove to be a useful model for psychiatric disorders, China and other countries that are investing heavily in research on monkeys, such as Japan, could gain an edge in brain research. Muotri says that such studies probably wouldn't be done in the United States, for example, where research on non-human primates is more expensive and controversial than it is in Japan or China. "China and Japan have a clear advantage over the US on this area," he says.

Scientific American January 25, 2016
http://www.scientificamerican.com/article/monkeys-are-genetically-modified-to-show-autism-symptoms/

Text 8. Why Studying Fertility in Sea Urchins Makes Sense [Excerpt]

Google "sea urchin fertilization" and you'll find dozens of animations and videos of lone sea urchin sperm finding its way home to an egg

Admittedly, spontaneous sex on the subway is a bit of a stretch (and probably nauseating for anyone who rides the G train on a regular basis). But the idea that sex depends upon a crowd is no exaggeration—especially for the less-than-agile lot.

Take sea urchins. A few leaky males can cause an entire nearby cluster of sea urchins to unleash clouds of sperm, which they pump out through holes in the tops of their heads. As is so often the case, males tend to let loose their loads first, followed by the females. It's a familiar pattern, occurring in sea cucumbers and abalone, among others. With sea cucumbers—squishy, sausage-shaped cousins of sea stars and sea urchins—researchers think the slight delay between male and female spawning times might help increase fertilization rates. The sperm released by males forms a dense cloud just off the bottom through which the females' buoyant eggs must float on their way to the surface.

In general, bottom-dwelling, or benthic, invertebrates such as sea cucumbers, sea stars, and sea urchins don't tend to travel very far, certainly not when compared with migrating bluefin, but even compared with horseshoe crabs. (Deep-sea species may be an exception, as they have to scavenge across fair distances for sparse food supplies.) But around coastal and shallow seas, many invertebrates stick close to home—tubed feet can only take you so far. When it's time to have sex, these species huddle up with their neighbors as individuals broadcast millions (sometimes billions) of sperm and eggs into the currents.

In an ironic twist, this strategy for boosting fertilization success can also pose a significant threat: even in the enormous expanse of sea, there can be such a thing as too much sperm. For most eggs in the animal kingdom, polyspermy—multiple sperm penetrating an egg—is fatal (those choosey female Beroë ctenophores are a rare exception). To understand what's going on, we've got to

dive down to the microscopic front line where sperm meets egg. The battle of the sexes rages on even at this unicellular level, and, believe it or not, sea urchins are the go-to animal for studying this kind of stuff. An extremely diverse group, sea urchins can be the size of a small brown bur or enormous, such as the softball-sized, long-spined black sea urchins, which wield four- to twelve-inch needlelike spines. They look more like a medieval weapon than the underwater lawn mowers they truly are. Impressive to behold, disastrous to touch, sea urchins can be found from the shallows to thousands of feet deep, from the warmest tropical seas to the undersea plains of the Arctic Ocean.

Google "sea urchin fertilization" and you'll find dozens of animations and videos of lone sea urchin sperm finding its way home to an egg. Why the plethora of sea urchin sex tapes? Because studying fertilization in sea urchins makes sense. They are easy to keep in aquaria, spawn on command (a quick injection of potassium chloride, aka sea urchin ejaculation juice, is all it takes), their gametes are easy to collect once in the water, and because fertilization takes place outside the body, it's much easier to observe and manipulate than, say, inside an elephant. And, not insignificantly, PETA has yet to march on behalf of captive sea urchins.

So, anyone out there who has ever received fertility treatments, any of the hundreds of thousands of couples who have successfully tried IVF, and especially any of the estimated five million or more people who now exist because of such assisted reproductive technology—thank sea urchins. All of those innovations stand on the shoulders of a basic understanding of what actually happens when sperm and egg collide. And we know that from studying sea urchins.

Scientific American February 13, 2016
http://www.scientificamerican.com/article/why-studying-fertility-in-sea-urchins-makes-sense-excerpt/

Text 9. Antibiotics in Animal Feed May Endanger Kids, Doctors Warn

The widespread practice of giving antibiotics to healthy livestock to promote growth and prevent disease is making the drugs ineffective, the American Academy of Pediatrics says

(Reuters Health) - Overuse of antibiotics in animal feed is making it harder for doctors to treat life-threatening infections in young children, a report from U.S. pediatricians warns.

The report from the American Academy of Pediatrics (AAP) says the widespread practice of giving antibiotics to healthy livestock to promote growth and prevent disease among animals is making the drugs ineffective when they are needed to treat infections in people.

“The antibiotics that are fed to the animals lead to the development of antibiotic resistant bacteria in the animal,” study co-author Dr. Theoklis Zaoutis of the University of Pennsylvania and the Children’s Hospital of Philadelphia said by email. “These bacteria can then be spread to other animals, the environment and to humans.”

More than two million Americans become ill with antibiotic-resistant infections each year, and 23,000 die as a result, Zaoutis and co-author Dr. Jerome Paulson report in the journal Pediatrics. Paulson formerly chaired the executive committee of the AAP’s Council on Environmental Health,

They estimate that national costs to the U.S. healthcare system attributable to antibiotic resistant infections run from $21 billion to $34 billion annually.

Infants and children are affected by antibiotic-resistant bacteria in the food supply, direct contact with animals and exposure in the environment, the researchers report.

For most infections, incidence was highest among children under age five, according to data the researchers cited from Center for Disease Control and Prevention's Foodborne Diseases Active Surveillance Network.

While people can't get antibiotics without a prescription, animals can, the researchers point out.

Pediatricians and parents can help combat antibiotic resistance by avoiding use of antibiotics to treat colds or other viral illnesses.

Parents and other consumers may also help discourage the use of antibiotics in livestock feed by choosing to buy only organic products or foods labeled as "raised without antibiotics," said Urvashi Rangan, executive director of the Consumer Reports Food Safety and Sustainability Center.

"Consuming foods from animals produced without the routine use of antibiotics is one important step in reducing personal risk; so is cooking our foods thoroughly," Rangan, who wasn't involved in the report, said by email.

But the long-term solution to antibiotic resistance may require changes in the way we produce animals for food, including stopping the use of antibiotics and other drugs in healthy animals and also implementing better drug-free hygiene and management practices to curb disease risk on farms, Rangan added.

Even purchasing organic doesn't guarantee that there will not be resistant bacteria present, noted Timothy Landers, an antibiotics researcher at Ohio State University in Columbus.

"From a farmer's perspective, the use of antibiotics helps ensure that food is safe, nutritious and affordable," Landers, who wasn't involved in the study, said by email. "What we have lacked is a coordinated, integrated approach to antibiotic resistance including experts on human health, food production animal health and the environment."

Scientific American November 16, 2015

http://www.scientificamerican.com/article/antibiotics-in-animal-feed-may-endanger-kids-doctors-warn/

Text 10. Gene-Modified Tomatoes Churn Out Healthy Nutrients

Plants, engineered to make extra substances that protect human cells, show GMO crops may improve health

A variety of tomato that has been genetically engineered to produce large quantities of potentially health-boosting compounds—including flavanols and anthocyanins—has been developed by researchers in the UK.

A single tomato of the new variety contains the same amount of resveratrol as 50 bottles of red wine, or the same amount of genistein (a compound found in soy beans that is thought to have health benefits) as 2.5kg of tofu. As tomato plants grow quickly and produce a lot of fruit, farming this new variety could be a way to produce these nutrients in industrial quantities much more cheaply than synthesising them chemically, or extracting small amounts from other plant sources.

The variety was made by introducing a gene from the model plant *Arabidopsis thaliana*—called AtMYB12—into the tomato genome. The gene codes for a transcription factor that binds to the promoter regions of genes encoding various metabolic enzymes. 'In *Arabidopsis* [the] MYB12 [transcription factor] regulates the production of flavonols … which are important in UV protection and signalling,' says Cathie Martin, who led the study at the John Innes Centre in Norwich, UK.

When AtMYB12 is introduced into tomato plants, it switches on metabolic pathways that shift the plant's energy and carbon pathways towards the production of flavonols and phenylpropanoids as the fruit develops. This causes these desirable metabolites to build up in the fruit. 'As much as 10% of the carbon in the fruit can be flavonols,' says Martin. 'This high level accumulation is a special feature of AtMYB12 because it targets both primary and secondary metabolism

and increases the supply of aromatic amino acid precursors as well as ATP and reducing power.'

By introducing additional plant genes—such as the enzymes necessary for the production of resveratrol in grapes—the plants can be engineered to produce specific compounds that are thought to have certain nutritional or health benefits. 'The tomato is a wonderful production system. If metabolic engineering is targeted to the end of fruit development, the fruit can serve as a bag in which to accumulate natural products, without impacting yield,' says Martin.

Broadening the debate

As well as being a good way to produce large quantities of the target nutrients, Martin says the tomatoes could potentially be sold for consumption down the line. 'If the different compounds we have engineered in tomato can be shown to protect against chronic diseases in preclinical studies, we could consider applying for regulatory approval for commercial sales. This we are doing with high anthocyanin tomato juice which has well-proven protective effects.'

Martin adds that the AtMYB12 gene could also be engineered into other fruits, and potentially certain vegetables where the products could be accumulated in a storage organ such as a tuber.

'This research gives us a better understanding of how healthy nutrients are genetically controlled in tomato, and maybe other fruits,' comments Huw Jones, a senior research scientist at agricultural science institute Rothamsted Research. 'It is a great example of how biotechnology could provide health benefits directly to consumers.' He adds that the work 'will further broaden the debate on GMOs in the EU'.

Martin agrees that boosting plants' ability to produce compounds that are seen as healthy may help improve the public perception of genetically engineered food. 'I think when consumers see a product which offers a benefit to them, and which could not be derived by natural breeding methods, they will understand much better the potential that biotech crops have to benefit society.'

Scientific American November 2, 2015

http://www.scientificamerican.com/article/gene-modified-tomatoes-churn-out-healthy-nutrients/

Web – Resources and Support

Web links to some useful and helpful resources

1. http://www.scientificamerican.com/article/snakebite-antivenom-development-is-stuck-in-the-19th-century-what-s-next/
2. http://www.scientificamerican.com/article/improving-humans-with-customized-genes-sparks-debate-among-scientists1/
3. http://www.scientificamerican.com/article/where-to-draw-the-line-on-gene-editing-technology/
4. http://www.scientificamerican.com/article/childhood-cancer-risk-hides-in-families/
5. http://www.scientificamerican.com/article/general-anesthesia-causes-no-cognitive-deficit-in-infants/
6. http://www.scientificamerican.com/article/bee-symbiosis-reveals-life-s-deepest-partnerships-q-a/
7. http://www.scientificamerican.com/article/disguised-nanoparticles-slip-past-body-s-immune-defense/
8. http://www.scientificamerican.com/article/where-could-the-first-crispr-baby-be-born/?
9. http://www.scientificamerican.com/article/the-woman-who-stared-at-wasps/
10. http://www.scientificamerican.com/article/dinosaurs-evolved-in-a-startlingly-short-time/
11. http://blogs.scientificamerican.com/guest-blog/beyond-resveratrol-the-anti-aging-nad-fad/
12. http://www.nature.com/news/crispr-tweak-may-help-gene-edited-crops-bypass-biosafety-regulation-1.18590
13. http://www.scientificamerican.com/article/a-battle-of-the-sexes-is-waged-in-the-genes-of-humans-bulls-and-more/
14. http://www.scientificamerican.com/article/biotech-interest-in-mini-organs-booms/
15. http://www.scientificamerican.com/article/synthetic-biology-s-first-malaria-drug-meets-market-resistance/
16. https://www.neb.com/tools-and-resources/feature-articles/crispr-cas9-and-targeted-genome-editing-a-new-era-in-molecular-biology
17. http://www.scientificamerican.com/article/scientists-discover-childrens-cells-living-in-mothers-brain/
18. http://www.scientificamerican.com/article/we-need-more-proof-that-prenatal-gene-screens-are-beneficial/

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