MINISTÉRIO DA EDUCAÇÃO
UNIVERSIDADE FEDERAL DA INTEGRAÇÃO LATINO-AMERICANA
INSTITUTO LATINO-AMERICANO DE CIÊNCIAS DA VIDA E DA NATUREZA
CENTRO INTERDISCIPLINAR DE CIÊNCIAS DA VIDA
PROGRAMA DE PÓS-GRADUAÇÃO EM BIOCIÊNCIAS
PROCESSO SELETIVO REGULAR 2021.1
PROCESSO SELETIVO REGULAR 2022.1
PROFICIÊNCIA EM LÍNGUA INGLESA

ANEXO III DO EDITAL PPG-BC Nº. 2022/24
PRUEBA DE DOMINIO DEL IDIOMA INGLÉS

Esta prueba es un requisito para que los estudiantes de maestría obtengan el título de maestría en Ciencias y una de las evaluaciones de los PSR's (Procesos Selectivos Regulares), de aspirantes a estudiantes regulares, del curso de maestría del PPG-BC (Programa de Posgrado en Biociencias), en los primeros semestres académicos de los años 2021 y 2022, regulados por los Edictos PPG-BC n. 2020/12 y n. 2021/29, sus correcciones y resultados.

Esta prueba evaluará a cada candidato por su capacidad para leer y comprender textos o artículos científicos en inglés, relacionados con el área de conocimiento del PPG-BC.

Esta prueba constituye etapa del proceso de selección; tiene carácter eliminatorio; contiene 12 (doce) preguntas referentes a 04 (cuatro) resúmenes de textos en inglés. Para responderlas, marque solo una alternativa por pregunta en la plantilla a continuación, sin tachaduras. Las respuestas en la plantilla con tachaduras o más de una alternativa marcada no se tendrán en cuenta.

La prueba evaluará a los candidatos a través de la comprensión de la idea central del texto y la interpretación y resolución de cuestiones relacionadas con el texto original.

Para ser aprobado en esta etapa de selección, es necesario obtener una calificación igual o superior a 50 (cincuenta) puntos. Esta prueba tendrá un valor de hasta 100 (cien) puntos, siendo 08,33 (ocho punto treinta y tres) puntos por pregunta.

Le recordamos que durante esta prueba está prohibida la consulta o uso de equipos o instrumentos electrónicos y/o audiovisuales. Sin embargo, sólo se permite el uso de diccionarios impresos.

La aplicación de esta prueba comenzará en el campus Jardim Universitário, edificio Gimnasio, oficina G-102-2, el día 26 de agosto de 2022, a las 19h00 y terminará a las 22h00, fecha límite para que los candidatos entreguen este hoja de respuestas identificada al PPG- BC

¡Les deseamos a todos buena suerte!

Foz do Iguaçu, Estado do Paraná, 26 de agosto de 2022

IDENTIFICACIÓN DE ESTUDIANTE DE MÁSTER
Nombre completo:
Registro:
Firma:

RESPUESTA DEL ESTUDIANTE DE MAESTRÍA											
Preguntas del primer texto			**Preguntas del segundo texto**			**Preguntas del tercer texto**			**Preguntas del cuarto texto**		
1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**
A	A	A	A	**A**	A	A	A	A	A	A	A
B	B	B	B	B	**B**	B	B	**B**	B	**B**	**B**
C	C	**C**	C	C	C	C	C	C	**C**	C	C
D	D	D	D	D	D	**D**	D	D	D	D	D
E	**E**	E	**E**	E	E	E	**E**	E	E	E	E

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

Texto 1. Las preguntas 01, 02 e 03 se refieren al texto de SANTOS-SÁNCHEZ, Norma Francenia; SALAS-CORONADO, Raúl; VILLANUEVA-CAÑONGO, Claudia; HERNÁNDEZ-CARLOS, Beatriz; *Antioxidant Compounds and Their Antioxidant Mechanism. In:* ***Antioxidants***. [S.l.]: Emad Shalaby, 2018. DOI: 10.5772/intechopen.85270. Disponible en: < https://www.intechopen.com/chapters/66259 >. Consultado el: 15/08/2022.

> *Oxidative stress in biological systems is a complex process that is characterized by an imbalance between the production of free radicals (FR) and the ability of the body to eliminate these reactive species through the use of endogenous and exogenous antioxidants. During the metabolic processes, a great variety of reactions take place, where the promoters are the reactive oxygen species (ROS), such as hydrogen peroxide (H2O2) and the superoxide radical anion (O2 •−), among others. A biological system in the presence of an excess of ROS can present different pathologies, from cardiovascular diseases to the promotion of cancer. Biological systems have antioxidant mechanisms to control damage of enzymatic and nonenzymatic natures that allow ROS to be inactivated. The endogenous antioxidants are enzymes, such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase, or non-enzymatic compounds, such as bilirubin and albumin. When an organism is exposed to a high concentration of ROS, the endogenous antioxidant system is compromised and, consequently, it fails to guarantee complete protection of the organism. To compensate this déficit of antioxidants, the body can use exogenous antioxidants supplied through food, nutritional supplements, or pharmaceuticals. Among the most important exogenous antioxidants are phenolic compounds carotenoids and vitamins C and some minerals such as selenium and zinc.*
>
> *In the study of antioxidant compounds and the mechanisms involved, it is important to distinguish between the concepts of antioxidant activity and capacity. These terms are often used interchangeably. However, antioxidant activity refers to the rate constant of a reaction between an antioxidant and an oxidant. The antioxidante capacity is a measure of the amount of a certain free radical captured by an antioxidant sample [1]. Therefore, during the selection of a method, the response parameter must be considered to evaluate the antioxidant properties of a sample, which may be a function of the concentration of the substrate or concentration and the time required to inhibit a defined concentration of the ROS.*
>
> *The reaction mechanisms of the antioxidant compounds are closely related to the reactivity and chemical structure of free racicals (FR) as well as the environment in which these reactive species are found. Therefore, it is very important to describe the ROS and, to a lesser degree, the reactive nitrogen species (RNS), which include both precursors and free radicals.*
>
> *In the literature, there are many in vitro methods to evaluate the effectiveness of antioxidant compounds present in a variety of matrices (plant extracts, blood serum, etc.) using lipophilic, hydrophilic, and amphiphilic media (emulsions). The in vitro methods can be divided into two main groups: (1) hydrogen atom transfer (HAT) reactions and (2) transfer reactions of a single electron (SET). These methods are widely used because of their high speed and sensitivity. When carrying out a study related to the antioxidant properties of a sample, more than one method is usually used to evaluate the antioxidant capacity/activity [2].*

Pregunta 01. En el apartado "*In the study of antioxidant compounds and the mechanisms involved, it is important to distinguish between the concepts of antioxidant activity and capacity. These terms are often used interchangeably. However, antioxidant activity refers to the rate constant of a reaction between an antioxidant and an oxidant*", el término "*interchangeably*", significa que los términos "*antioxidante acitivity and capacity*":

a) se utilizan incorrectamente;
b) con frecuencia se usan como sinónimos;
c) rara vez son intercambiables;
d) son equivalentes; o
e) son antagónicos.

Pregunta 02. En cuanto al mecanismo de acción de los antioxidantes, el texto establece que:

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

a) el método utilizado para evaluar el mecanismo de acción no influye en la respuesta;
b) están relacionados con la estructura química de los radicales libres como los compuestos fenólicos;
c) que el medio no influya en la acción antioxidante;
d) sólo hay dos mecanismos de acción; o
e) se debe utilizar más de un método para desentrañar el mecanismo de acción.

Pregunta 03. La palabra "*closely*" en el tercer párrafo puede ser reemplazada sin perjuicio del contexto por:

a) *warily*;
b) *close*;
c) *nearly*;
d) *gingerly*; ou
e) *earshot*.

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

Texto 2. Las preguntas 04, 05 e 06 se refieren al texto adaptado de GROOT, P.; et al. *Faecal microbiota transplantation halts progression of human new-onset type 1 diabetes in a randomised controlled trial*. **Gut microbiota**. Amsterdam, n. 70, p. 92-105, *October,* 2021. DOI: <10.1136/gutjnl-2020-322630>. Disponible en: <https://gut.bmj.com/content/gutjnl/70/1/92.full.pdf?with-ds=yes>. Consultado el: 24/08/2022.

> *Type 1 diabetes (T1D) is characterised by islet autoimmunity and beta cell destruction. A gut microbiota–immunological interplay is involved in the pathophysiology of T1D. A research verified microbiota-mediated effects on disease progression in patients with type 1 diabetes using faecal microbiota transplantation (FMT).*
>
> *The researchers reported for the first time that FMT can have an effect on residual beta cell function in new-onset T1D. This accords with recent observational studies supporting a role for the intestinal microbiota in T1D subjects. In contrast to their hypothesis, autologous FMT performed better than healthy donor FMT, while even in the allogenic group, the decline in MMT stimulated C peptide response appeared less than expected in T1D without treatment in 1 year.*
>
> *The study selected patients with recent-onset (<6 weeks) T1D (18–30 years old) and randomised into two groups to receive three autologous or allogenic (healthy donor) FMTs over a period of 4 months. Primary endpoint was preservation of stimulated C peptide release assessed by mixed-meal tests during 12 months. Secondary outcome parameters were changes in glycaemic control, fasting plasma metabolites, T cell autoimmunity, small intestinal gene expression profile and intestinal microbiota composition.*
>
> *Among the results, they found that stimulated C peptide levels were significantly preserved in the autologous FMT group (n=10 subjects) compared with healthy donor FMT group (n=10 subjects) at 12 months. Small intestinal Prevotella was inversely related to residual beta cell function, whereas plasma metabolites 1-arachidonoyl- GPC and 1-myristoyl-2-arachidonoyl-GPC levels linearly correlated with residual beta cell preservation. Finally, baseline CD4 +CXCR3+T cell counts, levels of small intestinal Desulfovibrio piger and CCL22 and CCL5 gene expression in duodenal biopsies predicted preserved beta cell function following FMT irrespective of donor characteristics.*
>
> *An appealing explanation would be that beneficial immunological effects of FMT (irrespective of donor source) are more pronounced and durable when the FMT donor microbiota is more immunologically compatible with the host. They suspected that allogenic FMT increases the already present increase in inflammation that is known to occur around the time of diagnosis, by offering immunologically foreign colonic microbiota to which the host is less tolerant to the small intestine (where the T cells are thought to be trained), which may overshadow beneficial effects that occur simultaneously and are caused by different agents. In contrast to animal studies, the beneficial effect of FMT was not associated with changes in short-chain fatty acid (SCFA)-producing strains. Nevertheless, observations point towards an immunological regulatory role of specific plasma metabolites that are derived from diet and converted by intestinal microbiota.*
>
> *FMT halts decline in endogenous insulin production in recently diagnosed patients with T1D in 12 months after disease onset. Several microbiota-derived plasma metabolites and bacterial strains were linked to preserved residual beta cell function. This study provides insight into the role of the intestinal gut microbiome in T1D.*

Pregunta 04. En relación con el estudio presentado anteriormente, se puede decir que:

a) los investigadores informaron por primera vez que el trasplante de microbiota fecal puede tener un efecto sobre la función residual de las células beta en la DM1 tardía;
b) el resultado primario observado en el estudio fue una reducción en la liberación de péptido C evaluada mediante pruebas de comidas mixtas durante 12 meses;
c) el papel regulador inmunológico de metabolitos plasmáticos específicos es independiente de la dieta que recibe el individuo para la conversión de la microbiota intestinal;
d) se cree que los efectos inmunológicos beneficiosos del trasplante de microbiota fecal son menos pronunciados y duraderos cuando la microbiota del huésped es inmunológicamente más compatible con el donante; o

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

e) los investigadores creen que el trasplante alogénico de microbiota fecal puede aumentar la inflamación ya detectada en el momento del diagnóstico de DM1.

Pregunta 05. Se puede entender de la conclusión del estudio que:

a) el trasplante de microbiota fecal detiene la disminución de la producción de insulina endógena en pacientes con DM1 recién diagnosticados dentro de los 12 meses posteriores al inicio de la enfermedad;
b) varios metabolitos plasmáticos derivados de microbiota y cepas bacterianas se asociaron con la función conservada de las células beta residuales;
c) el estudio mostró que el papel del microbioma intestinal en la DM1 tiene una relevancia insignificante en el curso de la DM1 durante 12 meses;
d) el trasplante de microbiota fecal favorece la disminución de la producción endógena de insulina en pacientes con diagnóstico reciente de DM1, lo que requiere una intervención clínica rápida tras el diagnóstico inicial de la enfermedad; o
e) la presencia de bacterias, como Prevotella en el intestino delgado, estaba directamente relacionada con la preservación de la función residual de las células beta, impidiendo la evolución de la DM1.

Pregunta 06. En cuanto a las expresiones o términos traducidos que se encuentran en el texto, es correcto lo expuesto a continuación:

a) *an appealing explanation would be that beneficial immunological effects of FMT*
= una explicación aparente de los efectos inmunológicos beneficiosos del trasplante de microbiota fecal;

b) *a gut microbiota–immunological interplay is involved in the pathophysiology of T1D*
= una interacción entre microbiota intestinal e inmunidad está involucrada en la fisiopatología de la DM1;

c) *by offering immunologically foreign colonic microbiota*
= ofreciendo microbiota colónica inmunológicamente conocida;

d) *which may overshadow beneficial effects that occur simultaneously*
= que pueda esclarecer los efectos benéficos que ocurren simultáneamente; o

e) *nevertheless, observations point towards an immunological regulatory role of specific plasma metabolites*
= las observaciones no siempre muestran el papel inmunológico regulador de metabolitos plasmáticos específicos.

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

Texto 3. Las preguntas 07, 08 e 09 se refieren al texto de LOU, C.; *et al*. *Ribozyme-based insulator parts buffer synthetic circuits from genetic context.* ***Nature Biotechnology***. Berlin, *Springer Nature*, n. 30, p. 1.137-1.142, *October*, 2012. DOI: <10.1038/nbt.2401>. Disponible en: <https://www.nature.com/articles/nbt.2401>. Consultado el: 24/08/2022.

> *A fundamental principle of synthetic biology is that genetic parts can be independently characterized and used to predict their combined behavior. This principle underpins the high-throughput fabrication of valuable parts, the storage of useful part information in a registry and the use of these data for computer-aided design. In practice, however, the functions of parts are influenced by their genetic, environmental and cellular context. This limits the designability of genetic systems, because each part needs to be characterized in its final context.*
>
> *The function of a part can be affected by the sequence of neighboring parts, and we refer to this effect as 'part-junction interference'. For example, a barcode part was found to contain a sequence similar to the –10 region of the constitutive promoter (http://2010.igem.org/Team:UC_Davis/notebook/c0051debug.html). Depending on the sequence of the upstream part, a promoter can be spontaneously formed at the junction between the barcode and neighboring part. Similarly, promoter parts are often defined by a relatively short (~50 bp) sequence, but regions 100 bp or more upstream can affect promoter strength, and the effect of remote sequences can be reduced by including an insulator region. A strong hairpin sequence has also been shown to insulate the ribosome binding site (RBS) from the sequence of the 5′ UTR. Recently, it was shown that the processing of mRNA using CRISPR RNA processing elements could be used to reliably maintain relative promoter strengths and reduce interference between genes within an operon.*
>
> *Genetic circuits use biochemical interactions to implement a computational operation. Transcriptional circuits implement this operation on the level of promoters, where promoters serve as both the input and the output of the circuit. Thus, the connection of a new input to a circuit creates the possibility of junction interference. How the output of a logic gate changes as a function of the inputs is described by its transfer function. This should be an intrinsic property of the circuit, which is independent from the inducible system that is used to measure it. As part of building a program for an unrelated application, we characterized the transfer function of a circuit and serendipitously discovered the influence of part-junction interference. The circuit is a NOT gate where the signal of an input promoter is inverted by having it drive the expression of the CI repressor that turns off an output promoter.*

Pregunta 07. El primer párrafo introduce el concepto de biología sintética. ¿Cuál de las siguientes afirmaciones está de acuerdo con lo anterior?

a) las partes genéticas son los diferentes alelos asociados a un sistema genético;
b) en la práctica las partes genéticas pueden estudiarse independientemente unas de otras;
c) la biología sintética garantiza la predictibilidad del comportamiento de las partes genéticas;
d) en la práctica, no se garantiza al 100% la previsibilidad de las partes genéticas, dada su interacción con otras partes del sistema; o
e) es prácticamente imposible predecir el comportamiento de una parte genética, lo que hace inviable la biología sintética como herramienta práctica.

Pregunta 08. En cuanto a los promotores, el texto asevera que:

a) los promotores son las únicas partes genéticas cuyo comportamiento puede predecirse;
b) una parte genética que codifica un promotor puede ubicarse a miles de bases del gen regulado;
c) con biología sintética es posible obtener promotores mucho más pequeños (10bp) que los conocidos hasta ahora;
d) utilizando la tecnología conocida como CRISPR, en el contexto de la biología sintética, sería posible prescindir del uso de promotores; o
e) una estructura de ARN puede, en la región promotora, bloquear la traducción.

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

Pregunta 09. Los circuitos genéticos:

a) están formados por alelos;
b) función basada en interacciones bioquímicas;
c) son teorías computacionales que no pueden implementarse en la práctica;
d) dependen de elementos traslacionales que funcionan en retroalimentación; o
e) no puedem transmitirse a generaciones posteriores.

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

Texto 4. Las preguntas 10, 11 e 12 se refieren al texto adaptado de DOLGIN, E. *Longevity data hint at no natural limit on lifespan: death rates plateau in elderly people, reviving a debate about how long humans can live*. **Nature**. Berlin, *Springer Nature*, n. 559, p. 14-15, June, 2018. DOI: <10.1038/d41586-018-05582-3>. Disponible en: <https://www.nature.com/articles/d41586-018-05582-3?utm_source=briefing-dy&utm_medium=email&utm_campaign=briefing&utm_content=20180629>. Consultado el: 24/08/2022.

There might be no natural limit to how long humans can live–at least not one yet in sight.

***That proposal** – which runs contrary to the claims of some demographers and biologists– comes from a statistical analysis published on 28 June in Science. It examined the probabilities ofsurvival of nearly 4,000 "super-elderly" people in Italy, all aged 105 and older.*

*The study was led by Sapienza University demographer Elisabetta Barbi and University of Roma Tre statistician Francesco Lagona, both based in Rome. Their team found that the risk of death– which, throughout most of life, seems to increase as people age–levels off after age 105, creating a '**mortality plateau**'. At that point, the researchers say, the odds of someonedying from one birthday to the next are roughly 50:50.*

"If there is a mortality plateau, then there is no limit to human longevity," says Jean-Marie Robine, a demographer at the French Institute of Health and Medical Research in Montpellier.

That would mean that someone such as Chio Miyako, a Japanese great-great-great-grandmother who, at 117, is the world's oldest known person, could live for years to come–oreven forever, at least hypothetically.

Researchers have long debated whether humans have an upper age limit. The consensus holds that the risk of death steadily increases in adulthood, up to about age 80 or so. But there's vehement disagreement about what happens as people enter their 90s and 100s.

Some scientists have examined demographic data and concluded that there is a fixed, natural 'shelf life' for our species, and that mortality rates keep increasing. Others have looked at the same data and concluded that the death risk flattens out in one's ultra-golden years, and therefore that human lifespan does not have an upper threshold.

In 2016, geneticist Jan Vijg and his colleagues at Albert Einstein College of Medicine in NewYork City rekindled the debate when they analysed the reported ages at death for the world's oldest individuals over half a century. They estimated that human longevity hit a ceiling at about 115 years–125 tops.

Vijg and his team argued that given few, if any, gains in maximum lifespan since the mid-1990s, human ageing had reached its natural limit. The longest known lifespan belonged to Jeanne Calment, a French super-centenarian who died in 1997 at age 122.

Experts challenged the statistical methods in the 2016 study, setting off a firestorm into which Barbi and Lagona now step. Working with colleagues at the Italian National Institute of Statistics, the researchers collected records on every Italian aged 105 years and older between 2009 and 2015 – gathering certificates of death, birth and survival in an effort to minimize the chances of 'age exaggeration', a common problem among the oldest old.

They also tracked individual survival trajectories from one year to the next, rather than lumping people into age intervals as previous studies that combine data sets have done. And by focusing just on Italy, which has one of the highest rates of centenarians per capita in the world, they avoided the issue of variation in data collection between different jurisdictions.

As such, says Kenneth Howse, a health-policy researcher at the Oxford Institute of Population Ageing, UK, "these data provide the best evidence to date of extreme-age mortality plateaus inhumans.

Ken Wachter, a mathematical demographer at the University of California, Berkeley, and anauthorof the latest study, suspects that previous disputes over the patterns of late-life mortalityhave largelystemmedfrom bad records and statistics. "If we can get data of this quality for other countries, I expect we're going to see much the same pattern."

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>

Robine is not sosure. He says that unpublisheddata from France, Japan and Canada suggest that evidence for a mortality plateau is "not as clear cut". A global analysis is still needed to determine whether the findings from Italy reflect a universal feature of human ageing, he says. Brandon Milholland, a co-author of the 2016 Nature paper, says that the evidence for a mortality plateau is "marginal", because the latest study included fewer than 100 people who lived to 110 or beyond. Leonid Gavrilov, a longevity researcher at the University of Chicago in Illinois, notes that even small inaccuracies in the Italian longevity records could lead to aspurious conclusion.

Others say the conclusions of the study are biologically implausible. "You run into basic limitations imposed by body design", says Jay Olshansky, a bio-demographer at the University of Illinois at Chicago, noting that cells that do not replicate, such as neurons, will continue towitherand die as a person ages,placing upper boundaries on humans' natural lifespan.

This study is thus unlikely to be the last word on the age-limit dispute, says Haim Cohen, a molecular biologist at Bar-Ilan University in Ramat-Gan, Israel. "I'm sure that thedebate is going to continue."

Pregunta 10. Marque la alternativa INCORRECTA respecto a la información presentada en el texto sobre la propuesta a la que se refiere la expresión "*that proposal*" (línea 02):

a) es una investigación realizada por un demógrafo y un estadístico;
b) el estudio no tiene aceptación unánime;
c) la propuesta argumenta que, después de los 105 años de edad, las posibilidades de supervivencia de un ser humano son prácticamente nulas;
d) los sujetos de la investigación son italianos de 105 años o más; o
e) el resultado de la investigación fue publicado en la revista Science, en el primer semestre.

Pregunta 11. La expresión "*mortality plateau*" (línea 08), tal como se utiliza en el texto, se refiere a:

a) los factores que determinan los niveles de mortalidad entre dos o más regiones;
b) una estabilización del riesgo de muerte después de los 105 años de edad;
c) las causas de muerte entre los ancianos participantes en un estudio;
d) una mayor tasa de mortalidad después de los 100 años de edad; o
e) el aumento del riesgo de muerte a medida que aumenta la edad.

Pregunta 12. Marque la alternativa que NO CORRESPONDA a la información contenida entre las líneas 10 y̆ 17:

a) para Robine, es cierto que no hay límite para la longevidad humana;
b) actualmente, la persona más anciana del mundo es una mujer china de 117 años;
c) la existencia o no de un límite de años de vida humana es una vieja preocupación entre los investigadores;
d) existe consenso entre los investigadores de que el riesgo de muerte aumenta hasta alrededor de los 80 años; o
e) hay desacuerdo sobre las predicciones de vida de las personas que entran en los años 90 y 100.

campus Jardim Universitário, Av. Tarquínio Joslin dos Santos, nº. 1.000, Edifício Ginásio, Sala nº. G-103-1
bairro Polo Universitário, Foz do Iguaçu, Estado do Paraná, Brasil, CEP nº. 85.870-901
Correio eletrônico <secretaria.ppgbc@unila.edu.br> - Telefone: +55 (45) 3522-9935 - <https://portal.unila.edu.br/mestrado/biociencias/>