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Trabajo Fin de Grado

An ELF Approach to False Friends in Pronunciation in the Context of English for Science and Technology

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RESUMEN

La pronunciación de palabras en inglés en el ámbito científico internacional puede ser muy problemática, lo cual puede llevar a la falta de o error en la comunicación entre los miembros de este campo en términos de inteligibilidad. La razón por la que un mensaje llega a ser ininteligible es porque el interlocutor cree haber oído otra palabra (y por consiguiente, otro significado) o incluso por no haber entendido nada. Este estudio se centra en aquellas palabras cuya ortografía es igual o similar en español e inglés, pero difieren a nivel fonético, en el patrón acentual o ambos. Esta idea de semejanza formal se ha unido a la noción de "falso amigo".

Dada la inexistencia de pautas normativas en cuanto a la pronunciación estándar de tecnicismos científicos, esta disertación tiene como objetivo demostrar la dificultad que conlleva la interacción entre científicos de diferentes nacionalidades en un contexto de inglés como Lingua Franca. Para ello, se han tomado como punto de partida dos variedades generales del inglés, RP (*Received Pronunciation*) y GA (*General American*). La selección de palabras utilizada en este estudio proviene de diferentes fuentes: resumen, clases teóricas y glosarios.

Este material se ha analizado desde un punto de vista cualitativo y cuantitativo. Mediante el estudio cualitativo se ha demostrado dónde se pueden encontrar las diferencias tanto a nivel segmental como suprasegmental. Además, el estudio cuantitativo ha servido para aclarar con qué frecuencia aparecen dichas dificultades.

El análisis de este trabajo se ha desarrollado a través de la comparación de la clasificación propuesta por Roca Varela y mi propia clasificación, concluyendo que es de gran importancia ser conscientes de dónde pueden estar las diferencias en pronunciación de dos palabras formalmente similares o iguales en dos idiomas diferentes ya que puede llevar a una interpretación errónea del mensaje.

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1. INTRODUCTION

This dissertation aims to prove that the pronunciation of words that have a formal resemblance in English and Spanish in the field of science may turn out to be very problematic and lead to speakers´ frustration and misunderstandings in spite of the etymology of those words. This study is going to be based on the context of English as a Lingua Franca (ELF), that is, on interactions in English between non-native speakers.

Nowadays the use of English in the field of international science is very frequent. A great amount of scientific literature is published in English so that it can be understood by all this particular community around the world. However, not only written publications are produced in English, but also lectures and conferences, among others, take place in this language, which has become the most relevant medium of communication. Being able to communicate in English adequately in this field of knowledge has always been very troublesome, both for native and non-native speakers, since it is very hard to find the correct pronunciation of technical words because of the lack of appropriate dictionaries of pronunciation of scientific terms.

Regarding the etymology of scientific terms, they come predominantly from Greek and Latin. It is not useful for the present study to keep in mind the origins of those technical terms since knowing the etymology of those words does not in this case come to the aid of providing any guidance on the pronunciation of those terms, but only on their meaning.

One of the main problems appears when an L2 speaker faces a word that is formally similar to another word in their L1. This is likely to provoke a breakdown in communication because of "the transfer of L1 sounds" (Jenkins, 2000, p. 88). Hence, non-native speakers of English will have to be careful when talking not only to native speakers (English as a Foreign Language (EFL)), but to other non-native speakers from

other nationalities in the context of English as a Lingua Franca (ELF), which is the central setting of my study.

The idea of formal resemblance between two words from different languages is linked in this study to the notion of false friends in pronunciation. The study of false friends remains a compelling subject for L2 learners of English to become more proficient in their learning process. The concept of false friends has been discussed by authors such as Chamizo Domínguez (2006), who defines them as "two given words which are similar or equivalent graphically or phonetically in two or more given languages but have different meanings" (p. 426) Nonetheless, most of them centre their studies and reflections around on the meaning, but not around the form of those false friends. Hence, they propose their own classifications taking semantics into account, a goal that is not related to the present study. Another type of classification of false friends is the one proposed by Postigo Pinazo (1997). Her classification is divided into four categories: "(a) phonetic false friends; (b) graphic false friends; (c) false friends derived from loanwords; and (d) semantic false friends, which could be subdivided into total or partial" (as cited in Chacón Beltrán, 2006, p.:33). This classification is too wide and extensive for my analysis but, if that were the classification of choice, all my study would be focused on the phonetic false friends category.

In spite of the existence of apparent shortcomings in the different typologies of false friends, the most suitable classification that I have found and that can be applied to my study is the one put forward in María Luisa Roca-Valera´ s doctoral thesis. She proposes three types of false friends regarding their formal resemblance: "orthographic false friends", "phonetic false friends" and "ortho-phonetic false friends" (Roca-Varela, 2012, p. 23). However, this classification still has a disadvantage, namely, that false

friends are classified paying attention to whether there are formal similarities as well as differences:

- Orthographic FF: similar spelling, different pronunciation
- Phonetic FF: similar pronunciation, different spelling
- Ortho-phonetic FF: similar spelling and pronunciation

For the present study false friends have to be understood as words that show formal resemblance in Spanish and English, that is, words that may have the same or similar spelling, but do differ in pronunciation at the segmental, suprasegmental or at both levels at the same time. This is the reason why I have proposed my own classification of false friends regarding pronunciation which will be compared to Roca-Varela´s classification and which will help to draw some conclusions about the difficulties of pronouncing not only technical but also common words that are also used in the scientific field. In addition, the parameters of the Non-Core Features stated by Jenkins (2000) will be applied to characterize the context of English as a Lingua Franca (ELF). .

Finally, I have to add that my main motivations for choosing this topic are, first, my interest for sciences since I was very young as I had always studied sciences until I entered the BA in English Studies; second, my conception that speaking is a skill as important as writing and teachers would have to make more emphasis on it, as pointed out by Sakale (2012), and third, I think it is of great interest for students of sciences to be able to have a conversation in English using their technical language and being aware of where the difficulties in pronunciation are, especially in the current situation in which many Spanish scientists have to migrate to other European countries, such as Germany, France and Sweden, seeking for a job related to their studies.

This dissertation is organised into several parts. First, I explain the methodology I have followed in this study to carry out my analysis since it is based on a wide variety of sources; second, results are commented on, including those obtained according to Roca-Varela´ s classification as well as those obtained according to mine. These results are presented through a table and two different graphs for each classification: the table contains the total numbers and percentages obtained; the graphs, one is a column graph so that results from the different texts can be observed and the other, a pie graph necessary to show the global results in a broader perspective. Finally, a discussion of the main conclusions from my analysis is provided.

2. METHODS

This study has been carried out during the second semester of the academic year 2014-2015. This has allowed me to carry out the analysis in the context of English as a Lingua Franca (ELF), leaving behind that of English as a Foreign Language (EFL); that is to say, the analysis has been done in connection with a context in which English is used "among speakers of different first languages for whom English is the communicative medium of choice, and often the only option" (Seidlhofer, 2011, p. 7)

Three different types of materials have been included in the analysis: at the rhetorical level, one abstract and two lectures; at the lexical level, two wordlists which have functioned as glossaries in this study. Written and spoken registers have been used in order to demonstrate that the same kind of lexis is utilised in both of them within the scientific field. The abstract has been taken from an article entitled "Heat Reduction in Semiconductors by Phonon Annihilation". The lectures come from MICASE (Michigan Corpus of Academic Spoken English): the first one is entitled "Structure and Reactivity II Lecture" and the second one, "Biology of Cancer. Lecture" In the case of the two

wordlists, they have been taken from the course books *English for Environmental Science in Higher Education Studies* and *English for ICT Studies in Higher Education Studies*. The selection of words from the previous books belongs to word sets that are said to be of particular use to students of those fields.

The three specific genres (abstract, lecture and glossary) are shared by different discourse communities, that is, they are related to "bounded groups of people (defined respectively by the texts they use and by the practices they engage in together)." (Scollon, 2012, p. 9) Here, the discourse communities I am referring to are health science, computing science, environmental science and engineering. Broadly speaking, this study focuses on the discourse system of science. The notion of discourse system has to be understood as a broader term than discourse community, since it is discourse system that is normally associated with very large groups of people who share ideas and beliefs about the world, who have the same behaviours when meeting other people, who use the same type of texts and whose methods of learning are very similar. (Scollon, 2012)

In the case of sciences, it is almost compulsory to know English if you are not an English-native speaker, since the great majority of scientific publications are produced in English. Consequently, it is a must to put both productive skills (speaking and writing) into practice since the scientific community differs from others "not only [in] the grammatical, lexical, and phonological features of their language…, but also [in] the topics they choose to talk about, the way they present information, the style with which they interact, in other words, [in] their discourse accent" (Kramsch, 1998, p. 7). The problem appears when undergraduates have to learn English oriented to a specific purpose (English for Specific Purposes), such as English for Environmental Science or English for ICT Studies (like the examples in the glossaries), but this is done with a

considerable emphasis on the learning of written language whereas the spoken language is almost forgotten. Hence, those students will have many difficulties when dealing with speech events such as lectures and conferences, as their pronunciation may not be understood because incorrect phonemes are used or because they are not able to place the word stress appropriately.

In order to round off the process of data collection for my study, three different Spanish-speaking EST students were interviewed: one junior student from design engineering, one senior student from computer science and one postgraduate from nursing. All these students have an intermediate level of English but they have had a very slight amount of exposure to technical English at university. The three of them come from the University of Zaragoza and only the nursing student was offered an optional module on English for Nursing.

After the above five sources of information were established, a selection of words was made in order to determine the words that appear to be false friends for the pronunciation of scientific and technical English by Spanish speakers. The selection includes both technical terms and common words of interest from the field in English that have a formal resemblance to Spanish words. These words have been treated in isolation, that is to say, the noun phrases that appeared in the glossaries were divided into single words so as to keep the same paradigm for both classifications.

The analysis was in the first place carried out on the basis of the classification proposed by Roca-Varela. However, this classification does not really pay attention to where the differences and the subsequent difficulties and mistakes reside in terms of pronunciation. Thus, I set out to propose my own classification dealing with false friends in English and Spanish which have a formal resemblance but differ in the pronunciation of specific phonemes, in the stress pattern or in both. Consequently, my

classification is more exclusive and it can only be applied when there is a consistent difference at the segmental level (Phoneme False Friends), at the suprasegmental level (Stress False Friends) or at both levels at the same time (Phoneme-Stress False Friends).

As I have previously said, this analysis takes place in the current context of English as a Lingua Franca, that is, a context in which English is used as a medium for communication among non-native English speakers. This means that in this situation certain aspects in pronunciation have to be prioritised whereas others have to be set aside. It was in the 1980s with the arrival of Communicative Language Teaching that there was a complete change in those priorities. As Stevens (1989) put forward, the reason why these changes happened was that "in the absence of complete mutilation of the phonemes by the non-native speaker, the suprasegmentals will carry the day because they bear the meaning of the message." (p. 183) However, yet nowadays teaching materials prepare students for interactions with native English speakers. (Walker, 2010, p. 26)

Regarding the previous mismatch, Jennifer Jenkins, among others ELT experts, studied the use of English pronunciation in the context of English as a Lingua Franca in both classroom and social settings. (Jenkins, 2000) Her data was recollected from non-native English speakers that were studying English in the UK and whose level was from "upper-intermediate to low advanced level". (2000, p.87) As a result, she could realise the fact that "pronunciation was found to be the most important cause of breakdowns in ELF communication." (Walker, 2010, p. 26) Consequently, she was able "to establish the Lingua Franca Core, a list of pronunciation items central to maintaining mutual intelligibility of ELF" (2010, p. 27) As well as the Lingua Franca Core, she put forward several Non-Core Features. These features are those that do not create intelligibility in

the context of ELF, but which paradoxically, do create intelligibility in the context of English as a Foreign Language (EFL).

In the present study, the analysis has been done using both Received Pronunciation and General American pronunciations as the starting point for both classifications because, as Spicer (2011) explained, "in order to safeguard mutual intelligibility [in ELF contexts]…there is general agreement that a degree of native speaker accent is desirable, but that this should be regarded not as the 'norm'…, but a point of reference, a 'model' for guidance and approximation" (p. 1). Therefore, although taking both varieties of English as points of departure, there are some important phonological features in both varieties which belong to the Non-Core Features proposed by Jennifer Jenkins that have to be clarified in terms of their use for this study. The phonological features I am referring to are:

- Word stress: Jenkins described word stress as a "grey area" (2000, p.150) because it can cause unintelligibility for native speakers. Nevertheless, the only situation in which there can be a breakdown is if there is a change in both word stress and a certain sound. (Walker 2010, p. 40) Hence, I had placed word stress in the "dark area", that is, from my point of view, word stress can cause misunderstandings since the words cannot be identified properly in terms of grammar, even if there is no phonological change in the nuclear syllable (the syllable that carries the stress).
- Vowel reduction, schwa, and weak forms: Jenkins found that "despite the fact that it is easy to formulate clear rules about weak form use, they are unteachable." (Jenkins 2000, p. 147) Taking this fact into account, my classification is not going to bear in mind any vowel reduction, the schwa and any weak form in the unstressed syllables of words since the message can be

understood if the word stress is properly placed in the word. However, the diphthongisation of any vowel in the stressed syllable, or even in unstressed words, is going to be taken into account, too, because it is a relevant change in the pronunciation of a sound that may cause a breakdown in communication.

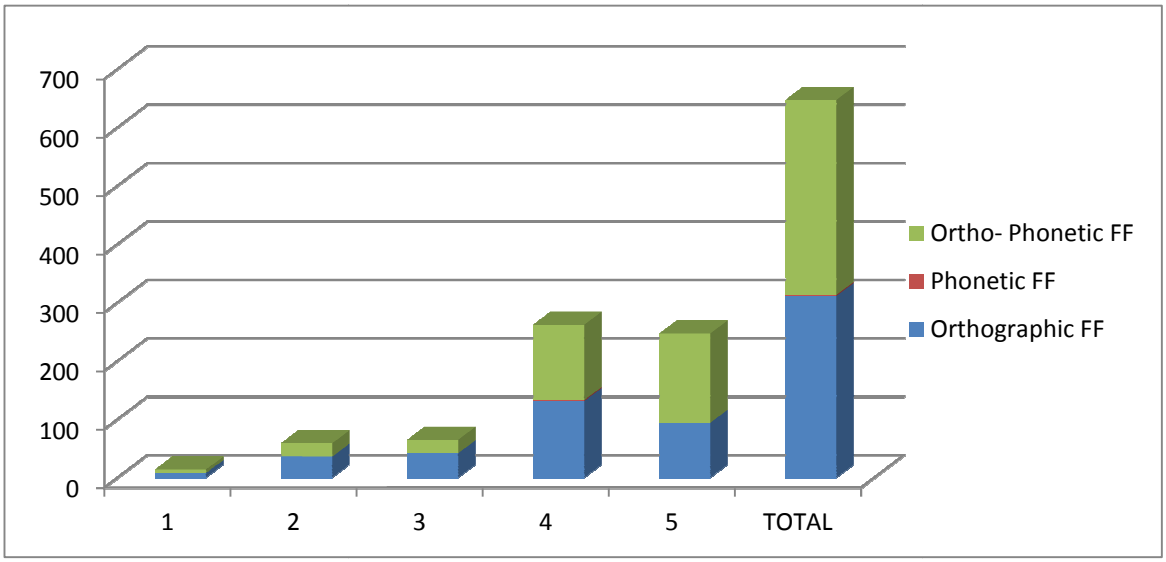
3. RESULTS

The comparison of both classifications yields a number of outstanding results. First, as I have mentioned in the previous section, the classification that I have proposed is more restrictive than that of Roca-Varela. This leads to a reduction of the total number of words to which we have to pay attention. As can be seen below, 5.8% of the total of words is not considered as being problematic according to my classification (647 in Table 1.1 and 609 in Table 2.1).

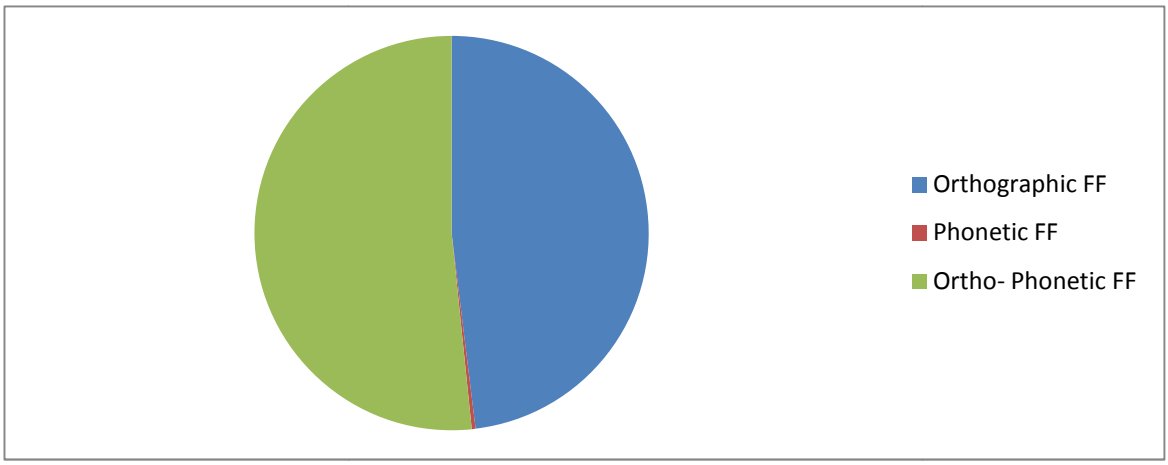
3.1. Frequency of false friends according to Roca-Varela´s classification:

TABLE 1.1	TOTAL (ROCA-VARELA´S CLASSIFICATION)			
	Orthographic FF	**Phonetic FF**	**Ortho- Phonetic FF**	**TOTAL**
1	8	0	6	14
2	36	0	23	59
3	42	0	23	65
4	131	2	129	262
5	94	0	153	247
TOTAL	311	2	334	**647**
TOTAL %	**48%**	**0%**	**52%**	100%

GRAPH 1.2



GRAPH 1.3



As can be seen in Table 1.1, the total of words that have been analysed is 647, including those words whose pronunciations differ sufficiently in RP and GA and those compounds which have been considered as single words in terms of pronunciation. These words are “epsilon” (Text 2), “motile” (Text 3), “diversity” and “stratum” (Text 4), and “privacy” (Text 5). The British pronunciation of “epsilon”, “motile” and “diversity” has been placed in the column of Orthographic FF as they have a similar spelling to the Spanish words but differ in pronunciation. However, the British pronunciations of “stratum” and “privacy” have been grouped in the column of Ortho-

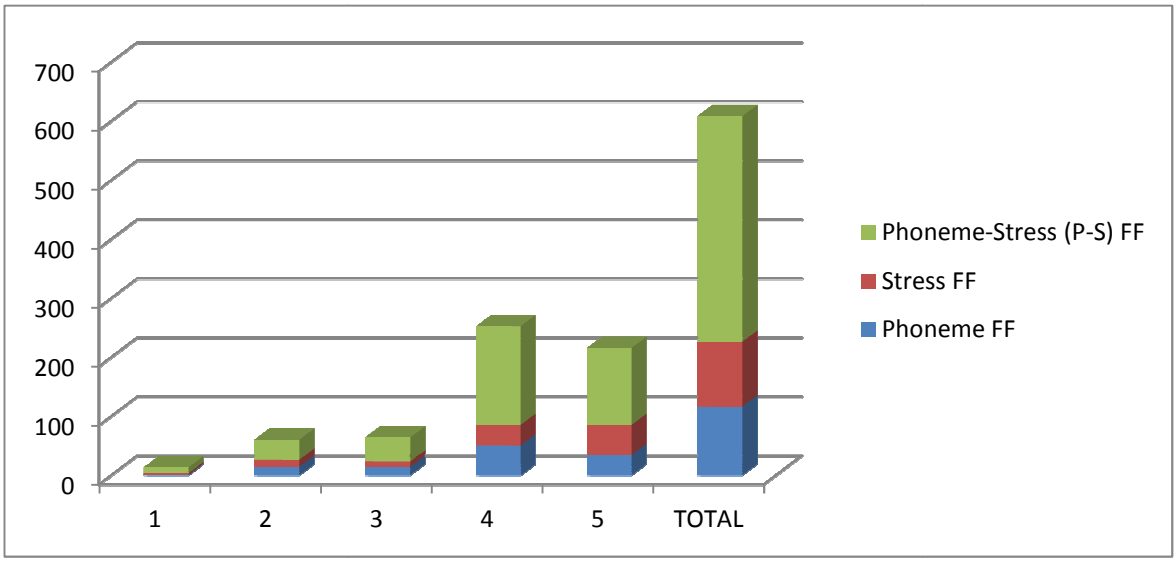
phonetic FF, as they are more similar to the Spanish words in terms of both form and pronunciation

It can be seen that there is 0% (exactly, 0, 003%) of Phonetic FFs (those that have a similar pronunciation but different spelling). The only two words that have been placed in this column are “gorilla” and “salmonella”, but even the difference in spelling of both words is minor. It can be seen as a diminutive red line in column 4 and in the total column of Graph 1.2., which represents these two cases. Therefore, most words have been distributed almost equally between the Orthographic FF column (311 words: 48%) and the Ortho-Phonetic FF one (334 words: 52%). These results show that those words that have a similar spelling present almost 50% of possibilities of having either a similar or a different pronunciation. (Graph 1.3)

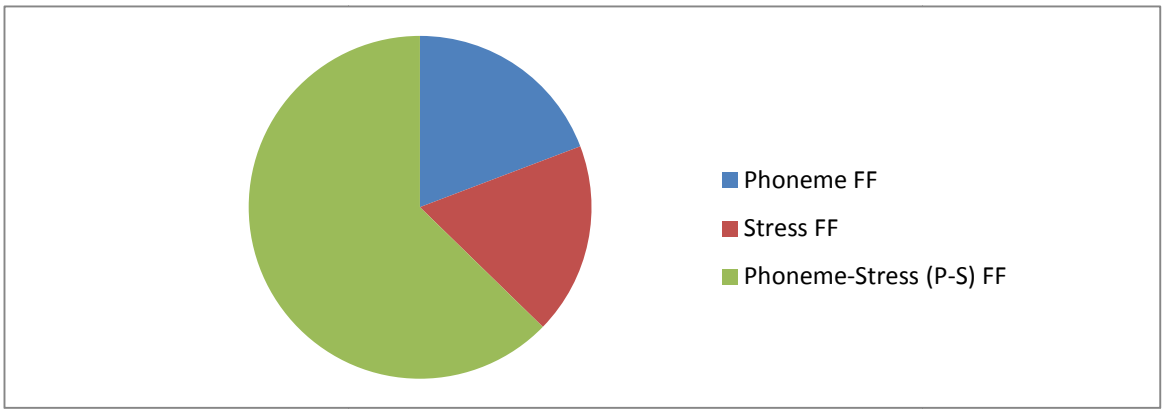
3.2.Frequency of false friends according to my classification.

TABLE 2.1	TOTAL (MY PROPOSED CLASSIFICATION)			
	Phoneme FF	**Stress FF**	**Phoneme-Stress FF**	**TOTAL**
1	1	3	10	14
2	15	12	34	61
3	15	9	41	65
4	51	35	167	253
5	35	51	130	216
TOTAL	117	110	382	609
TOTAL (%)	**19%**	**18%**	**63%**	100%

GRAPH 2.2



GRAPH 2.3



The number of words has been restricted to a total of 609, as can be seen in Table 2.1, taking into account once again both RP and GA pronunciations as well as isolated words. The words that have a significant variation in their pronunciation depending on these two accents and a type of variation that is relevant for this classification are “epsilon” and “methylamine” (Text 2), “nutrient” and “tumor” (Text 3), “altitude”, “consume” and “stratum” (Text 4), and “consume” again (Text 5). In this case, the British pronunciation of all these words has been classified in the column of Phoneme-Stress FFs, as they have differences at both levels.

382 out of 609 words are Phoneme-Stress FFs, that is, 63% of words belong to this group. There are 117 Phoneme FFs (19%) and 110 Stress FFs (20%). They correspond approximately to two fifths of the total of words (Graph 2.3). In this case, the table shows that more than half of the words present variations both at the segmental and at the suprasegmental level. The number of words that differs in either phonemes or word stress is far less significant, but they cannot be overlooked.

As I said before, Jenkins pointed out that the key problem in the context of English as a Lingua Franca is when the erroneous placement of stress comes together with unexpected phonemes. This case represents 63% of words that have been analysed. Consequently, it is really worth studying these words because they can be troublesome for students of sciences, without underestimating those that only differ at one of the two levels because they constitute a mere 37%, that is, 227 out of 609 words.

4. DISCUSSION

It has been shown above how the formal resemblance of pairs of words in English and Spanish can be dangerous and problematic for Spanish users of English in the field of science and it is, therefore, necessary to pay attention to this potential source of communicative difficulties. Leaving apart the semantics of words, this study has focused on those words that have a similar spelling but differ in pronunciation in terms of variations at the segmental, suprasegmental, or both levels. The issue of false friends is more prominent in some fields than in others as Chacón (2006) stated:

> "false friends are not exceedingly common in Spanish- English but in some contexts they represent a true learning problem as they become rather frequent. So, although their occurrence is not too high, it is not so scarce as not to require special attention on the part of researchers and language teachers." (p. 32)

This is the case of the field of sciences in which many words do not even have a standard pronunciation which represents a challenge for Spanish speakers at an epoch when “more than ever before, we hear and speak science as well as read it.” (Flood, 1957, p. 20) The words in question may go unnoticed because of their formal resemblance, when the case is that they carry the most common errors in pronunciation. As a consequence, students, in this case from the scientific field, should be aware of the pronunciation of those words as well.

The analysis of the words according to two classifications has allowed me, firstly, to show that when dealing with false friends in pronunciation, it is essential to go over the aspects that may lead to failure when a Spanish speaker tries to communicate in English with people from other nationalities. This is because those mispronunciations may not occur when the mother tongue is other than Spanish. Consequently, becoming aware of those aspects will certainly help to avoid any misinterpretation in communication. Secondly, it is not worth paying attention to where the similarities of false friends in pronunciation lie but where the variations can be found in order to avoid misunderstandings in the interaction with other speakers. Last but not least, most problems seem to arise when both phonemes and word stress are used incorrectly although words that only have changes at one of the two levels should not be disregarded. since in statistical terms they show enough proportion as well. However, I must point out that, in my opinion, in the context of ELF the most important false friends that should be studied are not only Phoneme- Stress FFs, but Phoneme FFs, because in the context of pronunciation the segmental level is the simplest and most basic level of language that needs to be used appropriately.

The exclusion of almost 6% of the total of words used in Roca-Varela´s classification has helped to focus on those words that really create a problem in

pronunciation. It must be indicated that the words that have been excluded come mostly from Text 4 and Text 5 (glossaries) because, in contrast to Texts 1, 2 and 3, those words have not been taken from complete texts examined in search of problematic items, but come from two long lists of words which are judged to be particularly useful in Environmental Science (Text 4) and ICT Studies (Text 5). There is only one word in Text 3 which has been excluded.

The 6% of words that have been excluded comprises:

- Loanwords from English that have been adopted into Spanish (above all, in ICT studies). As Castillo (2006) put forward, nowadays some of the semantic fields that have increased their number of Anglicisms [in Spanish] are, among others, "physical and natural sciences and computer sciences, fundamentally." (p. 11) There are two examples in Text 4: "smog" and "stock", but the majority of these words have been found in Text 5 (computer science). These are "blog", "botnet", "chip", "copyright", "flash", "gigabyte", "hacker", "hardware", "joystick", "malware", "online", "phishing", "spider", "tracking" and "web".

  One aspect that I want to highlight is the fact that these loanwords are pronounced in English by Spanish-speaking scientists in some cases as if they were read literally as we do in Spanish, as I noticed in my interview to the student of computer science, who pronounced, for example, "online" as /ˈɒnˌlaɪn/ but "plug-in" as /'plug ɪn/. As a consequence, those words that were pronounced in a Spanish way have not been excluded, although my interviewee said that they were totally aware of the correct pronunciation but it was simply a matter of convenience when talking in class. Furthermore, this is not an isolated case as the same situation happened in a study taken with Polish students who

"commented on such cases in the following way: `They look like Polish words so when I see them, I pronounce them in the Polish way though I know it´s wrong´." (Pawlak, Waniek-Klimczak and Majer, 2011, p. 291)

- Other terms with a slight level of exposure which are used in the field of sciences and which do not create any problems in pronunciation. In Text 3, "metastasis" is the only word to be excluded. In Text 4 these are "agronomist", "aluminium", "analytical, "atom", "catalytic", "deposit", "flora", "fossil", "genetic", "genetically", "graphical", "graphics", "habitat", "pest", "salmonella" and "toxic". In Text 5 these are "analytical", "digit", "digital", "magnetic", "optimal", "optimum", "scan", "scanner", "transmit" and "valve".
- Words to which students have already had some level of exposure, not only in the scientific field but in everyday language. Words excluded from Text 4 are "analysis", "atmospheric", "conserve", "convert", "gas", "map", "organic", "plant", "standard". In Text 5 these are "cost", "disk", "electronic", "physical", "technical", "technological" and "text".

The parameters for the exclusion of words because of a high or a low level of exposure, that is, because of the frequency with which they occur in communication have been applied according to the Longman Communication List. This list gathers "the 3000 most frequent words in both spoken and written English, based on statistical analysis of the 390 million words contained in the Longman Corpus Network – a group of corpuses or databases of authentic English language." (Longman, n.d., p. 1) This list already contains many words that are very often used in the scientific field. This means that there has been lexical acculturation from sciences to everyday communicative settings.

Regarding the interview with the three students of science, they felt astonished when they realised how many difficulties they had in pronouncing the words proposed from the five texts, words that they knew and understood when reading them, but felt incapable of pinpointing in terms of pronunciation. I was able to notice that they tended to pronounce many words with an American accent, rather than with a British one. There seem to be two reasons for this. The first is a historical reason and deals with the fact that there is a recurrent Spanish influence on America since the first settlement led by Christopher Columbus in 1942 which persists until nowadays because of the proximity of the USA to Latin America and the consequent Latin immigration to the USA. A second but also important reason is social. It has to do with the fact that nowadays Spanish-speaking learners only listen to English on the internet when they see films, play computer games or listen to music whenever they are not receiving any instruction in English. This made me think that most audiovisuals are produced in the USA and that, therefore, they are now more exposed to the American accent.

The specific pronunciations that led me to believe that Spanish speakers´ pronunciation of scientific terms is more similar to the American accent are:

- The monograph -o- was always pronounced as /oʊ/ (GA) and not /əʊ/ (RP). Examples of this case are in Text 1 “phonon” (/ˈfəʊn ɒn ‖ ˈfoʊn ɑːn/), in Text 2 “proton” (/ ˈprəʊt ɒn/ ‖ /ˈproʊt ɑːn/), in Text 3 “fibroblast” (/ ˈfaɪb rəʊ blɑːst ‖-roʊ blæst/), in Text 4 “decompose” (/ˌdiː kəm ˈpəʊz‖ -ˈpoʊz/) and in Text 5 “decode” (/diː ˈkəʊd ‖ -ˈkoʊd/)
- The pronunciation of the postvocalic -r- (typical of rhotic accents like General American). The interviewees always pronounced the monograph -r- no matter where it was placed within the word. There are some examples in Text 1 “transport” (/ˈtræns pɔːt‖ pɔːrt/), in Text 2 “energy” (/en ədʒ i ‖ -rdʒ i/), in Text 3

"tumor" (/ ˈtjuːm ə ‖ ˈtuːm r/), in Text 4 "carbon" (/ˈkɑːb ən ‖ ˈkɑːrb-/) and in Text 5 "microprocessor" (/ˈmaɪk rəʊ ˌprəʊs es əl -rə ˌprɑːs es r/).

By contrast, they did pronounce, for example, the /t/ sound as a fortis plosive between vowels in unstressed syllables, which does not happen in GA, where it is pronounced similar to /d/ (technically known as flap 't'). This sound is transcribed as /ɾ/. Some examples are in Text 3 "activator" (/ ˈækt ɪ veɪt ə ‖ -veɪɾ r/), in Text 4 "abiotic" (/ˌeɪ baɪ ˈɒt ɪk ‖ -ˈɑːɾ ɪk/) and in Text 5 "connectivity" (/ˌkɒn ek ˈtɪv ət i‖ ˌkɑːn ek ˈtɪv əɾ i/). The reason for this mixture is that as learners of English, Spanish speakers are influenced by many accents of English, from which they acquire different pronunciation features and end up mixing them without even noticing it, producing an amalgam of accents.

Moving on to the pronunciation in ELF, the previous aspects may or may not cause intelligibility in conversation. Robin Walker (2010, p. 130) proposed "the most relevant threats for intelligibility" that Spanish learners of English can face in the context of ELF. He explains that all vowels in Spanish are released with the same length and this is why it is so difficult for us to distinguish short and long vowels in English and be able to articulate those sounds correctly when they are to be long. The repercussion of this fact may not produce intelligibility with everyday language (e.g. pairs like 'rid/read' and 'ship/sheep'), but I do not think this will cause unintelligibility in sciences as terms in this field are very different from everyday language, at least when the vowels happen to be in unstressed syllables. Some examples can be in Text 2 "alanine" (/ˈæl ə niːn /), in Text 3 "protein" (/ˈprəʊt iːn‖ ˈproʊt-/), in Text 4 "carnivore" (/ˈkɑːn ɪ vɔː‖ ˈkɑːrn ə vɔːr/) and "decompose" (/ˌdiː kəm ˈpəʊz‖ -ˈpoʊz/) and in Text 5 "augmented" (/ɔːg ˈment ɪd/). Regarding consonant sounds, producing a flapped /t/ or sounding the 'r' does not constitute a problem in ELF. Therefore, the conclusion that I

have reached is that the different aspects of conflict between RP and GA accents are not relevant in the context of ELF as the fundamental goal here is to establish certain parameters in pronunciation in order to avoid breakdowns in communication.

As regards word stress, it is important to know where it changes, particularly when the shift in stress involves uttering different phonemes. In Text 1, two clear instances are “annihilation” (/ə ˌnaɪ ə ˈleɪʃ n/) and “coherent”, /kəʊ ˈhɪər ənt ‖ koʊ ˈhɪr-/. Text 2 contains words such as “acetate” (/ˈæs ə teɪt/), “adjacent” (/ə ˈdʒeɪs nt/), “anion” (/ˈæn ˌaɪ‿ən/), “dipole” (/ˈdaɪ pəʊl ‖ -poʊl/), “lysine” (/ˈlaɪ si:n/) and “nitrogen” (/ ˈnaɪtr ədʒ ən/). In Text 3 there are examples, such as “biopsia” (/ ˈbaɪ ɒps i /), “enzyme” (/ˈen zaɪm/), “lymphocyte” (/ ˈlɪmf əʊ saɪt /), “matrix” (/ ˈmeɪtr ɪks/), “protease” (/ ˈprəʊt i eɪz / ‖ /ˈproʊţ-/) and “vital” (/ ˈvaɪt l/). Other samples from Text 4 are “biosphere” (/ˈbaɪ əʊ sfɪə ‖ -ə sfɪr/), “drainage” (/ˈdreɪn ɪdʒ/), “eutrophication” (/ ju ˌtrɒf ɪ ˈkeɪʃ n‖ -ˌtroʊf-/), “finite” (/ˈfaɪn aɪt/), “hydrology” (/haɪ ˈdrɒl ədʒ i ‖ -ˈdrɑ:l-/), “ion” (/ˈaɪ‿ən/), “radiation” (/ˌreɪd i ˈeɪʃ n/) and “viable” (/ˈvaɪ‿əb l/). In Text 5 it can be found instances, such as “archive” (/ˈɑ:k aɪv ‖ ˈɑ:rk-/), “creator” (/kri ˈeɪt ə‖ -ˈeɪţ r/), “geospatial” (/ˌdʒi: əʊ ˈspeɪʃ l ‖ -oʊ/), “hypertext” (/ˈhaɪp ə tekst ‖ -r-/), “private” (/ˈpraɪv ət/), “spiral” (/ˈspaɪr əl/) and “voltage” (/ˈvəʊlt ɪdʒ‖ ˈvoʊlt-/). In addition, it is important to point out that a change in the stress pattern may bring up a different word class, for example, in Text 5 the word “arithmetic”. This word, depending on whether it is pronounced as /ˌær ɪθ ˈmet ɪk/ or /ə ˈrɪθ mə tɪk/, becomes an adjective or a noun, respectively.

Those people interested in knowing the pronunciation of scientific words may be lucky, as was I, because they can find them in proper pronunciation dictionaries such as the Longman Pronunciation Dictionary where phonetic transcriptions are provided, and also in online pronunciation dictionaries where many technical terms of science are

gathered accompanied by a recording of the pronunciation of the word that can easily be listened to by clicking on the words. However, it is important to emphasise that there is no pronunciation dictionary that only comprises scientific and technical terms. There are many glossaries and bilingual dictionaries where a translation is offered, but without any guidance in terms of pronunciation. Doing some research on the internet, I found that there are many blogs created to enable people to ask for the pronunciation of technical terms. In these blogs someone wants to know the pronunciation of a specific word and then random people answer how they utter them. Therefore, searching for the pronunciation of words in sciences can be seen as an absolute chaos as there are still many terms that do not have a standardised pronunciation. It is striking that although scientific ideas keep flowing with the continual creation of new words, there is no apparent interest in giving them a standardized pronunciation, let alone in representing it with a transcription or any other device. To make things worse, the above problem is compounded by the fact that many of those words that have existed for a long time now and have become part of the terminology of the field are very likely to become false friends in pronunciation. For this reason, raising learners´ awareness of this situation of paramount importance is essential, but we need an ELF approach to keep things in perspective.

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6. APPENDIX

6.1. ROCA VARELA´S CLASSIFICATION

6.1.1. SOURCE 1: ABSTRACT ("Heat Reduction in Semiconductors by Phonon Annihilation")

ROCA VARELA´S CLASSIFICATION 1		
ORTHOGRAPHIC FF	PHONETIC FF	ORTHO-PHONETIC FF
annihilation		electron
circumstance		interference
coherent		phonon
generation		project
phase		semiconductor
reduction		transport
simulation		
structure		
8	0	6

6.1.2. SOURCE 2: STRUCTURE AND REACTIVITY II LECTURE

ROCA VARELA´S CLASSIFICATION 2		
ORTHOGRAPHIC FF	PHONETIC FF	ORTHO-PHONETIC FF
abundance		acidity
acetate		additional
acetic		alanine
adjacent		carboxylic
anion		common
archetypal		component
base		constant
basicity		dipole
concentration		distance
difference		electrode
enolate		electron
enzyme		electronegative
epsilon (RP)		electrophoresis
equation		energy
hexane		epsilon (GA)
hydrogen		equilibrium
hydronium		equivalent
ion		inductive
isoelectric		negative
lysine		positive
medicine		proton
methylamine		succinic
molecule		system
neutral		
nitrogen		
protein		
ratio		
resonance		
scientific		
soluble		

species		
stabilization		
stationary		
substance		
technique		
Zero		
36	0	23

6.1.3. SOURCE 3: LECTURE ON BIOLOGY OF CANCER

ROCA VARELA´S CLASSIFICATION 3		
ORTHOGRAPHIC FF	**PHONETIC FF**	**ORTHO-PHONETIC FF**
activator		aggressive
aorta		artificial
benign		capillaries
biopsy		cerebral
cascade		detriment
cause		doctor
chemotherapeutic		existence
circulatory		factor
concentration		fraction
culture		immune
cure		immunosuppressant
diagram		intestine
diet		metastasis
enzyme		motile (GA)
fibroblast		nodule
final		nutrient
frequent		osteosarcoma
gene		ovary
glycoprotein		process
heterogeneous		system
importance		transplantable
isotope		tumor
lymphocyte		unusual
metastasize		
matrix		
membrane		
microscope		
microscopic		
motile (RP)		
penetration		
peptide		
primary		
production		
protease		
protein		
radioactivity		
region		
secretion		
sequential		
space		
spinal		
vital		
42	0	23

6.1.4. SOURCE 4: ENGLISH FOR ENVIRONMENTAL SCIENCE (GLOSSARY)

ROCA VARELA´S CLASSIFICATION 4		
ORTHOGRAPHIC FF	**PHONETIC FF**	**ORTHO-PHONETIC FF**
abiotic	gorilla	access
agricultural	salmonella	acid
agriculture		aerosol
air		agronomist
antibiotic		altitude
area		aluminium
atmosphere		analysis
benzene		analytical
biodiversity		analyze
biological		animal
biomass		anthropogenic
biosphere		aquifer
biota		artificial
biotic		atmospheric
bromide		atom
bromine		bacteria
carbohydrate		capacity
climate		carbon
climatic		carcinogen
conservation		carnivore
consumption		catalytic
cultivate		categorize
cultivation		chlorine
cycle		chlorofluorocarbon
degradation		collection
denitrify		commercial
designation		community
detritus		complex
destruction		complexity
detritivore		component
detritus		composition
displacement		congestion
diversity (RP)		coniferous
drainage		conserve
ecosphere		consume
ecosystem		consumer
elevation		contaminant
erosion		contaminate
eutrophication		continental
evapotranspiration		continentality
excrete		convert
exposure		decompose
fauna		decomposer
fertilizer		decomposition
filtration		deposit
finite		dissipate
foliage		diversity (GA)
gaseous		ecologist
gene		ecology
generation		educate
geothermal		efficiency
hierarchy		emission
human		energy
hydrocarbon		evaporate
hydrology		evolution

hydrosphere		extinction
image		facility
impermeable		fertilize
incinerator		flora
industrialized		fossil
infrastructure		fuel
ion		gas
irrigation		generate
lithosphere		genetic
maritime		genetically
mechanization		geology
mesosphere		global
meteorology		graphical
methane		graphics
microorganism		habitat
migration		herbivore
molecule		hormone
monoculture		impact
monoxide		incinerate
multi-media		incorporate
mutate		inefficient
natural		intensive
nitrogen		interaction
non-renewable		interconnected
operation		inventory
permeable		irrigate
pesticide		map
photovoltaic		menu
phytoplankton		metamorphosis
porous		mineral
power		model
preservation		modify
primary		nuclear
primate		nutrient
private		organic
producer		organism
productivity		oxygen
public		ozone
radiation		particle
recyclable		particulate
recycle		pest
recycling		photosynthesis
reduce		photosynthesize
reduction		plant
region		preserve
renewable		preventive
scheme		process
separation		produce
site		project
social		reserve
spatial		residential
specialization		residue
species		resistance
sphere		respiratory
state		secondary
stratosphere		sedimentary
stratum (GA)		smog
subspecies		solar
substance		solvent

sustainability		spray
sustainable		standard
synthesize		statistical
technique		stock
temperature		stratum (RP)
tertiary		subsistence
thermal		system
thermosphere		tabular
transformation		technology
transportation		toxic
troposphere		transport
turbine		tropical
ultraviolet		variable
vapour		volume
vegetation		zoology
viability		
viable		
131	2	129

6.1.5. **SOURCE 5:** ENGLISH FOR ICT STUDIES (GLOSSARY)

ROCA VARELA´S CLASSIFICATION 5		
ORTHOGRAPHIC FF	**PHONETIC FF**	**ORTHO-PHONETIC FF**
acceleration		access
analyze		action
annotation		active
antivirus		addition
application		analogue
archive		analytical
asynchronous		arithmetic
augmentation		assisted
augmented		avatar
automated		beneficial
balance		blog
barrier		botnet
binary		bulletin
biometric		calculate
cache		capacity
capability		chip
censorship		civil
client		cognitive
code		commercial
communication		common
creator		component
curve		complex
cycle		concept
data		confidentiality
decimal		conflict
decode		connect
dispute		connectivity
documentation		connector
ethical		consent
evaluation		consume
focus		consumer
fraud		contingency
function		control
functionality		convention
geospatial		convert
gigabyte		copyright

heuristic		cost
hypertext		criminal
identity		cryptography
immediate		dependent
information		digit
infrastructure		digital
innovation		disciplinary
infrastructure		disk
initiative		distribute
instruction		division
interface		document
laser		electronic
legal		embedded
legislation		encrypt
limitation		energy
machine		essential
menu		eventuality
microchip		evolutionary
microprocessor		expand
migration		export
multiplication		extensible
neural		factor
obligation		flash
obsolete		flexible
phase		flexibility
positioning		global
plug-in		graphical
prediction		hacker
privacy		hardware
private		illegal
procedure		impact
product		import
prototype		include
public		incorporate
qualitative		increment
quality		incremental
reduce		install
regulator		intellectual
regulatory		interaction
relay		interactive
reputation		interactivity
result		internet
security		intranet
social		invention
specialized		inventor
spider		iterative
spiral		joystick
stable		kilobit
telecommunications		limited
timescale		local
Trojan		magnetic
ubiquitous		malware
utilization		memory
vacuum		mental
virtualization		menu
virus		military
voltage		monitor
website		motor
		negligence

		non-commercial
		numerical
		object
		online
		optimal
		optimum
		perception
		perceptual
		persistent
		phishing
		physical
		prediction
		prevalent
		privacy (RP)
		process
		processor
		program
		programmer
		projector
		promotion
		protection
		protocol
		radical
		reflect
		refresh
		responsible
		reverse
		revolutionary
		revolutionize
		robot
		scan
		scanner
		sensor
		specific
		specification
		static
		subtraction
		system
		technical
		technological
		technology
		text
		tolerance
		tracking
		transaction
		transform
		transition
		transmit
		transistor
		usability
		valve
		variable
		version
		video
		virtual
		virtually
		visual
		web
94	0	153

6.2. MY CLASSIFICATION

6.2.1. SOURCE 1: ABSTRACT (Heat Reduction in Semiconductors by Phonon Annihilation)

MY CLASSIFICATION (1)		
Phoneme FF	Stress FF	Phoneme-Stress (P-S) FF
phase	electron	annihilation
	project	circumstance
	transport	coherent
		generation
		interference
		phonon
		reduction
		semiconductor
		simulation
		structure
1	3	10

6.2.2. SOURCE 2: STRUCTURE AND REACTIVITY II LECTURE

MY CLASSIFICATION (2)		
Phoneme FF	Stress FF	Phoneme-Stress (P-S) FF
acetic	acidity	abundance
archetypal	carboxylic	acetate
base	common	additional
constant	constant	adjacent
electrophoresis	difference	alanine
epsilon (GA)	distance	anion
equilibrium	electron	basicity
hydronium	energy	component
isoelectric	negative	concentration
methylamine (GA)	positive	dipole
ratio	resonance	electrode
scientific	system	electronegative
species		enolate
succinic		enzyme
zero		epsilon (RP)
		equation
		equivalent
		hexane
		hydrogen
		inductive
		ion
		lysine
		medicine
		methylamine (RP)
		molecule
		neutral
		nitrogen
		protein
		proton
		soluble
		stabilization
		stationary
		substance
		technique
15	12	34

6.2.3. **SOURCE 3:** LECTURE ON BIOLOGY OF CANCER

MY CLASSIFICATION (3)		
Phoneme FF	Stress FF	Phoneme-Stress (P-S) FF
aorta	aggressive	activator
benign	capillaries	artificial
cause	detriment	biopsy
chemotherapeutic	doctor	cascade
cure	factor	cerebral
diet	intestine	circulatory
gene	nutrient (GA)	concentration
heterogeneous	system	culture
immune	tumor (GA)	diagram
microscopic		enzyme
motile		existence
nodule		fibroblast
osteosarcoma		final
peptide		fraction
space		frequent
		glycoprotein
		immunosuppressant
		importance
		isotope
		lymphocyte
		metastasize
		matrix
		membrane
		microscope
		nutrient (RP)
		ovary
		penetration
		primary
		production
		process
		protease
		protein
		radioactivity
		region
		secretion
		sequential
		spinal
		transplantable
		tumour (RP)
		unusual
		vital
15	9	41

6.2.4. SOURCE 4: ENGLISH FOR ENVIRONMENTAL SCIENCE (GLOSSARY)

MY CLASSIFICATION (4)		
Phoneme FF	Stress FF	Phoneme-Stress (P-S) FF
abiotic	access	aerosol
acid	altitude (GA)	agricultural
air	animal	agriculture
analysis	capacity	altitude (RP)
analytical	carbon	analyze
anthropogenic	carnivore	aquifer
antibiotic	complex	artificial
area	conserve	atmosphere
atom	consume (GA)	benzene
bacteria	contaminant	biodiversity
biological	continental	biomass
biota	continentality	biosphere
biotic	convert	bromide
bromine	ecologist	carbohydrate
catalytic	ecology	carcinogen
chlorine	energy	categorize
climate	facility	chlorofluorocarbon
climatic	geology	coastal
coniferous	herbivore	collection
consumption	impact	commercial
convert	intensive	community
cycle	metamorphosis	complexity
deposit	mineral	component
detritus	model	composition
excrete	organism	congestion
fauna	oxygen	conservation
gene	preventive	conserve
genetic	project	consultancy
genetically	residential	consume (RP)
geothermal	sedimentary	consumer
gorilla	solvent	contaminate
graphical	subsistence	cultivate
interrelated	system	cultivation
intersect	technology	decompose
monoxide	transport	decomposer
multi-media	tropical	decomposition
mutate		degradation
photosynthesis		denitrify
photovoltaic		designation
phytoplankton		destruction
public		detritivore
reserve		displacement
scheme		dissipate
site		diversity
species		drainage
sphere		ecosphere
spray		ecosystem
state		educate
statistical		efficiency
stratum (GA)		elevation
thermal		emission
usage		erosion
		eutrophication
		evaporate
		evapotranspiration

		evolution
		excrete
		exposure
		extinction
		fertilize
		fertilizer
		filtration
		foliage
		fuel
		gaseous
		generate
		generation
		global
		hierarchy
		hormone
		human
		hydrocarbon
		hydrosphere
		hydrology
		image
		impermeable
		incinerate
		incinerator
		incorporate
		industrialized
		inefficient
		infrastructure
		interaction
		interconnected
		inventory
		ion
		irrigate
		irrigation
		lithosphere
		maritime
		mechanization
		menu
		mesosphere
		meteorology
		methane
		microorganism
		migration
		modify
		molecule
		monoculture
		natural
		nitrogen
		non-renewable
		nuclear
		nutrient
		operation
		ozone
		particle
		particulate
		permeable
		pesticide
		photosynthesize
		pollinator

		porous
		power
		preservation
		preserve
		primary
		primate
		private
		process
		produce
		producer
		productivity
		radiation
		recyclable
		recycle
		recycling
		reduce
		reduction
		region
		regulation
		renewable
		requirement
		residue
		resistance
		respiratory
		secondary
		separation
		social
		solar
		spatial
		specialization
		stratosphere
		subspecies
		substance
		sustainability
		sustainable
		synthesize
		tabular
		technique
		temperature
		tertiary
		texture
		thermosphere
		transformation
		transportation
		troposphere
		turbine
		ultraviolet
		vapour
		variable
		vegetation
		viability
		viable
		volume
		zoology
51	35	167

6.2.5. **SOURCE 5:** ENGLISH FOR ICT STUDIES (GLOSSARY)

MY CLASSIFICATION (5)		
Phoneme FF	Stress FF	Phoneme-Stress (P-S) FF
antivirus	access	acceleration
arithmetic (adj)	active	action
assisted	analogue	addition
biometric	avatar	analyze
cache	cognitive	annotation
code	complex	application
common	confidentiality	arithmetic (n)
control	conflict	archive
curve	connect	asynchronous
cycle	connectivity	augmentation
data	connector	augmented
decode	consent	automated
dispute	consume (GA)	balance
ethical	contingency	barrier
factor	convert	beneficial
focus	criminal	binary
fraud	cryptography	bulletin
graphical	dependent	calculate
heuristic	disciplinary	capability
interactive	embedded	capacity
interactivity	encrypt	censorship
intranet	energy	civil
laser	expand	client
numerical	export	commercial
phase	flexibility	communication
plug-in	impact	component
prevalent	import	concept
public	include	consume (RP)
result	incremental	consumer
reverse	internet	convention
specific	inventor	creator
static	iterative	decimal
ubiquitous	limited	distribute
video	memory	division
virus	mental	document
	military	documentation
	monitor	essential
	object	evaluation
	personal	eventuality
	persistent	evolutionary
	projector	extensible
	radical	flexible
	reflect	function
	sensor	functionality
	system	geospatial
	technology	global
	transistor	hypertext
	transform	identity
	transmit	illegal
	transistor	immediate
	visual	incorporate
		increment
		information
		infrastructure
		initiative

		innovation
		install
		instruction
		intellectual
		interaction
		interface
		invention
		kilobit
		legal
		legislation
		limitation
		local
		machine
		menu
		microchip
		microprocessor
		migration
		motor
		multiplication
		negligence
		neural
		non-commercial
		obligation
		obsolete
		penetration
		perception
		perceptual
		positioning
		prediction
		privacy
		private
		procedure
		process
		processor
		product
		program
		programmer
		promotion
		protection
		protocol
		prototype
		qualitative
		quality
		reduce
		refresh
		regulator
		regulatory
		relay
		reputation
		responsible
		revolutionary
		revolutionize
		robot
		security
		social
		specification
		specialized
		spiral

		stable
		subtraction
		telecommunication
		timescale
		tolerance
		transaction
		transition
		Trojan
		usability
		utilization
		vacuum
		variable
		version
		virtual
		virtualization
		virtually
		voltage
		website
35	51	130

6.3. COMPLETE LIST OF WORDS (English-Spanish-Phonetic Transcription in English: RP ‖ GA)

6.3.1. SOURCE 1: ABSTRACT (Heat Reduction in Semiconductors by Phonon Annihilation)

LIST OF WORDS (1)		
ENGLISH	SPANISH	TRANSCRIPTION
annihilation	aniquilación	/ə ˌnaɪ ə ˈleɪʃ n/
circumstance	circunstancia	/ˈsɜːk əm stæns/
coherent	coherente	/kəʊ ˈhɪər ənt ‖ koʊ ˈhɪr-/
electron	electrón	/i ˈlek trɒn/
generation	generación	/ˌdʒen ə ˈreɪʃ n/
interference	interferencia	/ˌɪnt ə ˈfɪər əns ‖ ˌɪnt r ˈfɪr-/
phase	fase	/feɪz/
phonon	fonón	/ˈfəʊn ɒn ‖ ˈfoʊn ɑːn/
project	proyecto	/ˈprɒdʒ ekt‖ ˈprɑːdʒ-/
semiconductor	semiconductor	/ˌsem i kən ˈdʌkt ə‖ -r/
simulation	simulación	/ˌsɪm ju ˈleɪʃ n/
reduction	reducción	/ri ˈdʌk ʃn/
structure	estructura	/ˈstrʌk ʧə /
transport	transporte	/ˈtræns pɔːt‖ pɔːrt/
		14 words

6.3.2. SOURCE 2: STRUCTURE AND REACTIVITY II LECTURE

LIST OF WORDS (2)		
ENGLISH	SPANISH	TRANSCRIPTION
abundance	abundancia	/ə ˈbʌnd əns/
acetate	acetato	/ˈæs ə teɪt/
acetic	acético	/ə ˈsiːt ɪk/
acidity	acidez	/ə ˈsɪd ət i/
additional	adicional	/ə ˈdɪʃ ən‿l/
adjacent	adyacente	/ə ˈdʒeɪs nt/
alanine	alanina	/ˈæl ə niːn /
anion	anión	/ˈæn ˌaɪ‿ən/
archetypal	arquetípico	/ˌɑːk i ˈtaɪp l/
base	base	/beɪs/
basicity	basicidad	/beɪˈsɪs ɪ ti/
carboxylic	carboxílico	/kɑː ˈbɒks ɪl ɪk‖ kɑːr ˈbɑːks l ɪk/
common	común	/ˈkɒm ən ‖ ˈkɑːm-/
component	componente	/kəm ˈpəʊn ənt‖ -ˈpoʊn-/
concentration	concentración	/ˌkɒns n ˈtreɪʃ n‖ ˌkɑːns-/
constant	constante	/ ˈkɒnst ənt ‖ ˈkɑːnst-/
difference	diferencia	/ˈdɪf rəns /
dipole	dipolo	/ˈdaɪ pəʊl ‖ -poʊl/
distance	distancia	/ˈdɪst əns/
electrode	electrodo	/i ˈlek trəʊd‖ -troʊd/
electron	electrón	/i ˈlek trɒn/
electronegative	electronegativo	/i ˌlek trəʊ ˈneg ət ɪv(‖ -troʊ-) /
electrophoresis	electroforesis	/ɪ ˌlek trəʊ fə ˈriːs ɪs ‖-troʊ fəˈri sɪs/
energy	energía	/en ədʒ i ‖ -rdʒ i/
enolate	enolato	/ˈin l ˌeɪt/
enzyme	enzima	/ˈen zaɪm/
epsilon	épsilon	/ep ˈsaɪl ən/‖ /ˈeps ə lɑːn/
equation	ecuación	/i ˈkweɪʒ n/
equilibrium	equilibrio	/ˌiːk wi ˈlɪb ri‿əm/
equivalent	equivalente	/ɪ ˈkwɪv əl ənt/
hexane	hexano	/ˈheks eɪn/

hydrogen	hidrógeno	/ ˈhaɪdr ədʒ ən/
hydronium	hidronio	/haɪ ˈdrɒ ni əm‖ -ˈdroʊ/
inductive	inductivo	/ɪn ˈdʌkt ɪv/
ion	ión	/ ˈaɪ‿ən/
isoelectric	isoeléctrico	/ˌaɪs əʊ i ˈlek trɪk ‖-oʊ/
lysine	lisina	/ˈlaɪ siːn/
medicine	medicina	/ˈmed sn/
methylamine	metilamina	/me ˈθaɪl ə miːn/‖/ˌmeθ əl ə ˈmiːn/
molecule	molécula	/ˈmɒl ɪ kjuːl‖ ˈmɑːl-/
negative	negativo	/ ˈneg ət ɪv /
neutral	neutral	/ˈnjuːtr əl‖ ˈnuːtr əl/
nitrogen	nitrógeno	/ ˈnaɪtr ədʒ ən/
positive	positivo	/ˈpɒz ət ɪv/
protein	proteína	/ˈprəʊt iːn‖ ˈproʊt-/
proton	protón	/ ˈprəʊt ɒn/ ‖ /ˈproʊt ɑːn/
ratio	ratio	/ˈreɪʃ i‿əʊ/ ‖ /oʊ/
resonance	resonancia	/ ˈrez n‿əns/
scientific	científico	/ˌsaɪ‿ən ˈtɪf ɪk/
soluble	soluble	/ˈsɒl jʊb l/
species	especies	/ ˈspiːʃ iːz/
stabilization	estabilización	/ˌsteɪb əl aɪ ˈzeɪʃ n/
stationary	estacionario	/ˈsteɪʃ ən ər‿i‖ -ə ner i/
substance	substancia	/ˈsʌb stəns/
succinic	succínico	/sʌk ˈsɪn ɪk /
system	sistema	/ˈsɪst əm/
technique	técnica	/tek ˈniːk/
zero	cero	/ˈzɪər əʊ ‖ ˈzɪr oʊ/
		58 words

6.3.3. **SOURCE 3:** LECTURE ON BIOLOGY OF CANCER

LIST OF WORDS (3)		
ENGLISH	SPANISH	TRANSCRIPTION
activator	activador	/ ˈækt ɪ veɪt ə ‖ -veɪţ r/
aggressive	agresivo	/ə ˈgres ɪv/
aorta	aorta	/ eɪ ˈɔːt ə/ ‖ /eɪ ˈɔːrţ ə /
artificial	artificial	/ ˌɑːt ɪ ˈfɪʃ l‖ ˌɑːrţ-/
benign	benigno	/ bə ˈnaɪn/
biopsy	biopsia	/ ˈbaɪ ɒps i /
capillaries	capilares	/ kə ˈpɪl ər iz/ ‖ /ˈkæp ə ler iz /
cascade	cascada	/ kæ ˈskeɪd /
cause	causa	/kɔːz/
cerebral	cerebral	/ ˈser əb rəl/
chemotherapeutic	quimioterapeuta	/ ˌkiːm əʊ ˌθer ə ˈpjuːt ɪk /
circulatory	circulatorio	/ˌsɜːk ju ˈleɪt‿ər i‖ ˈsɝːk jəl ə tɔːr i/
concentration	concentración	/ˌkɒns n ˈtreɪʃ n‖ ˌkɑːns-/
culture	cultura	/ ˈkʌltʃ ə ‖ -r/
cure	cura	/kjʊə‖ kjʊr/
detriment	detrimento	/ˈdetr ɪ mənt/
diagram	diagrama	/ ˈdaɪ‿ə græm/
diet	dieta	/ˈdaɪ‿ət/
doctor	doctor	/ˈdɒkt ə ‖ ˈdɑːkt r/
enzyme	enzima	/ ˈen zaɪm /
existence	existencia	/ɪg ˈzɪst əns/
factor	factor	/ ˈfækt ə ‖ -r/
fibroblast	fibroblasto	/ ˈfaɪb rəʊ blɑːst ‖-roʊ blæst/
final	final	/ˈfaɪn l/
fraction	fracción	/ˈfræk ʃn/
frequent	frecuente	/ˈfriːk wənt/

gene	gen	/dʒiːn/
glycoprotein	glicoproteína	/ ˌglaɪk əʊ ˈprəʊt iːn ‖oʊ ˈproʊt- /
heterogeneous	heterogéneo	/ ˌhet◡ər əʊ ˈdʒiːn i◡əs ‖ ˌheţ ə roʊ-/
immune	inmune	/ɪ ˈmjuːn/
immunosuppressant	inmunosupresor	/ ˌɪm ju nəʊ sə ˈpres ənt ‖ˌɪm jə noʊ-/
importance	importancia	/ɪm ˈpɔːt ns ‖ -ˈpɔːrt-/
intestine	intestino	/ɪn ˈtest ɪn/
isotope	isótopo	/ˈaɪs ə təʊp‖ -ə toʊp/
lymphocyte	linfocito	/ ˈlɪmf əʊ saɪt /
metastasis	metástasis	/me ˈtæst əs ɪs/
metastasize	metastatizar	/me ˈtæst ə saɪz/
matrix	matriz	/ ˈmeɪtr ɪks/
membrane	membrana	/ ˈmem breɪn/
microscope	microscopio	/ ˈmaɪk rə skəʊp/ ‖ /-skoʊp/
microscopic	microscópico	/ ˌmaɪk rə ˈskɒp ɪk /
motile	móvil	/ ˈməʊt aɪəl/ ‖ /ˈmoʊţ əl /
nodule	nódulo	/ˈnɒd juːl ‖ ˈnɑːdʒ uːl/
nutrient	nutriente	/ˈnjuːtr i◡ənt ‖ ˈnuːtr-/
osteosarcoma	osteosarcoma	/ˈɒst i◡əʊ sɑː ˈkəʊm ə‖ˈɑːst i◡ə sɑːr ˈkoʊm ə/
ovary	ovario	/ ˈəʊv ər i/ ‖ /ˈoʊv-/
penetration	penetración	/ˌpen ə ˈtreɪʃ n/
peptide	péptido	/ ˈpept aɪd/
primary	primario	/ˈpraɪm ər i ‖ -er-/
production	producción	/prə ˈdʌk ʃn/
process	proceso	/ˈprəʊs es‖ ˈprɑːs es/
protease	proteasa	/ ˈprəʊt i eɪz / ‖ /ˈproʊţ-/
protein	proteína	/ˈprəʊt iːn‖ ˈproʊt-/
radioactivity	radioactividad	/ ˌreɪd i◡əʊ æk ˈtɪv ət i‖ -oʊ æk ˈtɪv əţ i/
region	región	/ˈriːdʒ ən/
secretion	secreción	/sɪ ˈkriːʃ n/
sequential	secuencial	/sɪ ˈkwenʃ l/
space	espacio	/speɪs/
spinal	espinal	/ˈspaɪn l/
system	sistema	/ˈsɪst əm/
transplantable	trasplantable	/ ˌtrænts ˈplɑːnt əb l /
tumor	tumor	/ ˈtjuːm ə ‖ ˈtuːm r/
unusual	inusual	/ ʌn ˈjuːʒ u◡əl /
vital	vital	/ ˈvaɪt l /
		64 words

6.3.4. **SOURCE 4:** ENGLISH FOR ENVIRONMENTAL SCIENCE (GLOSSARY)

LIST OF WORDS (4)		
ENGLISH	SPANISH	TRANSCRIPTION (ENGLISH)
abiotic	abiótico	/ˌeɪ baɪ ˈɒt ɪk ‖ -ˈɑːţ ɪk/
access	acceso	/ˈæk ses/
acid	ácido	/ˈæs ɪd/
aerosol	aerosol	/ˈeər əʊ sɒl ‖ ˈer ə sɑːl/
agricultural	agrícola	/ˌæg rɪ ˈkʌltʃ r◡əl/
agriculture	agricultura	/ˈæg rɪ ˌkʌltʃ ə‖ -r/
agronomist	agrónomo	/ə ˈgrɒn əm ɪst‖ ə ˈgrɑːn-/
air	aire	/eə ‖ er/
altitude	altitud	/ˈælt ɪ tjuːd‖ -tuːd/
aluminium	aluminio	/ˌæl ə ˈmɪn i◡əm/
analysis	análisis	/ə ˈnæl əs ɪs/
analytical	analítico	/ ˌæn ə ˈlɪt ɪk l/
analyze	analizar	/ˈæn ə laɪz/
animal	animal	/ˈæn ɪm l/
anthropogenic	antropogénico	/ˌæn θrəp əʊˈdʒɛn ɪk/

antibiotic	antibiótico	/ˌænt i baɪ ˈɒt ɪk ‖ ˌæntˌ i baɪ ˈɑːţ ɪk/
aquifer	acuífero	/ˈæk wɪf ə‖ -r/
area	área	/ˈeər i‿ə ‖ ˈer-/
artificial	artificial	/ˌɑːt ɪ ˈfɪʃ l/
atmosphere	atmósfera	/ˈæt məs fɪə ‖ -fɪr/
atmospheric	atmosférico	/ˌæt məs ˈfer ɪk/
atom	átomo	/ˈæt əm/
bacteria	bacteria	/bæk ˈtɪər i‿ə ‖ -ˈtɪr-/
benzene	benceno	/ˈbenz iːn/
biodiversity	biodiversidad	/ˌbaɪ əʊ daɪ ˈvɜːs ət i ‖ ˌbaɪ oʊ də ˈvɝːs əţ i/
biological	biológico	ˌbaɪ‿ə ˈlɒdʒ ɪk l ‖ -ˈlɑːdʒ-/
biomass	biomasa	/ˈbaɪ əʊ mæs ‖ -oʊ-/
biosphere	biosfera	/ˈbaɪ əʊ sfɪə ‖ -ə sfɪr/
biota	biota	/baɪ ˈəʊt ə ‖ -ˈoʊţ-/
biotic	biótico	/baɪ ˈɒt ɪk ‖ -ˈɑːţ-/
bromide	bromuro	/ˈbrəʊm aɪd ‖ ˈbroʊm-/
bromine	bromo	/ˈbrəʊm iːn ‖ ˈbroʊm-/
capacity	capacidad	/kə ˈpæs ət i /
carbohydrate	carbohidrato	/ˌkɑːb əʊ ˈhaɪdr eɪt ‖ ˌkɑːrb oʊ-/
carbon	carbono	/ˈkɑːb ən ‖ ˈkɑːrb-/
carcinogen	carcinógeno	/kɑː ˈsɪn ədʒ ən‖ kɑːr/
carnivore	carnívoro	/ˈkɑːn ɪ vɔː‖ ˈkɑːrn ə vɔːr/
catalytic	catalítico	/ˌkæt ə ˈlɪt ɪk ‖ ˌkæţ l ˈɪţ ɪk /
categorize	categorizar	/ˈkæt ɪg ə raɪz/
chlorine	cloro	/ˈklɔːr iːn/
chlorofluorocarbons	clorofluorocarbono	/ˌklɔːr əʊˌflʊər əʊ ˈkɑːbən‖ ˌklɔːr ə ˌflʊr oʊ ˈkɑːrb ən/
climate	clima	/ˈklaɪm ət/
climatic	climático	/klaɪ ˈmæt ɪk/
collection	collección	/kə ˈlek ʃn/
commercial	comercial	/kə ˈmɜːʃ l/
community	comunidad	/kə ˈmjuːn ət i /
complex	complejo	/ˈkɒm pleks‖ ˌkɑːm ˈpleks/
complexity	complejidad	/kəm ˈpleks ət i /
component	componente	/kəm ˈpəʊn ənt‖ -ˈpoʊn-/
composition	composición	/ˌkɒmp ə ˈzɪʃ n/
congestion	congestión	/kən ˈdʒes tʃən/
coniferous	conífera	/kəʊ ˈnɪf ər‿əs‖ koʊ-/
conservation	conservación	/ˌkɒns ə ˈveɪʃ n ‖ ˌkɑːns r-/
conserve	conservar	/kən ˈsɜːv/
consume	consumir	/kən ˈsjuːm‖ -ˈsuːm/
consumer	consumidor	/kən ˈsjuːm ə‖ -ˈsuːm r/
consumption	consumo	/kən ˈsʌmp ʃn/
contaminant	contaminante	/kən ˈtæm ɪn ənt/
contaminate	contaminar	/kən ˈtæm ɪ neɪt /
continental	continental	/ˌkɒnt ɪ ˈnent l ‖ ˌkɑːnt n ˈenţ l/
continentality	continentalidad	/ˌkɒn tn ənˈtæl ɪ ti/
convert	convertir	/kən ˈvɜːt /
cultivate	cultivar	/ˈkʌlt ɪ veɪt/
cultivation	cultivo	/ˌkʌlt ɪ ˈveɪʃ n/
cycle	ciclo	/ˈsaɪk l/
decompose	descomponer	/ˌdiː kəm ˈpəʊz‖ -ˈpoʊz/
decomposer	descomponedor	/ˌdiː kəm ˈpəʊz ə‖ -ˈpoʊz r/
decomposition	descomposición	/ˌdiː ˌkɒmp ə ˈzɪʃ n ‖ -ˌkɑːmp-/
degradation	degradación	/ˌdeg rə ˈdeɪʃ n/
denitrify	desnitrifcar	/ˌdiː ˈnaɪtr ɪ faɪ /
deposit	depósito	/di ˈpɒz ɪt ‖ -ˈpɑːz ət/
designation	designación	/ˌdez ɪg ˈneɪʃ n/
destruction	destrucción	/di ˈstrʌk ʃ n/
detritivore	detritívoro	/dɪˈtraɪ təˌvɔr ‖-ˌvoʊr/

detritus	detrito	/di ˈtraɪt əs/
displacement	desplazamiento	/dɪs ˈpleɪs mənt/
dissipate	disiparse	/ˈdɪs ɪ peɪt/
diversity	diversidad	/daɪ ˈvɜːs ət i ‖ də ˈvɝːs əţ i/
drainage	drenaje	/ˈdreɪn ɪdʒ/
ecologist	ecologista	/ ɪ ˈkɒl ədʒ ɪst ‖ -ˈkɑːl-/
ecology	ecología	i ˈkɒl ədʒ i ‖ -ˈkɑːl-/
ecosphere	ecosfera	/ˈiːk əʊ sfɪə‖ -oʊ sfɪr/
ecosystem	ecosistema	/ˈiːk əʊ ˌsɪst əm ‖ -oʊ-/
educate	educar	/ˈed ju keɪt‖ ˈedʒ ə-/
efficiency	eficiencia	/ə ˈfɪʃ ns i/
elevation	elevación	/ˌel ɪ ˈveɪʃ n/
emission	emisión	/i ˈmɪʃ n/
energy	energía	/ˈen ədʒ i ‖ -rdʒ i/
erosion	erosión	/ɪ ˈrəʊʒ n ‖ ɪ ˈroʊʒ n/
eutrophication	eutroficación	/ ju ˌtrɒf ɪ ˈkeɪʃ n‖ -ˌtroʊf-/
evaporate	evaporar	/ i ˈvæp ə reɪt/
evapotranspiration	evapotranspiración	/ɪˌvæp oʊˌtræn spəˈreɪ ʃn/
evolution	evolución	/ˌiːv ə ˈluːʃ n ‖ ˌev-/
excrete	excretar	/ɪk ˈskriːt /
exposure	exposición	/ɪk ˈspəʊʒ ə‖ -ˈspoʊʒ r/
extinction	extinción	/ɪk ˈstɪŋk ʃn/
facility	facilidad	/fə ˈsɪl ət i /
fauna	fauna	/ˈfɔːn ə/
fertilize	fertilizar	/ ˈfɜːt ə laɪz /
fertilizer	fertilizante	/ˈfɜːt ə laɪz ə‖ ˈfɝːţ l aɪz r/
filtration	filtración	/fɪl ˈtreɪʃ n/
finite	finito	/ˈfaɪn aɪt/
flora	flora	/ˈflɔːr ə/
foliage	follaje	/ˈfəʊl i◡ɪdʒ ‖ ˈfoʊl-/
fossil	fósil	/ˈfɒs l ‖ ˈfɑːs l/
fuel	fuel	/ˈfjuː◡əl/
gas	gas	/gæs/
gaseous	gaseoso	/ˈgæs i◡əs/
gene	gen	/dʒiːn/
generate	generar	/ˈdʒen ə reɪt/
generation	generación	/ ˌdʒen ə ˈreɪʃ n/
genetic	genético	/dʒə ˈnet ɪk/
genetically	genéticamente	/dʒə ˈnet ɪk l◡i/
geology	geología	/dʒi ˈɒl ədʒ i ‖ -ˈɑːl-/
geothermal	geotérmico	/ˌdʒiː əʊ ˈθɜːm l ‖ -oʊ ˈθɝːm-/
global	global	/ˈgləʊb l ‖ ˈgloʊb l/
gorilla	gorila	/gə ˈrɪl ə/
graphical	gráfico	/ˈgræf ɪk l/
graphics	gráficos	/ˈgræf ɪks/
habitat	hábitat	/ˈhæb ɪ tæt/
herbivore	herbívoro	/ ˈhɜːb ɪ vɔː‖ ˈɝːb ə vɔːr/
hierarchy	jerarquía	/ˈhaɪr ɑːk i ‖ -ɑːrk i/
hormone	hormona	/ˈhɔːm əʊn ‖ ˈhɔːrm oʊn/
human	humano	/ˈhjuːm ən/
hydrocarbon	hidrocarbono	/ˌhaɪdr əʊ ˈkɑːb ən ‖ -ə ˈkɑːrb-/
hydrology	hidrología	/haɪ ˈdrɒl ədʒ i ‖ -ˈdrɑːl-/
hydrosphere	hidrosfera	/ˌhaɪdr əʊ sfɪə ‖ ˌhaɪdr ə sfɪr/
image	imagen	/ˈɪm ɪdʒ/
impact	impacto	/ˈɪm pækt/
impermeable	impermeable	/ɪm ˈpɜːm i◡əb l /
incinerate	incinerar	/ɪn ˈsɪn ə reɪt/
incinerator	incinerador	/ɪn ˈsɪn ə reɪt ə ‖ -reɪţ r/

incorporate	incorporar	/ɪn ˈkɔːp ə reɪtǁ -ˈkɔːrp-/
industrialized	industrializado	/ɪn ˈdʌs tri‿ə laɪz d/
inefficient	ineficiente	/ˌɪn ə ˈfɪʃ nt/
infrastructure	infraestructure	/ˈɪnf rə ˌstrʌk ʧə ǁ -ʧr/
intensive	intensivo	/ɪn ˈtens ɪv/
interaction	interacción	/ˌɪnt ər ˈæk ʃn/
interconnected	interconectado	/ˌɪnt ə kə ˈnekt ɪd ǁ ˌɪnţ r-/
inventory	inventario	/ˈɪn vənt‿ər iǁ ˈɪn vən tɔːr i/
ion	ión	/ˈaɪ‿ən/
irrigate	irrigar	/ˈɪr ɪ geɪt/
irrigation	irrigación	/ˌɪr ɪ ˈgeɪʃ n/
lithosphere	litosfera	/ˈlɪθ əʊ sfɪə ǁ -ə sfɪr/
map	mapa	/mæp/
maritime	marítimo	/ˈmær ɪ taɪm/
mechanization	mecanización	/ˌmek ən aɪ ˈzeɪʃ nǁ -ən ə-/
menu	menú	/ˈmen juː /
mesosphere	mesosfera	/ˈmes əʊ sfɪə ǁ -ə sfɪr/
metamorphosis	metamorfosis	/ˌmet ə ˈmɔːf əs ɪs ǁ ˌmeţ ə ˈmɔːrf-/
meteorology	meteorología	/ˌmiːt i‿ə ˈrɒl ədʒ i ǁ ˌmiːţ i‿ə ˈrɑːl-/
methane	metano	/ˈmiːθ eɪn ǁ ˈmeθ-/
microorganism	microorganismo	/ˌmaɪk rəʊ ˈɔːg ə ˌnɪz əm/ǁ -roʊ ˈɔːrg-/
migration	migración	/maɪ ˈgreɪʃ n/
mineral	mineral	/ˈmɪn r‿əl/
model	modelo	/ˈmɒd l ǁ ˈmɑːd l/
modify	modificar	/ˈmɒd ɪ faɪǁ ˈmɑːd-/
molecule	molécula	/ˈmɒl ɪ kjuːlǁ ˈmɑːl-/
monoculture	monocultivo	/ˈmɒn əʊ ˌkʌltʃ əǁ ˈmɑːn ə ˌkʌltʃ r/
monoxide	monóxido	/mə ˈnɒks aɪdǁ mə ˈnɑːks-/
multi-media	multimedia	/ˌmʌlt i ˈmiːd i‿ə/
mutate	mutar	/mju ˈteɪt ǁ ˈmjuː t eɪt/
natural	natural	/ˈnæʧ r‿əl/
nitrogen	nitrógeno	/ˈnaɪtr ədʒ ən/
non-renewable	no renovable	/ˌnɒn ri ˈnjuː əb lǁ ˌnɑːn ri ˈnuː-/
nuclear	nuclear	/ˈnjuːk li‿əǁ ˈnuːk li‿r /
nutrient	nutriente	/ˈnjuːtr i‿əntǁ ˈnuːtr-/
operation	operación	/ˌɒp ə ˈreɪʃ n ǁ ˌɑːp-/
organic	orgánico	/ɔː ˈgæn ɪk ǁ ɔːr-/
organism	organismo	/ˈɔːg ə ˌnɪz əm ǁ ˈɔːrg-/
oxygen	oxígeno	/ˈɒks ɪdʒ ənǁ ˈɑːks-/
ozone	ozono	/ˈəʊz əʊn ǁ ˈoʊz oʊn/
particle	partícula	/ ˈpɑːt ɪk l /
particulate	particulado	/pɑː ˈtɪk jʊl ətǁ pɑːr ˈtɪk jəl ət/
permeable	permeable	/ ˈpɜːm i‿əb l/
pest	peste	/pest/
pesticide	pesticida	/ˈpest ɪ saɪd/
photosynthesis	fotosíntesis	/ˌfəʊt əʊ ˈsɪnθ əs ɪsǁ ˌfoʊţ oʊ-/
photosynthesize	fotosintetizar	/ ˌfəʊt əʊ ˈsɪnθ ə saɪzǁ ˌfoʊţ oʊ-/
photovoltaic	fotovoltaico	/ ˌfəʊt əʊ vɒl ˈteɪ ɪk ǁ ˌfoʊţ oʊ vɑːl-/
phytoplankton	fitoplancton	/ ˌfaɪt əʊ ˈplæŋkt ənǁ ˌfaɪţ oʊ -/
plant	planta	/plɑːntǁ plænt/
porous	poroso	/ˈpɔːr əs/
power	poder	/ˈpaʊ‿əǁ ˈpaʊ‿r/
preservation	preservación	/ˌprez ə ˈveɪʃ n ǁ -r-/
preserve	preservar	/pri ˈzɜːv/
preventive	preventivo	/pri ˈvent ɪv/
primary	primario	/ˈpraɪm ər i ǁ -er i/
primate	primate	/ˈpraɪm ət/
private	privado	/ˈpraɪv ət/

process	proceso	/ˈprəʊs es‖ ˈprɑːs es/
produce	producir	/prə ˈdjuːs‖ -ˈduːs/
producer	productor	/prə ˈdjuːs ə‖ -ˈduːs r/
productivity	productividad	/ˌprɒd ʌk ˈtɪv ət i ‖ ˌproʊ dʌk ˈtɪv əţ i/
project	proyecto	/ˈprɒdʒ ekt‖ ˈprɑːdʒ-/
public	público	/ˈpʌb lɪk/
radiation	radiación	/ˌreɪd i ˈeɪʃ n/
recyclable	reciclable	/ˌriː ˈsaɪk l‿əb l/
recycle	reciclar	/ˌriː ˈsaɪk l/
recycling	reciclaje	/ˌriː ˈsaɪk l‿ɪŋ/
reduce	reducir	/ri ˈdjuːs‖ -ˈduːs /
reduction	reducción	/ri ˈdʌk ʃn/
region	región	/ˈriːdʒ ən/
regulation	regulación	/ˌreg ju ˈleɪʃ n‖ -jə-/
renewable	renovable	/ri ˈnjuː‿əb l‖ -ˈnuː əb l/
reserve	reserva	/ri ˈzɜːv/
residential	residencial	/ˌrez ɪ ˈdenʃ l /
residue	residuo	/ˈrez ɪ djuː‖ -duː /
resistance	resistencia	/ri ˈzɪst əns/
respiratory	respiratorio	/ri ˈspɪr ət‿ər i‖ ˈresp ər‿ə tɔːr i /
salmonella	salmonela	/ˌsælm ə ˈnel ə /
scheme	esquema	/skiːm/
secondary	secundario	/ˈsek ənd‿ər i‖ -ən der i/
sedimentary	sedimentario	/ˌsed ɪ ˈment‿ər i/
separation	separación	/ˌsep ə ˈreɪʃ n/
site	sitio	/saɪt/
smog	esmog	/smɒg ‖ smɑːg/
social	social	/ˈsəʊʃ l ‖ ˈsoʊʃ l/
solar	solar	/ˈsəʊl ə‖ ˈsoʊl r/
solvent	solvente	/ˈsɒlv ənt‖ ˈsɑːlv-/
spatial	espacial	/ˈspeɪʃ l/
specialization	especialización	/ˌspeʃ əl‿aɪ ˈzeɪʃ n‖ -əl‿ə-/
species	especies	/ˈspiːʃ iːz/
sphere	esfera	/sfɪə ‖sfɪr/
spray	espray	/spreɪ/
standard	estándar	/ˈstænd əd ‖ -rd/
state	estado	/steɪt/
statistical	estadístico	/stə ˈtɪst ɪk l/
stock	stock	/stɒk ‖ stɑːk/
stratosphere	estratosfera	/ˈstræt ə sfɪə ‖ ˈstræţ ə sfɪr/
stratum	estrato	/ˈstrɑːt əm‖ ˈstreɪţ əm/
subsistence	subsistencia	/səb ˈsɪst əns/
subspecies	subespecies	/ˈsʌb ˌspiːʃ iːz/
substance	substancia	/ˈsʌb stəns/
sustainability	sostenibilidad	/sə ˌsteɪn ə ˈbɪl ət i /
sustainable	sostenible	/sə ˈsteɪn əb l/
synthesize	sintetizar	/ˈsɪnθ ə saɪz/
system	sistema	/ˈsɪst əm/
tabular	tabular	/ˈtæb jʊl ə ‖ -jəl r/
technique	técnica	/tek ˈniːk/
technology	tecnología	/tek ˈnɒl ədʒ |i ‖ -ˈnɑːl-/
temperature	temperatura	/ˈtemp r‿ətʃ ə ‖ ‿ətʃ r/
tertiary	terciario	/ˈtɜːʃ ər i‖ ˈtɝːʃ i er i/
texture	textura	/ˈteks ʧə ‖ -ʧr/
thermal	térmico	/ˈθɜːm l/
thermosphere	termosfera	/ˌθɜːm əʊ ˈsfɪə ‖ ˌθɝːm ə sfɪr/
toxic	tóxico	/ˈtɒks ɪk ‖ ˈtɑːks ɪk/
transformation	transformación	/ ˌtræns fə ˈmeɪʃ n/‖ -fr-/

transport	transporte	/ˈtræns pɔːt ‖ ˈtrænts pɔːrt/
transportation	transportación	/ˌtræns pɔː ˈteɪʃ n‖ ˌtræns pr-/
tropical	tropical	/ˈtrɒp ɪk l ‖ ˈtrɑːp-/
troposphere	troposfera	/ˈtrɒp ə sfɪə ‖ ˈtroʊp ə sfɪr/
turbine	turbina	/ˈtɜːb aɪn/
ultraviolet	ultravioleta	/ˌʌltr ə ˈvaɪ‿əl ət /
vapour	vapor	/ˈveɪp ə ‖ -r/
variable	variable	/ˈveər i‿əb l ‖ ˈver-/
vegetation	vegetación	/ˌvedʒ ə ˈteɪʃ n/
viability	viabilidad	/ˌvaɪ‿ə ˈbɪl ət i /
viable	viable	/ˈvaɪ‿əb l/
volume	volumen	/ˈvɒl juːm‖ ˈvɑːl jəm/
zoology	zoología	/zəʊ ˈɒl ədʒ i‖ zoʊ ˈɑːl-/
		261 words

6.3.5. SOURCE 5: ENGLISH FOR ICT STUDIES (GLOSSARY)

LIST OF WORDS (5)		
ENGLISH	SPANISH	TRANSCRIPTION
acceleration	aceleración	/ək ˌsel ə ˈreɪʃ n/
access	acceso	/ˈæk ses/
action	acción	/ˈæk ʃn/
active	activo	/ˈækt ɪv/
addition	adición	/ə ˈdɪʃ n/
analogue	analógico	/ˈæn ə lɒg‖ ˈæn l ɔːg/
analytical	analítico	/ˌæn ə ˈlɪt ɪk l/
analyze	analizar	/ˈæn ə laɪz/
annotation	anotación	/ˌæn əʊ ˈteɪʃ n ‖ -ə-/
antivirus	antivirus	/ˌænt i ˈvaɪər əs/
application	aplicación	/ˌæp lɪ ˈkeɪʃ n/
arithmetic (adj)	aritmético	/ˌær ɪθ ˈmet ɪk/
arithmetic (n)	aritmético	/ə ˈrɪθ mə tɪk/
archive	archivo	/ˈɑːk aɪv ‖ ˈɑːrk-/
assisted	asistido	/ə ˈsɪst ɪd/
asynchronous	asincrónico	/eɪ ˈsɪŋk rən əs/
augmentation	aumento	/ˌɔːg men ˈteɪʃ n/
augmented	aumentado	/ɔːg ˈment ɪd/
automated	automatizado	/ˈɔːt ə meɪt ɪd/
avatar	avatar	/ ˈæv ə tɑː‖ -tɑːr/
balance	balance	/ˈbæl əns/
barrier	barrera	/ˈbær i‿ə ‖- r/
beneficial	beneficioso	/ˌben ɪ ˈfɪʃ l /
binary	binario	/ˈbaɪn ər i/
biometric	biométrico	/ˌbaɪ əʊ ˈmetr ɪk ‖ -oʊ-/
blog	blog	/blɒg ‖ blɔːg/
botnet	botnet	/bɒt net‖ bɑːt/
bulletin	boletín	/ ˈbʊl ət ɪn /
cache	caché	/kæʃ/
calculate	calcular	/ˈkælk ju leɪt‖ -jə-/
capability	capacidad	/ˌkeɪp ə ˈbɪl ət i /
capacity	capacidad	/kə ˈpæs ət i /
censorship	censura	/ ˈsens ə ʃɪp ‖ -r-
chip	chip	/tʃɪp/
civil	civil	/ˈsɪv l/
client	cliente	/ˈklaɪ‿ənt/
code	código	/kəʊd ‖ koʊd/
cognitive	cognitivo	/ˈkɒg nət ɪv‖ ˈkɑːg nəţ ɪv/
commercial	comercial	/kə ˈmɜːʃ l/
common	común	/ ˈkɒm ən ‖ ˈkɑːm-/

communication	comunicación	/kə ˌmjuːn ɪ ˈkeɪʃ n/
component	componente	/kəm ˈpəʊn ənt‖ -ˈpoʊn-/
complex (adj)	complejo	/ˈkɒm pleks‖ ˌkɑːm ˈpleks/
concept	concepto	/ˈkɒn sept ‖ ˈkɑːn-/
confidentiality	confidencialidad	/ˌkɒn fɪ ˌdenʃ i ˈæl ət i ‖ ˌkɑːn fə ˌdentʃ i ˈæl əţ i/
conflict	conflicto	/ˈkɒn flɪkt ‖ ˈkɑːn-/
connect	conectar	/kə ˈnekt/
connectivity	conectividad	/ˌkɒn ek ˈtɪv ət i‖ ˌkɑːn ek ˈtɪv əţ i/
connector	conector	/kə ˈnekt ə ‖ -r/
consent (v)	consentir	/kən ˈsent/
consume	consumir	/kən ˈsjuːm‖ -ˈsuːm/
consumer	consumidor	/kən ˈsjuːm ə‖ -ˈsuːm r/
contingency	contingencia	/kən ˈtɪndʒ əns i /
control	control	/kən ˈtrəʊl‖ -ˈtroʊl/
convention	convención	/kən ˈvenʃ n/
convert	convertir	/kən ˈvɜːt /
copyright	copyright	/ˈkɒp i |raɪt ‖ ˈkɑːp-/
cost	coste	/kɒst‖ kɔːst /
creator	creador	/kri ˈeɪt ə‖ -ˈeɪţ r/
criminal	criminal	/ˈkrɪm ɪn l/
curve	curva	/kɜːv/
cryptography	criptografía	/krɪp ˈtɒg rəf i ‖ -ˈtɑːg-/
cycle	ciclo	/ˈsaɪk l/
data	data	/ˈdeɪt ə/
decimal	decimal	/ˈdes əm l/
decode	decodificar	/diː ˈkəʊd ‖ -ˈkoʊd/
dependent	dependiente	/di ˈpend ənt/
digit	digito	/ˈdɪdʒ ɪt/
digital	digital	/ˈdɪdʒ ɪt l /
disciplinary	disciplinario	/ˈdɪs ə plɪn ər‿i ‖ -plə ner i/
disk	disco	/dɪsk/
dispute	disputa	/dɪ ˈspjuːt/
distribute	distribuir	/dɪ ˈstrɪb juːt‖ -jət/
division	división	/dɪ ˈvɪʒ n/
document	documento	/ˈdɒk ju mənt‖ ˈdɑːk jə-/
documentation	documentación	/ˌdɒk ju men ˈteɪʃ n‖ ˌdɑːk jə-/
electronic	electrónico	/ˌel ek ˈtrɒn ɪk ‖ i ˌlek ˈtrɑːn ɪk /
embedded	embebido (sistema)	/ɪm ˈbed ɪd /
encrypt	encriptar	/ɪn ˈkrɪpt/
energy	energía	/ ˈen ədʒ i ‖ -rdʒ i/
essential	esencial	/ɪ ˈsenʃ l/
ethical	ético	/ˈeθ ɪk l/
evaluation	evaluación	/i ˌvæl ju ˈeɪʃ n/
eventuality	eventualidad	/ɪ ˌventʃ u ˈæl ət i /
evolutionary	evolultivo	/ˌiːv ə ˈluːʃ n‿ər i‖ ˌev ə ˈluːʃ ə ner i/
expand	expandir	/ɪk ˈspænd/
export	exportar	/ɪk ˈspɔːt ‖ ɪk ˈspɔːrt/
extensible	extensible	/ɪk ˈstents əb l /
factor	factor	/ˈfækt ə ‖ -r/
flash	flash	/flæʃ/
flexible	flexible	/ˈfleks əb l/
flexibility	flexibilidad	/ˌfleks ə ˈbɪl ət i/
focus	foco	/ˈfəʊk əs ‖ ˈfoʊk əs/
fraud	fraude	/frɔːd/
function	función	/ˈfʌŋk ʃn/
funtionality	funcionalidad	/ˌfʌŋk ʃə ˈnæl ət i /
geospatial	geoespacial	/ˌdʒiː əʊ ˈspeɪʃ l ‖ -oʊ /
gigabyte	gigabyte	/ˈgɪg ə baɪt/
global	global	/ˈgləʊb l ‖ ˈgloʊb l/

graphical	gráfico	/ˈgræf ɪk l/
hacker	hacker	/ˈhæk ə ‖ -r/
hardware	hardware	/ ˈhɑːd weə ‖ ˈhɑːrd wer /
heuristic	herístico	/hjuˈrɪst ɪk/
hypertext	hipertexto	/ˈhaɪp ə tekst ‖ -r-/
identity	identitdad	/aɪ ˈdent ət i /
illegal	ilegal	/ɪ ˈliːg l/
immediate	inmediato	/ɪ ˈmiːd i‿ət/
impact	impacto	/ˈɪm pækt/
import	importar	/ ɪm ˈpɔːt ‖ -ˈpɔːrt/
include	incluir	/ɪn ˈkluːd/
incorporate	incorporar	/ɪn ˈkɔːp ə reɪt‖ -ˈkɔːrp-/
increment	incrementar	/ ˈɪŋ krɪ ment/
incremental	incremental	/ˌɪŋ krɪ ˈment l/
information	informatción	/ˌɪnf ə ˈmeɪʃ n ‖ -r-/
infrastructure	infraestructura	/ˈɪnf rə ˌstrʌk tʃə ‖ -ʧr-/
initiative	inciativa	/ɪ ˈnɪʃ ət ɪv/
innovation	innovación	/ ˌɪn əʊ ˈveɪʃ n ‖ -ə-/
install	instalar	/ɪn ˈstɔːl/
instruction	instrucción	/ɪn ˈstrʌk ʃn/
intellectual	intelectual	/ˌɪnt ə ˈlek tʃu‿əl /
interaction	interacción	/ˌɪnt ər ˈæk ʃn /
interactive	interactivo	/ˌɪnt ər ˈækt ɪv/
interactivity	interactividad	/ˌɪnt ər æk ˈtɪv ət i/
interface	interfaz	/ˈɪnt ə feɪs /
internet	internet	/ˈɪnt ə net ‖ ˈɪnʧ r-/
intranet	intranet	/ˈɪntr ə net/
invention	invención	/ɪn ˈvenʃ n/
inventor	inventor	/ ɪn ˈvent ə ‖ ˈvenʧ r/
iterative	iterativo	/ˈɪt‿ər ət ɪv‖ ˈɪʧ ə reɪʧ ɪv /
joystick	joystick	/ˈdʒɔɪ stɪk/
kilobit	kilobit	/ˈkɪl əʊ ˌbɪt ‖ ˈkil ə- /
laser	laser	/ˈleɪz ə ‖ -r/
legal	legal	/ˈliːg l/
legislation	legislación	/ˌledʒ ɪ ˈsleɪʃ n/
limited	limitado	/ˈlɪm ɪt ɪd ‖ əʧ əd/
limitation	limitación	/ˌlɪm ɪ ˈteɪʃ n/
local	local	/ˈləʊk l ‖ ˈloʊk l/
machine	máquina	/mə ˈʃiːn/
magnetic	magnético	/mæg ˈnet ɪk/
malware	malware	/ˈmæl weə ‖ -wer/
memory	memoria	/ˈmem ər‿i/
mental	mental	/ˈment l /
menu	menú	/ˈmen juː/
microchip	microchip	/ˈmaɪk rəʊ tʃɪp ‖ -roʊ-/
micropocessor	microporcesador	/ˈmaɪk rəʊ ˌprəʊs es ə‖ -rə ˌprɑːs es r/
migration	migración	/maɪ ˈgreɪʃ n/
military	militar	/ˈmɪl ɪ‿tər i‖ -ə ter i/
monitor	monitor	/ˈmɒn ɪt ə‖ ˈmɑːn əʧ r/
motor	motor	/ˈməʊt ə ‖ ˈmoʊʧ r/
multiplication	multiplicación	/ˌmʌlt ɪ plɪ ˈkeɪʃ n/
negligence	neglicencia	/ˈneg lɪdʒ əns/
neural	neural	/ˈnjʊər əl ‖ ˈnʊr əl/
non-commercial	no comercial	/ ˌnɒn kə ˈmɜːʃ l‖ ˌnɑːn-/
numerical	numérico	/nju ˈmer ɪk l‖ nu-/
object	objeto	/ˈɒb dʒekt‖ ˈɑːb-/
obligation	obligación	/ˌɒb lɪ ˈgeɪʃ n‖ ˌɑːb-/
obsolete	obsoleto	/ˈɒb sə liːt ‖ ˌɑːb sə ˈliːt/

online	on line/en línea	/ˌɒn ˈlaɪn ‖ ˌɑːn-/
optimal	óptimo	/ˈɒpt ɪm əl‖ ˈɑːpt-/
optimum	óptimo	/ˈɒpt ɪm əm ‖ ˈɑːpt-/
personal	personal	/ˈpɜːs n◡əl/
penetration	penetración	/ˌpen ə ˈtreɪʃ n/
perception	percepción	/pə ˈsep ʃn ‖ pər-/
perceptual	perceptual	/pə ˈsep tʃu◡əl ‖ pər-/
persistent	persistente	/pə ˈsɪst ənt ‖ pər-
phase	fase	/feɪz/
phishing	phishing	/ˈfɪʃ ɪŋ/
physical	físico	/ˈfɪz ɪk l/
positioning	posicionamiento	/pə ˈzɪʃ n ◡ɪŋ/
plug-in	plug-in	/ˈplʌg ɪn/
prediction	predicción	/pri ˈdɪk ʃn/
prevalent	prevalente	/ˈprev əl ənt/
privacy	privacidad	/ˈprɪv əs i‖ ˈpraɪv-/
private	privado	/ˈpraɪv ət/
procedure	procedimiento	/prəʊ ˈsiːdʒ ə‖ prə ˈsiːdʒ r/
process	proceso	/ˈprəʊs es‖ ˈprɑːs es/
processor	procesador	/ˈprəʊs es ə‖ ˈprɑːs es r/
product	producto	/ˈprɒd ʌkt‖ ˈprɑːd-/
program	programa	/ˈprəʊ ɡræm‖ ˈproʊ-/
programmer	programador	/ˈprəʊ ɡræm ə‖ ˈproʊ ɡræm r/
projector	proyector	/prə ˈdʒekt ə‖ -r/
promotion	promoción	/prə ˈməʊʃ n ‖ -ˈmoʊʃ n/
protection	protección	/prə ˈtek ʃn/
protocol	protocolo	/ˈprəʊt əʊ kɒl ‖ ˈproʊţ ə kɑːl/
prototype	prototipo	/ ˈprəʊt əʊ taɪp ‖ ˈproʊţ ə-/
public	público	/ˈpʌb lɪk/
qualitative	cualitativo	/ˈkwɒl ɪt ət ɪv‖ ˈkwɑːl ə teɪţ ɪv/
quality	calidad	/ˈkwɒl ət i ‖ ˈkwɑːl əţ i/
radical	radical	/ˈræd ɪk l/
reduce	reducir	/ri ˈdjuːs‖ -ˈduːs/
reflect	reflejar	/ri ˈflekt/
refresh	refrescar	/ri ˈfreʃ/
regulator	regulador	/ˈreg ju leɪt ə ‖ -jə leɪţ r/
regulatory	regulador	/ˌreg ju ˈleɪt ər i ‖ ˈreg jəl ə tɔːr i/
relay	relé	/ˈriː leɪ/
reputation	reputación	/ˌrep ju ˈteɪʃ n‖ -jə-/
responsible	responsable	/ri ˈspɒns əb l‖ ri ˈspɑːnts-/
result	resultado	/ri ˈzʌlt/
reverse	reverso	/ri ˈvɜːs/
revolutionary	revolucionario	/ˌrev ə ˈluːʃ ən r◡i ‖ -ə ner i/
revolutionize	revolucionar	/ˌrev ə ˈluːʃ ə naɪz/
robot	robot	/ˈrəʊb ɒt‖ ˈroʊb ɑːt /
scan	escanear	/skæn/
scanner	escáner	/ˈskæn ə ‖ -r/
security	seguridad	/sɪ ˈkjʊər ət i ‖ -ˈkjʊr əţ i/
sensor	sensor	/ˈsens ə ‖ -r/
social	social	/ˈsəʊʃ l ‖ ˈsoʊʃ l/
specific	específico	/spə ˈsɪf ɪk/
specialized	especializado	/ˈspeʃ ə laɪzd/
spider	spider	/ˈspaɪd ə ‖-r/
spiral	espiral	/ˈspaɪr əl/
stable	estable	/ˈsteɪb l/
static	estático	/ˈstæt ɪk /
subtraction	sustracción	/səb ˈtræk ʃn/
system	sistema	/ˈsɪst əm/
technical	técnico	/ˈtek nɪk l/

technological	tecnológico	/ˌtek nə ˈlɒdʒ ɪk l ‖ -ˈlɑːdʒ-/
technology	tecnología	/tek ˈnɒl ədʒ |i ‖ -ˈnɑːl-/
telecommunications	telecomunicaciones	/ˌtel i kə ˌmjuːn ɪ ˈkeɪʃ ənz‖ ˌtel ə-/
text	texto	/tekst/
timescale	escala de timepo	/ˈtaɪm skeɪl/
tolerance	tolerancia	/ˈtɒl ər ənts‖ ˈtɑːl-/
tracking	tracking	/ˈtræk ɪŋ/
transaction	transacción	/træn ˈzæk ʃn/
transform	transformar	/træns ˈfɔːm‖ -ˈfɔːrm/
transition	trransición	/træn ˈzɪʃ n/
transmit	transmitir	/trænz ˈmɪt /
transistor	transistor	/træn ˈzɪst ə‖ -r/
trojan	troyano	/ˈtrəʊdʒ ən ‖ ˈtroʊdʒ ən/
ubiquitous	ubícuo	/ju ˈbɪk wɪt əs‖ -wəţ əs/
usability	usabilidad	/ˈjuːz əb ət i/
utilization	utilización	/ˌjuːt ɪl aɪ ˈzeɪʃ n/
vacuum	vacío	/ˈvæk juˍəm/
valve	válvula	/vælv/
variable	variable	/ˈveər iˍəb l ‖ ˈver- /
version	versión	/ˈvɜːʃ n /
video	vídeo	/ˈvɪd i əʊ ‖ -oʊ/
virtual	virtual	/ˈvɜːtʃ uˍəl/
virtualization	virtualización	/ˈvɜːtʃ u əl aɪ ˈzeɪʃ n /
virtually	virtualmente	/ ˈvɜːtʃ uˍəl i
virus	virus	/ˈvaɪr əs/
visual	visual	/ˈvɪʒ uˍəl/
voltage	voltaje	/ˈvəʊlt ɪdʒ‖ ˈvoʊlt-/
web	web	/web/
website	sito web	/ˈweb saɪt/
		245 words

6.4. COMPLETE SOURCES

6.4.1. SOURCE 1: ABSTRACT (Heat Reduction in Semiconductors by Phonon Annihilation)

The objective of this project is to develop multiscale simulation tools for coupled carrier-phonon transport in micro/optoelectronic devices and to explore novel concepts of phonon engineering for heat removal and termal management in advancedsemiconductor devices. The phase I effort resulted in a prototype software modules for multiscale simulation of phonon dynamics in LD semiconductors and proven feasibility of using phonon annihilation for heat reduction in electronic devices. It has beenobserved that if phonon waves of different phase interact, they could, under the right set of circumstances, cause either constructive or destructive interference. CFDRC and UCR team plans to use existing commercial Multiphysics CFD-ACE+ softwareenvironment as a platform to implement comprehensive multiscale (quantum to continum) modeling tools for phonon engineering and couple it to existing carrier, optical and thermal models. We will utilize the tools for detailed examination of new methods ofheat reduction by phonon engineering such as generation of coherent confined acoustic phonons by drifting electrons in LD structures, investigation of the effects of the acoustic impedance mismatch, phonon filters by using Q dots/wire gratings, periodic phonon bandgap structures, "phase shifted" QWs, QD etc. We will also conduct experiments at UCR and collaborate with physicists from academia and with DoD expers (ARL, ONR) to identify and screen new ideas. CFDRC will use established partnerships with US defense electronic industry (Honeywell, Harris, Raythoon, Hughes) for practical verification and software commercialization.

6.4.2. SOURCE 2: STRUCTURE AND REACTIVITY II LECTURE

S1: good morning. today we're going to be talking about polyprotic acids, which are acids that have more than one easily ionizable hydrogen, and among the most important of these are the amino acids. which exist in proteins and peptides, and so we're going move to those as soon as we, cover some of the basics, with, some simpler, polyprotic acids. i have on the board acetic acid, as the, archetypal carboxylic acid, and then i have two... diprotic acids, succinic acid *<PAUSE WHILE WRITING ON BOARD>* and adipic acid. *<PAUSE WHILE WRITING ON BOARD>* these are the common names for these, dicarboxylic acids. their scientific names, are, one-two-three-four butane, dioic acid, and one-two-three-four-five-six, hexane, dioic acid. however, they're usually called succinic acid, and adipic acid so we'll, stick with their common names. now notice that we have two P-K-A values once we have, two carboxylic acid groups, and i've given the P-K-A values here, and we want to compare them then to the P-K-A value of the single carboxylic group we have in acetic acid. and you can see, that, of course these two protons are equivalent and so the first ionization can be either one of those, and so, either one for the first ionization, has the P-K-A value of four-point-two-one. but once i have removed one of the protons, then the other proton becomes significantly less acidic. and so we want to talk about, first why the first proton, is more acidic than the one in acetic acid, and then secondly, why does the, it becomes so much harder to remove the second proton once we've removed the first one. and i think you know the answers to both of those questions, why is succinic acid more acidic, in its first ionization than acetic acid?

<PAUSE:04>

S2: resonance?

S1: hm? [S2: (isn't it resonance?)] i just didn't hear.

S2: is it resonance?

S1: well the resonance if i draw the, structure of the anion in each case *<PAUSE WHILE WRITING ON BOARD>* so this is the anion for acetic acid the acetate ion... and lemme draw the corresponding, anion, for succinic acid. *< PAUSE WHILE WRITING ON BOARD>* and it wouldn't have mattered which of these protons i removed, i have similar resonance in both of 'em. i can draw a resonance contributor in which i delocalize the charge to the other oxygen in either case. so resonance is not the, reason for the difference. it's a, good reason for why it's as acidic as it is, but not for the difference. *<PAUSE:04>* any other ideas? yep?

S3: you have (inductive and that's supposed to) take away the negative charge a little bit (xx)

S1: and what is the thing that's the indu- doing the inductive effect?

S3: the oxygen atoms.

S1: the other carboxylic acid group here. okay? so i have the partially positive carbon these oxygens more electronegative than carbon and so i have, an electron withdrawing effect, through the bonds, of one carboxylic acid group stabilizing the carboxylate anion in the other case. so this, base, is weaker, than the base, in, acetic- acetate ion. <*PAUSE WHILE WRITING ON BOARD*> and where the base is weaker, the corresponding conjugate acid is stronger. well why is it so hard to remove the second, proton? why does the P-K-A go up, with the second one? <*PAUSE:08*> yep?

S4: because there's two negative charges after that?

S1: because i already have a negative charge in the molecule so let's just write a little, equation for, the removal of the second proton <*PAUSE WHILE WRITING ON BOARD*> and i'm going to put the, hydronium ion... that has resulted sort of up there to remind us, that we're trying to pull this positively charged species away from an ion that now is doubly, negatively charged. and so we have to exert more energy it is, harder to do, to remove that proton from something that is already negatively charged. so the first proton is easier to remove, than it was for acetate ion, but the second proton is harder to remove. now notice what happens with adipic acid. the first proton, is, more like the proton in acetic acid, in other words the P-K-A has gone up relative, to the P-K-A of succinic acid, for the first ionization, but the P-K-A for the second ionization has gone down. what's going on here?

<*PAUSE:13*>

S5: the dipole is, weaker when more carbon atoms (xx)

S1: i have a l- greater distance between the two carboxylic acid groups. so my inductive effect, if i ionize this one <> first, the inductive effect of the second carboxylic acid group is weaker, than it is in the case of succinic acid. because inductive effect falls off with distance, so with the greater distance between the two carboxylic acid groups, i have less effect, on sta- the stabilization of the base resulting from the loss of the first proton. what is also true then about the loss of the second proton?

S6: (negative charges) (xx)

S1: again with negative charges are further apart from each other, and therefore it's not as hard to remove a proton from this end of the molecule, when this end is negative, as it was with succinic acid where the charges were closer together. okay? any questions about what we've done so far? <*PAUSE:05*> okay i'd like to move on then to talk about amino acids, as po- polyprotic acids <*PAUSE WHILE WRITING ON BOARD*> and the amino acids that we're gonna talk about are the alpha amino acids the amino acids that are found in peptides and proteins. <*PAUSE WHILE WRITING ON BOARD*> and they're interesting to us of course because they are the components, of, the very important, substances such as enzymes muscle tissue cellular tissues, which are made up of, proteins... there's alanine, representative of a simple... amino acid... and i'm drawing it this way first, to emphasize the fact that it is an alpha amino acid here, it is the carboxylic acid group, and the carbon adjacent to it, (will) you remember we talked about, is the alpha carbon, and i've got an amino group, on that alpha carbon so this is an alpha amino acid. and it has, two sites of aci- possible acidity-basicity, um, this, is the, carboxylic acid group and the P-K-A there if we compare it to acetic acid, we would expect it to have a P-K-A of approximately four-point-eight. <*PAUSE WHILE WRITING ON BOARD*> on the other hand, we have the basic site here which can be protonated to give us an acid <*PAUSE WHILE WRITING ON BOARD*> and that is sort of like ammonia, ammoniamine, methylammoniamine, and that has a P-K-A, we would expect of about nine-point-four. so if we compare this these two structures, we will see that the structure to the right, is the one that we should be, considering... because, if we consider this as an equilibrium, between two acids, this is the stronger acid this is the weaker acid and the proton resides on, the weaker acid. and so we have an equilibrium, where the proton is mostly on the nitrogen, and only a little bit on the oxygen atom of the amino acid. so the, true structure of the amino acid, is in this form, where it is an internal salt, the proton has been transferred from the oxygen to the nitrogen, and we call that form the zwitterion. the internal, salt where we have the negative charge and the, positive charge within the same, species not two separate ions that can, migrate apart from each other. <*PAUSE:04*> well let's write, a series of equilibria for alanine <*PAUSE WHILE WRITING ON BOARD*> and i'm going to start with the form <*PAUSE WHILE WRITING ON BOARD*> that will exist at low P-H. where i have both of the basic sites in the molecule protonated <*PAUSE WHILE WRITING ON BOARD*> so at low P-H, i will be protonated at the nitrogen and protonated at the oxygen. but as i add base to the system... i will deprotonate <*PAUSE WHILE WRITING ON BOARD*> and if we look up the actual P-K-A values of the, two acidic protons here, it turns out that this one is about two-point-

three-four, and this one, is nine-point-six-nine. (okay,) so when i add base to the system, which proton is going to leave first the one on oxygen or the one on nitrogen?

SS: <>

S1: the one on oxygen is gonna leave first. so the first deprotonation of this polyprotic acid and this is why i'm calling it a polyprotic acid i've got two protons here that i can lose <*PAUSE WHILE WRITING ON BOARD*> well the first ionization, gives me that, species, and if i were to add acid to that, it would go back to being, this species here. okay? so at low P-H i have this at intermediate P-H <*PAUSE WHILE WRITING ON BOARD*> i have that form, and i can go on and add more base <*PAUSE WHILE WRITING ON BOARD*> and that will deprotonate, the next, acidic proton <*PAUSE WHILE WRITING ON BOARD*> so i get to the form that i have at high P-H. <*PAUSE WHILE WRITING ON BOARD*> let's take a moment to consider, these two P-K-A values. why is, the carboxylic acid group in an amino acid, more acidic, than the one in acetic acid? <*PAUSE:08*> what's going on there?

S3: positive charge (xx) increase (xx) stabilize the (xx)

S1: so i have the positive charge on this nitrogen, exerting an inductive effect, stabilizing, the negative charge, on the carboxylate anion. okay? so that this is, considerably more acidic, than what we expected by just comparing it to acetic acid. the amino group, it depends on whether you compare it to ammonia, ammonium ion which has a P-K-A of about nine-point-four, or methylammonium ion which is about ten, it lies somewhere in that range. and you can argue, two ways, you can say well it's a little more basic than you expect these electrons are a little more available because they're close to a negative charge, or you can argue well... you have the carboneal group there still, therefore it makes it a little more, unavailable, it lies right at the cusp of where you expect it to be and so it's a little hard to make an argument about that as well. well we're interested, in two numbers. one of them is, that for a technique that is very often used in analyzing for amino acids or peptides proteins and in separating them. and that's called electrophoresis, where you put a substance on a gel, and you put two electrodes on it, and then you watch to see where, the compound migrates, in this electric field. and there is, a point at which no migration takes place. and that P-H is called the isoelectric point. the point in whi- at which the charges within the molecule are balanced, so that it migrates neither to the positive electrode nor to the, negative electrode. <*WRITING ON BOARD DURING NEXT 1:00 OF UTTERANCE*> so one, value we're going to be interested in, is, P-H for what we call the isoelectric point. <*PAUSE:15*> the point at which <*PAUSE:08*> the amino acid <*PAUSE:07*>does not migrate <*PAUSE:05*> to either electrode <*PAUSE:06*> in an electric field. <*PAUSE:09*> for example, at low P-H the amino acid has a net positive charge, and therefore, it will migrate to the negative electrode... and at high P-H it has a net, negative charge, and therefore it will migrate to the positive electrode. and i can manipulate the P-H to do to make it do either one of those things. but at this intermediate value whatever that is, i have a situation, where the charges are balanced within the same molecule, and therefore, the compound is stationary, in an electric field. this also happens to be the point at which the amino acid is least soluble. so you can sometimes get it to precipitate from solution, at this isoelectric point. so that's another reason that we're interested, in knowing what that isoelectric point is so that we can isolate, (a) compound. the other thing that we are really interested in, especially when you get into biochemistry, medicine, is what is the form of the amino acid, at physiological P-H? what is the form in which it exists, the cells and the tissues, enzymes things like that? so that is the second P-H value that we're gonna be interested in. <*PAUSE WHILE WRITING ON BOARD*> and for the, sake of argument it varies from tissue to tissue, but let's just take, a P-H of six-point-five as physiological P-H. okay...? well last time... we derived, the equation for the acidity constant, of a weak acid, the K-A, and from that then <*WRITING ON BOARD DURING NEXT :31 OF UTTERANCE*> by certain manipulations we arrived at the, Henderson-Hasselbach equation, which said that the P-H was equal to the P-K-A <*PAUSE:05*> plus the log, of the ratio, of the concentration of the, conjugate base of the acid... to the concentration of the conjugate acid itself. <*PAUSE:04*> so this is a relationship that relates the P-K-A of the particular acid we're talking about to the P-H of the solution. <*WRITING ON BOARD DURING NEXT :32 OF UTTERANCE*> and we went further and said that when, at the special point at which we had an equal concentration of the conjugate base and the conjugate acid, so when the concentration of the conjugate base equaled that of the conjugate acid, then the log... this term the log of the concentration of the conjugate base, over that of the conjugate acid, was equal, to zero... so that allows us to cancel out this term... and we have this one special point, at which, the P-H of the solution, is equal to the P-K-A of the acid that we're talking about. okay? so now let's see what, it looks like, if we try to plot <*PAUSE:06*> what kinds of species we have, at different P-Hs, for ana- for alanine, (xx) for the amino acid, (alanine.) <*PAUSE WHILE WRITING ON BOARD*> so i'm going to take, P-H as this axis <*PAUSE WHILE WRITING ON BOARD*> and on this axis i'm going to plot the relative abundance, of the different species there whether the, concentration of the, conjugate, base or that of the acid. so at some point i have hun- a hundred percent, of whichever species i'm talking about, down here i have zero percent, and in between, that point i have equal amounts of the two species... so i start with alanine at low P-H. <*PAUSE:06*>and almost all, of the, species in solution are this form, of alanine. the_ form in which it has it's doubly protonated. okay, but as i add base, the concentration of the, acid falls, H-A falls and for

every molecule of H-A that is deprotonated i get one molecule of A-minus formed, so i have a loss of H-A, and a gain of A-minus. okay? and our equation tells us that at a P-H value equal to the P-K-A value, which is two-point-three-four for the first proton that i'm taking off, so somewhere around here... the concentration of A-minus will equal the concentration, of H-A.*<PAUSE WHILE WRITING ON BOARD>* and then, the concentration of H-A will continue to decline. *<PAUSE WHILE WRITING ON BOARD>* and growing in then starting from almost zero, i will begin to see the conjugate base, forming. and again at this point, half of the species in solution will be that of the conjugate base. *<PAUSE WHILE WRITING ON BOARD>* then i continue to add more base, i'm now at this point, that's what we said at intermediate P-H, here, and i continue to add base let me remind you that i can go this way too with acid, i continue to add base, and so at the P-K-A value corresponding to this proton which is the one i'm now going to remove, at nine-point-six-nine, i again have a crossing point. *<PAUSE WHILE WRITING ON BOARD>* so all in this region, it is that species and then it starts falling off, comes down to half of it's value, over here, and tails off *<PAUSE WHILE WRITING ON BOARD>* and the third species builds in. and i'm deliberately drawing these longish tails, to remind you, that this doesn't happen all at once. that all three of these things are really in equilibrium with each other we're just talking about what predominates at any one, P-H. and so somewhere around here we begin to see, some of the third species coming in *<PAUSE WHILE WRITING ON BOARD>* and at high P-H then, it becomes the predominant species. so here *<PAUSE WHILE WRITING ON BOARD>* we have that species *<PAUSE WHILE WRITING ON BOARD>* this region we have predominantly that species... down here *<PAUSE WHILE WRITING ON BOARD>* we have this species... now with this we can answer two questions, one is, what is the isoelectric point? and that is the, point at which we have a... maximum for our... balanced ionic species the zwitterion. *<PAUSE WHILE WRITING ON BOARD>* which is actually if you wanna be more accurate the arithmetic mean of the two P-K-A values that, border it. okay? so if we took the arithmetic mean of two-point-three-four and nine-point-six-nine, you would get, the, isoelectric point for alanine. and the other question was, what form does the amino acid exist in, at physiological P-H? and physiological P-H we defined as six-point-five. *<PAUSE WHILE WRITING ON BOARD>* okay? so again the zwitter- zwitterionic form exists at physiological P-H... do you have any questions about what i've done so far? yeah?

S7: when you say physiological P-H of the zwitterions, (ones) what about the (alpha) classes of blue (lines) doesn't, the uh basic form also exist?

S1: yes, but the, you know the blue is just beginning to grow in at those P-Hs, there's very little of it. so what is the predominant form there? that's what we're talking about. and if i did it accurately if i calculated at every point what the concentrations were, i may have exaggerated how early the blue really begins to appear. but the point i'm really trying to make is that it doesn't suddenly become blue. that there is some blue, way back... any other questions? *<PAUSE:05>* okay well is this true of all amino acids? and the answer is no... alanine is what we call a neutral. *<PAUSE WHILE WRITING ON BOARD>* but other amino acids have additional amino groups, called other basic groups, or other acidic groups on the side chain. and therefore, they tend to be, not just diprotic as alanine is but triprotic and so on. so let's take a look at lysine *<WRITING ON BOARD DURING NEXT :12 OF UTTERANCE><PAUSE>* as an important basic amino acid. *<PAUSE>* it's very often found in the active site of enzymes, and so we're interested in knowing, what form it exists in, at thou- those positions. *<WRITING ON BOARD DURING NEXT :32 OF UTTERANCE><PAUSE:04>* so lysine has two amino groups. one of them is at the alpha position *<PAUSE:07>* the other one is at the *<PAUSE:04>* epsilon position. *<PAUSE:09>* so it is known as an epsilon amino, acid as well as an alpha amino acid. and here are my three acidic protons, so i have three different P-K-A values for them, this one is *<WRITING ON BOARD DURING NEXT :08 OF UTTERANCE>* two-point-one-eight... this one is eight-point-nine-five *<PAUSE:04>* and this one is ten-point-five-three. okay? so this one is almost like methylamine, this one is more acidic than en-methylamine and this one is definitely more acidic than acetic acid. so i've drawn the form that exists at low P-H... and we can do the same thing we did, with alanine, add base and explore what happens, at each stage. so as i add base to the system i'm gonna lose one of these protons, which one's gonna go first?

SS: *<UNINTELLIGIBLE ANSWERS>*

S1: the one on the carboxylic acid, the one with the lowest P-K-A *<PAUSE WHILE WRITING ON BOARD>* so i have an acid-base equilibrium here, as i add base i go to this form if i were to add acid i would go back that way this is at some intermediate P-H. *<PAUSE WHILE WRITING ON BOARD>* but notice this is not the zwitterion. this is not what exists at the isoelectric point, because i have two positive charges and one negative charge. so this is not balanced in its charge yet. *<PAUSE:09>* but i can add some more base to it, (set up) another equilibrium, what's the next proton that's going to be removed? which nitrogen? the alpha nitrogen or the, epsilon nitrogen? [SU-f: alpha] the alpha nitrogen is going to be the one to go next. *<PAUSE WHILE WRITING ON BOARD>* notice that this is the zwitterion. this is the form in which i have a balance of charges there. *<PAUSE WHILE WRITING ON BOARD>* and also presumably then the form that exists at the isoelectric point. *<PAUSE WHILE WRITING ON BOARD>* well i still have another proton i can remove relatively easily *<PAUSE WHILE WRITING ON BOARD>* i'll set up

another equilibrium <*PAUSE WHILE WRITING ON BOARD*> and this is the form that exists at high P-H. <*PAUSE WHILE WRITING ON BOARD*> where i have removed all of the easily ionizable protons. this is a triprotic acid, and i have three ionizations, for it. well let's do the same thing we did for alanine, draw a curve for this <*PAUSE:23*> so here are my axes again. <*PAUSE WHILE WRITING ON BOARD*> of relative abundancies on on- abundances on one axis, P-H on the other. <*PAUSE WHILE WRITING ON BOARD*> okay? and once again i start with the most acidic form, the one at low P-H where everything is protonated, and i have about a hundred percent of the specie up there, but then very soon, at a P-H of two-point-one-eight <*PAUSE WHILE WRITING ON BOARD*> as i add base, i have lost half of that species... it continues to die off as i add more and more base <*PAUSE WHILE WRITING ON BOARD*> and a new species, comes in. <*PAUSE WHILE WRITING ON BOARD*> so at that P-H, half of the s- species in solution is this, monodeprotonated form... which increases as i make the system more basic <*PAUSE WHILE WRITING ON BOARD*> but then, at a P-H corresponding to the P-K-A of the next proton that's going to be removed, in other words at eight-point-nine-five, so right around here <*PAUSE WHILE WRITING ON BOARD*> this species, starts falling off <*PAUSE WHILE WRITING ON BOARD*> and, a third species, comes in, crosses at that point... increases in concentration... then at the P-K-A corresponding to the ionization of this, third proton in other words at ten-point-five-three, you see this happens very fast. <*PAUSE WHILE WRITING ON BOARD*> this falls off, and my final most basic species <*PAUSE WHILE WRITING ON BOARD*> comes out. now the structures are too big to write here so what i'm going to do is i'm going to label them, this is structure A <*PAUSE:04*> this is structure B, this is structure, C... and this is structure D. okay? so we have A, predominating in this region... we have B, predominating in this region, here we are at C, and we finally get, to D. so let's ask our two questions. <*PAUSE:05*> what is the isoelectric point, for this species? well, the species that exists, at the isoelectric point is C, so our isoelectric point is, here. <*PAUSE WHILE WRITING ON BOARD*> so it has a high i- isoelectric point. and what is the form that exists, at physiological P-H? now remember we defined that as six-point-five. <*PAUSE WHILE WRITING ON BOARD*> so which form is it? B, is the species we have at physiological P-H. so at physiological P-H... notice that the, amino group, at the epsilon position, of the amino acid lysine is protonated. in other words lysine is positively charged at physiological P-H. and this is significant. for example in the structure of a protein, to have the positive charge on one lysine molecule interacting in an ionic fashion with the negative charge of a carboxylic acid group somewhere else in the molecule. so you have ionic, intermolecular type of interactions, nonbonding but still, helping to shape the structure of the protein. okay? so the overall shape of the protein, is a multiple, of these various interactions through the molecules you have a question?

S8: you said that the isoelectric point was also the arithmetic mean, is that only for diprotic?

S1: no it's the arithmetic mean <*INDICATING TWO POINTS ON BOARD*>

S8: oh just between those two.

S1: between those two. what is the H-A and which_ what is the A-minus, for that particular range okay? any other questions about what i've done. <*PAUSE:06*> okay next time we'll start talking about carbon acids, and enolate ions and then move on to their reactions.

{END OF TRANSCRIPT}

6.4.3. **SOURCE 3:** LECTURE ON BIOLOGY OF CANCER

S1: let me remind you, one more time about the discussion sections tomorrow those of you who happen to be in the one o'clock, discussion session we're moving from fourteen hundred, to thirteen hundred Chemistry Building, uh if by any chance you go to fourteen hundred Chem tomorrow i think you'll end up in a P chem class, which i assume most of you would, prefer not to be at, some of you might wanna be there but, uh if you end up in a chemistry class remember we're in the lecture room on the opposite side, of the building. also remember for tomorrow's discussion that i gave you kind of a thought exercise, at the end of last Wednesday's discussion after we had critiqued, um that article that i read you from the Ann Arbor News found all the things that were wrong with that study, i asked you to think, in this intervening week about the kinds of experiments you might do. uh, and do a better job of trying to find out whether there's a relationship between birth control pill usage, and cancer risk, and i think i said there is no single experiment it's not like there's one right answer it's a very complicated question how scientists go about, establishing cause and effect, uh so i would hope to hear a variety of approaches which you might come up with, in terms of trying to experimentally address that question so we'll discuss those tomorrow, and see both the strengths and weaknesses of the various experimental approaches. today as you know from looking at the lecture schedule we're gonna to be talking about metastasis, which uh, the process refers to the spread of, tumor cells from their primary site of origin where they originated, to another organ via

body fluids and most of the time we're talking about the blood stream the circulatory system. but you should keep in, mind that there are other body fluids there's the lymphatic system there's the cerebral spinal fluid, there's also the peritoneal fluid in the abdominal cavity. anywhere there's a fluid in the body that cancer cells can get into, those cancer cells could float through that fluid and end up somewhere else in the body. so basically it's uh, getting the cancer cell into a fluid somewhere and transporting it to a distant site. and usually we're talking about, the blood stream. uh by now you have a pretty good idea of the importance of metastasis, um, as a phenomenon first of all it's part of the inherent definition of cancer. remember the only defining characteristic of cancer, was the ability to spread by the process of engaging in metastasis. that's the only thing that distinguishes it unequivocally from a benign tumor. so metastasis is part of the defining, features, of a cancer. also last time you learned that uh, metastasis is one of the main ways that, cancer uses to kill people. uh the main cause of death is usually, not the primary tumor, but metastases spreading throughout the body and often getting into the one one of the vital organs, such as the, brain or liver or kidney, and this is often what cancer patients will die of. so this means that if we could do anything to interfere with the process of metastasis, in essence we could cure people of cancer, or we could at least cure people of the most debilitating and threatening life threatening, aspect of cancer which is the ability, of these cancer cells to metastasize. so today we're gonna focus on metastasis. and in essence we'll be focusing on the question of what is it about cancer cells, that in fact, allows them, to metastasize, while the benign tumor cells, can't do this nor do other, normal kinds of cells do this. in addressing this question, first of all you have to realize that metastasis is not a single event. and we talk about metastasis it's not really, a single, process that we're talking about it's actually, a sequential series of events, all of which must take place, in order for this phenomenon, of metastasis to occur. we commonly we therefore divide, metastasis into a series of stages and i'm gonna use three major steps, to divide this process, today. *<:13 PAUSE WHILE WRITING ON OVERHEAD>* the first, step, in the process of metastasis, involves the ability of cancer cells, to invade surrounding tissues, and vessels *<:16 PAUSE WHILE WRITING>* perhaps i should remind you that the definition of cancer remember was, the tumor that had the capability of invading and metastasizing, really, the first step in the process of metastasis is invasion. *<PAUSE:04>* so the first part of the definition of a tumor cell, is in fa- of a cancer cell is in fact this, process that is the first step, in metastasis. why do cancer cells do this invade surrounding tissue why do they tend to wander off and infiltrate, penetrate surrounding tissue while normal cells don't do that. actually there's several factors that appear to be involved, first of all, cancer cells don't stick together. uh, if a group of you in this in this room wanted to think of yourselves as a cluster of cancer cells, if you were all holding hands and joined to each other clearly it'd be very difficult to metastasize you couldn't very, easily invade, uh you'd all hafta, you know move as a as an attack unit, and if all the cells in a tumor, were attached to each other, it would be virtually impossible. so the first thing that cancer cells exhibit to get over this, problem is decreased adhesiveness... they don't stick together. they don't hold hands. unlike normal cells. um, if you were to take, a clump of, tumor tissue take a biopsy a specimen of tumor tissue and, put those cells into a test tube and, put 'em in, some kind of a, a nutrient solution, and shake that test tube very very vigorously, you'd find that the cancer cells would readily come apart. and you'd soon have a solution a suspension of individual largely individual cancer cells. now you might not think that's unusual, until you hear what happens with normal cells if you took a normal, chunk of tissue, a biopsy from a normal region of tissue, uh and put them in a test tube with a nutrient solution and try to shake them up, they would not come apart. there are very very strong structures, that hold adjacent cells together, in most tissues of the body. especially in epithelia, which remember are the main tissues in which, cancers arise. so cancer cells don't, stick to each other they're not structurally joined to each other, the way normal cells are. and since they're not stuck to each other, individual cells can easily wander off... the second thing that cancer cells, exhibit that allows them to invade is increased motility *<:09 PAUSE WHILE WRITING>* and again, picture yourself as part of a clump of cancer cells, if you were all, holding hands if you let go, but you just stood there, again, you're not gonna invade, you're not gonna go anywhere. you gotta be able to walk you gotta be able to move. most cells in the body don't move. most cells are not motile there are some exceptions, some cells as part of their normal functions need to move, but in an adult organism very few cells actually do this. and even cells in the_ blood cells you might think of them moving but it's really the fluid that's moving more than the cells themselves. so most cells in the body don't tend to wander off, walk away, so to speak, from their site of origin. untrue of cancer cells. cancer cells tend to exhibit increased motility they have, uh, contractural proteins within them, now most cells have these but in cancer cells they're being used, to actively move the cancer cell from one location to another. the final thing you gotta worry about, yo- you've seen we we've gotten rid of the links that hold cells together we've given them the ability to move, the final thing is you've got to_ you're not just moving through open space, tumors are surrounded by tissue, and if cancer cells are gonna move they have to somehow get through that tissue. and the way they do this is through the secretion, of a family of enzymes called proteases, and presumably from your biology background you know what this word

protease means it's any protein, digesting enzyme <*:06 PAUSE WHILE WRITING*> and cancer cells use these proteases because, much of the supporting tissue around the tumor, has protein structures and protein fibers that represent a barrier. so in essence what these proteases do is digest a path, through the surrounding tissue. <*:06 PAUSE WHILE WRITING*> and especially the tissue that we call, the stroma. the stroma is the supporting tissue. perhaps connective tissue you may have heard that term the supporting tissue, of the body. and it consists of, cell types called fibroblasts, which secrete, protein fibers, and these protein fibers come together to form a structure called the extracellular matrix <*:05 PAUSE WHILE WRITING*> a dense network of intertwined protein fibers, and finally so we've got fibroblasts we've got these protein fibers to form the extracellular matrix, and we also have blood vessels, in the stroma. and we talked about the importance of those, uh to some extent last time. so this supporting tissue contains a mixture of, cells of protein fibers forming this matrix, and blood vessels. and if cancer cells are gonna wander off and invade and infiltrate surrou- surrounding tissues they've gotta digest a path, through this stroma. and as i said they do it through the secretion of proteases, and there's actually, two main proteases that they use, one is called plasminogen activator <*:07 PAUSE WHILE WRITING*> and plasminogen activator, actually catalyzes a reaction. it catalyzes the conversion of a protein called plasminogen... and converts it to a protein called plasmin. and plasmin is an active protease... while plasminogen represents an inactive precursor of that protease. <*PAUSE:06*> now remember i told you that plasminogen activator itself is a protease what is a protease by definition it's something that breaks peptide bonds. so plasminogen activator breaks one of the peptide bonds in this precursor called plasminogen which is normally inactive, by breaking that, peptide bond (and) cleaving off a fragment from its inactive precursor, it converts it to plasmin, which now, in turn can also function as a protease. now you might wonder what is the purpose of, cancer cells secreting one protease whose sole function seems to be, to create a second, different protease. in essence this is an amplification mechanism. cancer cells don't have to, secrete very much plasminogen activator, cuz this is in essence an enzyme. remember enzymes being catalysts required in very very small quantities, so a small amount of plasminogen activator can convert large amounts of plasminogen, to large amounts of plasmin. and plasminogen is found in very high concentration, in the stroma. okay so most of the body tissues and the supporting tissues of the body in the stroma, contain large amounts of plasminogen. it's normally not doing anything it's just sitting there, as an inactive precursor. but when the cancer cells secrete plasminogen activator, it only takes a few molecules they can convert millions of molecules of plasminogen into plasmin, till you get a very very high local concentration of protease, from secreting just a few molecules from the cancer cells. now another protease that's active in, cancer tissue is a, family of protei- proteases called, matrix metalloproteases <*:06 PAUSE WHILE WRITING*> or M-M-Ps <*:04 PAUSE WHILE WRITING*> and these matrix metalloproteases are often, actually produced by the stroma itself the cancer cells somehow stimulate the stroma, to again do something rather, dumb from the point of view of, of the host organism remember last week i told you that, cancer cells cause the normal tissue to produce blood vessels which is sort of dumb in terms of the host because, the blood vessels supported the tumor. now the ho- normal host tissue is doing another thing that's pretty dumb from the point of view of the host, which is producing matrix metalloproteases, on the existence of these cancer cells, and these matrix metalloproteases again help to break down the extracellular, matrix. it appears that both of these families of proteases they do somewhat different things and both of them are needed in order to get efficient, invasion by cancer cells through the stroma. now once, these enzymes have allowed the cancer cells to digest a path through the stroma the, cancer cells can migrate away from the primary site of origin, until they encounter a blood vessel, usually it's a small capillary, a large bus- blood vessel would be harder for cancer cells to get into. but small capillaries which have a wall that's only a single layer thick, cancer cells can actually penetrate that wall, again with the aid of these proteolytic enzymes helping to break down, the structure of that capillary wall, the cancer cells can penetrate and now get into these small blood vessels. so the cancer cells end up doing three things, that allow them, to start off the process of metastasis. first of all they don't stick together, so you don't have_ you know remember i told you you can have enormous benign tumors. benign tumors can weigh fifty or a hundred pounds. and the reason they are benign, is that those tu- cells are all stuck to each other. no matter how, big the tumor gets, you still have just one mass of cells, surgeon goes there removes it no matter how big the tumor, uh, and you're cured. the only case where that's really a problem is if that tumor happens to be growing in your brain, uh, where, clearly if the surgeon doesn't get to it quickly it can cause significant brain damage. but in benign tumors if the cells stick together, you have no problem. malignancy the cells don't stick together, the cells are mobile they wander off, finally they secrete these enzymes and stimulate the stroma to produce more enzymes, which ends up digesting a path through the, surrounding tissues and into blood vessels. so that's the first, of the three steps of metastasis the, uh invasion, of cancer cells in the surrounding tissues and in the blood vessels. once they've gotten into blood vessels, we'll go to roman numeral two now, the second major stage in the process of metastasis, once the cancer cells get through

the walls of these small blood vessels they're now in the circulatory system. and at this point, cancer cells are transported, *<:04 PAUSE WHILE WRITING>* through the circulatory system, to distant place. so through the bloodstream *<:11 PAUSE WHILE WRITING>* in essence the cancer cells are now free to circulate everywhere in the body. *<PAUSE:05>* now you might think, intuitively that once, cancer cells, had entered into the circulatory system the game is sort of over. that there'd be s- no problem getting these cancer cells transported everywhere around the body uh to set up distant metastases. but actually it turns out that the blood stream is not a particularly hospitable place, for cancer cells and in fact very few cancer cells, actually survive the trip. how do we know that? uh, we make lots of statements how do we know these things? well clearly you can't do experiments on how cancers cells survive in the blood stream by injecting cancer cells into the, veins and arteries of humans right? that would not only be unethical, it actually wouldn't work because of the immunological rejection problem we talked about, last week so, you can't study it in humans, again you can't study it in test tube experiments because test tubes don't have circulatory systems, so the only way you can test this is in animals. and the way it's tested in animals, you've gotta have a way, once you've injected the cancer cells into the bloodstream, to follow them, to find out where they are to follow their fates and the way this is done, is by creating populations of radioactive cancer cells,*<:04 PAUSE WHILE WRITING>* so you could take a transplantable tumor for a mouse for example, and grow it in a mouse for a while in the presence of radioactive isotopes put some isotopes into the mouse's diet, or do this in culture, expose these cells to radioactive isotopes, making the rad- the cancer cells radioactive. now it's easy to spot these cells because they're radioactive. so now you isolate some of these radioactive cells, inject them into another animal's, bloodstream *<:12 PAUSE WHILE WRITING>* and you wait to see what happens and you can use a Geiger counter or some other, mechanism for monitoring radioactivity, to find out what's happened to these cancer cells. what you find is that ninety-nine-point-nine percent of those cancer cells are quickly destroyed in the bloodstream... less than zero-point-one percent, will typically survive the trip... a few days later most of radio- most of those radioactive cells, most of the radioactivity has been, uh, discharged from the body. broken down gotten rid of. so most cancer cells, and this is_ remember, with inbre- inbred strains of animals, these animals should be able to genetically accept these tumor cells that we're working with, yet very few of the cancer cells survive. so it's been concluded that in fact only a tiny, fraction of the cancer cells of a typical tumor, can in fact survive in the bloodstream. and therefore, lead to the process of metastasis. now what determines, whether or not a cancer cell in fact can do this, if they're supplied in the blood stream, ultimately set up a distant metastasis. is it random, is the one cell or so out of a thousand that survives the trip and ends up setting up a metastasis, is that just a lucky cell that somehow escaped all the, pitfalls of traveling through the bloodstream? or was there something special about that cell something unusual, that allowed it, to survive the trip? well if you open your coursepack to page twenty-nine, uh sorry page twenty-one, let's talk about this experiment, which addresses that question, of whether there's something special about the cells, that metastasize. *<PAUSE:07>* these experiments involve a uh, a melanoma cell line, remember this is a malignancy of pigmented cells of the skin, uh a melanoma called, B-sixteen. it's a mouse, transplantable tumor. and it rarely metastasizes. if you want to implant some of these cancer cells under the skin of a typical mouse, um, and you wait a few weeks for it to grow into large tumors eventually you will find, a few metastases in the lungs. you might find one or two or three if you wait several weeks but not very many. but let's take, some of the cancer cells out of one of those successful metastases, isolate those cancer cells, put them under the skin of another mouse, and repeat the process a second time. wait a few weeks, look in the lungs, you'll find this time there may be four or five metastases a few more than you got the first time. again isolate those cells, inject them into a third mouse. and repeat this over and over, ten successive times. after ten repeats we have a cell population which we refer to as melanoma, B-sixteen they're still the cell same, cancer cell type, but we call it the F-ten generation it's the tenth, transplanted generation. where you've selected each time, four cells that metastasize, and reinject the dose. the melanoma B-sixteen F-ten line, metastasizes very very frequently. you inject this into an animal, and in a few weeks you'll find hundreds and hundreds of metastases in the lungs. so it is metastasizing very very frequently. so what can you conclude from this? although it might not be apparent immediately to you in fact these experiments show you that cancer cells vary in the frequency, with which they metastasize. *<:15 PAUSE WHILE WRITING>* now how do we know that cell populations vary, that they're not all the same? let's go back and think about this experiment again. what you're obviously doing is selecting each time, for those cells that su- successfully metastasize. if it was sheer luck, just a random fluke that they managed to do it, then the next time you injected those cells in the whole population shouldn't do any better at metastasizing, than the first population did, if it were sheer luck. but that's not what's happening each time it's getting better, because you are selecting, specifically for those cells that know how to metastasize. and by the end you have a highly enriched population you've taken a, cancer cell line where initially, those cells represented a very very tiny fraction of the total population, now those cells capable of metastasizing represent a very high percentage of the

population, because you have been selecting for that particular type of cell. so the cancer cells vary in the frequency with which they metastasize and in this experiment we are selecting preferentially for those cells, that do have that capability. now although these experiments involve, what i might call a gradual change, in the cancer cell population induced by an artificial selection, the experimenter is selecting for this, generation after generation, there is something similar, that actually occurs, in normal situations in people that have cancer. and we refer, to this as tumor progression.<*PAUSE:07*> and this simply refers to the process by which, the cellular composition of the tumor changes with time. <*:26 PAUSE WHILE WRITING*> and this concept is based on the notion that i've just introduced you to that cancer cell, tumors cancer cell populations are heterogeneous, populations of cells they're not all the same. in the experiment we just saw, we selected, for the ability of cells to metastasize. um, in cancer patients sometimes you get that kind of selection for example, you have a primary tumor, and maybe very few of those cells can metastasize, but some of them do say you have a- a primary melanoma in the skin somewhere, and it metastasizes to the liver a couple of them, well those tumors that now grow up on the liver, are cells that, have the ability to metastasize. so those secondary tumors can in turn, metastasize and they'll do it at a much higher frequency, because you'd already selected once, so now you may get cancer cells all over the body, that are quite different from the initial population in the primary tumor. you've gotten a gradual, progression change in the characteristics with time, as metastasis has taken place. uh, this phenomenon may be especially important for understanding what happens, uh during attempts to treat cancer with some of the cancer chemotherapeutic drugs that we're gonna talk about, later in the semester. and what you're gonna tr- learn later in the semester but let me, just give you a preview today, is that cancer patients who are treated with chemotherapeutic drugs often go into what we call a remission. and this word remission simply... refers to a temporary disappearance... of the disease. temporary disappearance. and we know it's temporary because that's what the word remission means. if by some, chance, the drug actually cured the person and it never came back, then we would call it a cure. uh, when it initially happens you don't know whether it's cure or remission, doctors to be safe will usually initially say, well the patient's gone into remission. there's no sign of the tumor now. we don't know whether it's a cure or not we'll have to wait you know five ten years maybe, depending on how fast that tumor was growing and spreading, to know whether it was actually a cure, or just a remission. unfortunately for people who already have metastases and were treated with chemotherapeutic drugs, uh, most of the time, it's actually a remission that occurs. it's rare that a single treatment of chemotherapeutic drugs will completely cure a patient of cancer. why does the disease come back? well what happens is, with the drug you kill maybe ninety-nine-point-nine-nine percent of the cancer cells, but the point-oh-oh-one percent of the cells left behind, again it's not a random, group that's left behind, the cells that are left behind that weren't killed by the drug, are cells that are inherently resistant, to the effects of the drug. so when that, cancer cell population eventually grows back, it's usually, uh can't be successfully treated with that same initial drug. so it's often, when people have remission and then the cancer comes back it's often much much more difficult to treat them the second time, because that population has undergone progression, the cells are more aggressive these are now resistant to the drug, cuz those are the ones you selected for. remember at the end of the exper- uh, class last time, uh we were looking at Folkman's experiment with endostatin and i said the remarkable thing about that experiment, was remember he treated, the cancer cells once in the animal with endostatin, the tumor went away, he removed the treatment for a while and let the tumor come back, when the tumor came back the second time it was just as sensitive to treatment as it was the first time. it wasn't resistant. that's very unusual. with most, cancer drugs, in fact the second time the tumor comes back it is resistant, and it's because this phenomenon, of, uh, tumor progression, over time you are selecting for either the body or with drugs or with metastasis, you are selecting for a more aggressive (composition.) are there any questions on this, phenomenon? it's often misunderstood, so i wanna make sure it's clear to you yeah in back?

S2: um, is tumor progression, necessarily uh, come with uh an enhanced grading, remember how you graded the tumor, it's microscopic

S1: that's a good question. it's not exactly the same thing. remember i told you that tumor grading, was based, solely on the way cancer cells look under the microscope what size of nuclei, metallic index lots of things that you see in the microscope. those traits don't have to change, in order for you to select, for a population that's resistant to a drug or can metastasize more readily, those things tend not to show up under the microscope. so often the tumor has progressed and it's it's quote unquote more aggressive, and yet to you look at it under the microscope and it looks pretty much the same. that's not always the case. sometimes it will look different. it would, it would be great if it always looked different because then you could recognize it. unfortunately it doesn't usually look different, we're looking at different properties here. <*PAUSE:05*> anything else? <*PAUSE:04*> okay, well we've seen that the tumors are

heterogeneous populations of cells they're not uniform but heterogeneous populations of cells, only a tiny fraction of which are capable, of carrying out this process of metastasis. but you can select for those cells over time, and get a population that eventually, is enriched in those cells that carry out metastasis. the next question i wanna address is, how are these cells different? if we are selecting for cells, that are more efficient at the process, of metastasizing, uh what makes that possible? how are they different at the fundamental molecular cellular level, that they can in fact, uh metastasize more readily? as i just indicated from my answer to that question, it's not the way they look. it's obviously gonna be something else. well although we are far from being able to answer this question in detail, it appears that surface properties properties of the surface membrane of a cancer cell have a lot to do, with whether or not it can efficiently metastasize. now let me describe for you one, simple experiment, which makes this point. this is on the next page i believe of the coursepack. <*PAUSE:05*> page, twenty-two.<*PAUSE:04*> this experiment sort of, picks up where the last one took off remember the experiment on page twenty-one, ended up with this, cell line melanoma B-sixteen F-ten, that metastasizes frequently. so we've selected for these cells, that are very very efficient in metastasizing, now we're trying to find out why are we so good at it. what is it about these cells? well if you take these cells and rupture them put 'em into a big blender and, you know, or grind them or something like that to rupture the cells, break them apart into their individual components, isolate out the membranes, using centrifugation to separate the membranes from the rest of the cell, now remember_ not from the rest of the cell from the rest of the cell components we have no cells left at this point. we have only isolated membranes. clearly these by themselves are not cancer they can't grow, these are not cells they're just membrane fragments. but under certain experimental conditions, we can kind of, promote the fusion, of these membrane fragments to intact cells. so let's take these membrane fragments and under these conditions fuse them, to melanoma B-sixteen F-one cells. remember that B-sixteen F-one cells were the first transplant generation from the preceding experiment. these are cells that rarely metastasize. so we're taking cells that are very inefficient at metastasizing, and we're fusing, into their plasma membranes membrane fragments pieces of membrane, from cells, that readily metastasize. what do you end up getting? you end up getting cells that frequent metastasize. so this clearly shows that the plasma membrane, influences the ability, to metastasize. <*PAUSE:13*> so you change the membrane composition, you change the ability of the cell to metastasize. now why is that? what's going on here? well we don't know for sure, uh but if you read the article that i assigned that you were supposed to read prior to today's lecture, you know that we think that interaction between the immune system and this plasma membrane is somehow involved. now for you to thoroughly understand these experiments you need to know, a few basic things, about the principles of immunology, i assume that most of you know these but let me quickly review them, just to make sure we're all on even footing here. if you were to attempt, to graft an organ, a kidney or a heart or even a skin graft something like that, from one person or another, uh the recipient's immune system obviously this is a problem with heart transplants all the time with kidney transplants, the person receiving the organ their immune system recognizes that organ, as being foreign, as coming from somewhere else. and the immune system wants to attack that invading tissue in essence. in order for heart transplants and kidney transplants to work the patients have to be, treated with drugs so-called immunosuppressant drugs, which uh inhibit the immune system suppress the immune system, so that their immune system will not in fact reject those tissues as being foreign. the way the immune system normally, attacks foreign tissues is using a special cell type called the T-lymphocyte. <*PAUSE:05*>the T-lymphocyte is is the main component of the immune system, which attacks, foreign tissues foreign cells, when they're introduced into the body. and when they attack, a foreign cell, what signals them that they should be doing this, is a composition of that foreign cell's plasma membrane. it's something about the plasma membrane that the T-lymphocytes recognize as being foreign. and part of what rec- part of what it recognizes, not completely, but part of what it recognizes, is a plasma membrane glycoprotein. <*PAUSE:05*> i assume you know what this term glycoprotein means it's a, a protein that has some sugar (roots) attached to it, the T-lymphocyte recognizes a plasma membrane glycoprotein, called the major, histocompatibility complex. <*:12 PAUSE WHILE WRITING*> or M-H-C, as we usually abbreviate it. and again you should be familiar with this from the reading that you did for today's lecture. in mice, uh where the experiments i'm, about to talk about took place in mice, several different genes, for M-H-C exist and depending on the cell type or of the inheritance uh, how that mouse was bred, who his parents were, it may get different forms of the gene for M-H-C. and these have been used, to study the process of metastasis. uh the experiments i'm about to describe to you actually i think were pretty well described in that coursepack article, so i will pretty quickly, go over them with you and make sure you completely understand them. remember in that article you read about, two, mouse cancer cell lines yeah, turn to the page in the course pack yeah right uh, this is now on page twenty-two... remember you read about, two cancer cell populations in mice. the D-one-twenty-two cells, and the A-nine cells. remember that these two cancer cell lines differ in their ability to metastasize when you inject them into animals. D-one-twenty-two cells metastasize very very frequently,

A-nine cells metastasize quite rarely. these cell lines also differ in the ability of, their ability to elicit an immune response. if we just measure the ability of the recipient mouse's, immune system see whether its T-lymphocytes, are attacking mounting a response against those cancer cells, you find that the D-one-twenty-two cells when injected, elicit only a weak immune response, while the A-nine cells, elicit a very, strong immune response. this goes along with the fact that these two cancer cell lines D-one-twenty-two and A-nine, have different M-H-C genes being expressed. so the D-one-twenty-two cells have only the H-two-D form, of M-H-C in their plasma membranes, while the A-nine cells have both the H-two-D form, and the H-two-K form, of the M-H-C molecule in their plasma membranes. now if you were to just look at these experiments, all you could really conclude, is that a strong immune response *<:05 PAUSE WHILE WRITING>* is correlated *<:05 PAUSE WHILE WRITING>* is correlated with... a low rate of metastasis. *<:10 PAUSE WHILE WRITING>* in other words the A-nine cells that metastasize rarely that's correlated with a strong immune response vice versa, if a weak immune response that's correlated with a high rate of metastasis. but the key word here is correlation. you cannot prove cause and effect here, from this kind of observation. if i were to have jumped to the conclusion that the immune strong immune response elicited by the A-nine cell, is responsible for their low rate of metastasis if i concluded that, solely based, on these data i would be committing the post hoc fallacy. the kind of fallacy we were talking about in discussion section last week. the fallacy that when you see two things that go together, A goes with B therefore concluding that A causes B. clearly that's a logical fallacy. it might be something else, that's causing, this low rate of metastasis other than the strong immune response. so this is just a correlation. how can we move from a correlation, to a cause and effect, proving that there really is a cause and effect relationship. this is an important issue, and something hopefully you've been thinking about this past week because i sort of challenged you, at the end of last week's discussion to think about, how could you design a better experiment, to show the relationship between birth control pill usage and cancer, if there was in fact a relationship how could you unequivocally show one way or another, whether there was a cause and effect relationship so you've hopefully been thinking about this, and, one of the things that you might have been thinking about is doing some experiments. these are just passive observations. you gotta do some experiments, if you wanna prove cause and effect. one of the experiments, you again should have read about, in the coursepack article, was this experiment. *<PAUSE:04>* the D-one-twenty-two cells remember, metastasize, very very frequently and that's the_ correlated with a weak immune response and we thought maybe the immune system, has something to do with it. well if the immune system does have something to do with it, we could test that, by changing the M-H-C gene being expressed in the D-one-twenty-two cell. so let's take these D-one-twenty-two cells and transfect them with H-two-K D-N-A. transfection is a technique if you haven't heard it about it from your biology background let me just quickly say it's a, it's a laboratory method for introducing D-N-A into cells i'll talk more about it next week in m- in more detail, but it's just a way it was it was briefly described in the coursepack article as well, just a way to get foreign D-N-A into cells so we're getting this, D-N-A coding for the H-two-K, M-H-C gene, into the D-one-twenty-two cells. as soon as we do that and inject those cells now, back into mice, now they metastasize quite rarely. they're suddenly behaving like A-nine cells. in terms, of their ability to metastasize. and they now elicit a strong immune response. just like, the A-nine cells. so this is much more direct, evidence to the fact that the immune system does in fact influence *<:07 PAUSE WHILE WRITING>* the ability to metastasize. *<:08 PAUSE WHILE WRITING>* when we use the word influence we're talking cause and effect. well we know now it's cause and effect because we've done an experiment. we've changed the immune response by changing the M-H-C gene that's being expressed, on the surface of those cells, the M-H-C protein that's being expressed, once we do that the immune system now recognizes the D D-one-twenty-two cells, it attacks them just like it attacked the A-nine cells, and drives down their rate of metastasis. so the immune, response the ability of the immune system to recognize and potentially destroy, cancer cells, clearly can interfere with their ability to metastasize. these experiments, clearly show that. but is the immune system, the only, factor involved? or are there other variables that influence the ability to metastasize? well there was one more, very clever experiment described in the coursepack article, involving the use of immunosuppressed animals. if you wanna find out, whether or not, the immune response, differences in the immune response is the only variable, influencing the rate of metastasis the way to test that, is to inject cancer cells, by comparison into immunosuppressed animals. these are animals that have been treated with drugs that suppress their immune system, or there are strains of animals that have an inherently defective immune system, either way we're dealing with immune-immunosuppressed animals which cannot, mount an immune response. if the immune response were the only thing determining whether cancer cells can metastasize or not, then the difference between the D-one-twenty-two behavior and the A-nine behavior in terms of metastasis oughta be completely obliterated, in immunosuppressed animals right? if the only, reason that they're behaving differently, in rates of metastasis is because they elicit different immune responses, then you get rid of the immune

response, you should get rid of the difference. and those two cell lines should metastasize at the same rate. notice that that's not what happens. the D-one-twenty-two cells still metastasize at their initially high rate as you would expect, this tells you the immune system is, basically not capable, under these conditions of attacking, the D-one-twenty-two cells. the A-nine cells in the immunosuppressed animals, remember, in normal animals they gave you a low rate, of metastasis, now they go to a medium, rate of metastasis they metastasize more frequently, telling you that the immune system was in fact inhibiting their ability to metastasize, but they still don't metastasize as well as D-one-twenty-two. fo- so this suggests that there are additional factors that the immune system yes is one factor but there are other factors as well, that can differentiate between different cell populations in terms of their ability, to metastasize. any questions on, this set of experiments? <*PAUSE:05*> clear? if you have any, any trouble at all when you go over your notes trying to follow what's going here again this set of experiments is pretty well described, in the coursepack article that i, assigned (to take home.) <*PAUSE:04*> okay. so we've now talked in detail about the first two steps in the process, of metastasis. we talked about the ability of cancer cells to invade through surrounding tissues in terms of penetrating the vessels, and now we just talked about the transport of cancer cells via the bloodstream, uh to distant sites of the body, and we've seen that most of the cancer cells die along the way most of them don't make it, and clearly the immune system is part of the explanation, for why, all the cells don't make it the immune system can clearly attack cells along the way, but there must be some other factors involved as well. this now brings us to the third, step in the process of metastasis, and that is the ability of cancer cells to reinvade and grow at various sites. <*:18 PAUSE WHILE WRITING*> now i'm gonna say at specific sites and you'll see why i use this particular word, in the next few minutes. what determines, where cancer cells will actually end up, reinvading into the tissue somewhere else setting up housekeeping and forming an actual metastasis. turns out that there are two different factors that play a role here. one of 'em is, pure topography. it's based on the anatomy, of the circulatory system. <*:15 PAUSE WHILE WRITING*> and if you'll turn to page twenty-four, in the coursepack i've got a diagram here, to help you understand... what's going on... this is pure geography we're talking about. and we're gonna talk about three potential locations, where primary tumors might arise there are, only three, i should say or three major categories of places where tumors can arise. now let's start off with the broadest category which is category one down here, which is called body tissues and organs, and that, literally means every tissue and organ in the body, where cancer cells can arise with the exception of the lungs, and the stomach and intestines cuz the lungs and stomach and intestines behave a little differently, than we'll see in a few moments. so let's talk about (with the) majority of the organs in the body let's say, an individual had an osteosarcoma in the leg bone for example. where are those cells likely to metastasize to? well as you saw in step one, they're gonna_ cancer cells as they invade through the bone are eventually gonna, encounter blood vessels somewhere, and, the blood vessel they're, li- most likely to invade into is gonna be a very tiny capillary cuz it's got the thinnest wall. so it invades into the capillaries and the capillaries immediately_ now the fluid flow the blood flow will be going towards the small veins, and the small veins feed into the larger veins which feed into the larger veins and finally end up, in the right chamber of the heart. so we're going into bigger and bigger plumbing. okay we're starting out with cancer cells in a very very narrow tube, going into bigger and bigger plumbing where there's more and more room for them, and so we end up in the right side of the heart. from the right side of the heart, these cancer cells in along with the blood are gonna be pumped into the lungs. in the lungs the, pulmonary arteries break up into a series of veins so the blood, uh in a series of capillaries, so that the blood can become oxygenated. so we go back down to very very very tiny, vessels again. which is the way we started. well when we started down here in capillaries of course the cancer cell was burrowing a hole into the vessel, uh through its protease and so forth, when it gets back, here back into capillaries, it's possible_ certainly the cancer cell's gonna be, slowed down and often, the diameter of a cancer cell is such that it's gonna ver- have a very hard time fitting through a capillary. and if you have a happen to have a couple of cancer cells sticking together, it will be almost impossible for them, to pass through the capillary. so this is the first place, where the cancer cells are gonna have a hard time getting through the plumbing, because the plumbing has gotten so small so narrow. so for most cancers that arise in the various body tissues and organs other than the lung and the stomach and intestines for most cancers, the first place they're likely to get hung up, is in the lung. so the first place you would tend to look for metastases in th- is in the lungs. and the lungs are in fact a major site for metastases, for many kinds of cancers. what about for tumors that start in the stomach and intestines, the so-called gastrointestinal tract? here again the cancer cells will infiltrate into very tiny capillaries which will feed in the small veins which go into larger veins which go into yet larger veins, but these veins will then enter the liver. and in the liver, they will break up back into a capillary bed. uh the purpose of this being for the exchange of the, the nutrients that have been taken up and detoxification and so forth all kinds of things have to happen in the, in the liver, and the blood supply therefore breaks stuff into these little tiny capillaries in the liver, so you can have the various exchange of molecules that needs to take

place there, but lo and behold you have the same problem that you had before in the lungs. these cancer cells now are likely to be as big as or bigger than the diameter of these capillaries, and therefore likely to get hung up and stuck. so the most common site or one of the most common sites for stomach cancer and colon cancer to metastasize to, is the liver. because that's the first capillary bed, those cells encounter. finally what about lung cancer? in ways lung cancer's the worse scenario of all, we've talked about the terrible prognosis for lung cancer, and here's one of the reasons that lung cancer has such a terrible prognosis. kills so many people within five years. a cancer starting out in the lungs will go into these capillaries. the, blood flow will then push them into the, vessels they get bigger and bigger, as they go into the, left chamber of the heart, from the left chamber of the heart, uh, the vessel gets even bigger it's pumped out into the aorta, the huge vessel, cancer cells have no problem, they're gonna be pumped all over the body, through all the arteries of the body they'll be distributed everywhere, and, everywhere, they go they will eventually encounter a capillary bed. okay? they could end up in here they could end up in the stomach and intestines they could end up in the liver they could end up in all the body tissues and organs so lung cancer is a very very nasty actor. because its cells immediately are pumped out into the entire circulatory system, and get access to capillary beds where they get stuck, all over the body. so anatomy is the first factor the basic anatomy of the circulatory system is the first factor, that determines, where cancer cells are gonna metastasize. but those simple rules those three rules i just gave to you, aren't the whole story. how do i know they're not the whole story? let's look at another experiment. and there's, an experiment on page, twenty-five in the coursepack. <*PAUSE:08*> if you take mouse melanoma cells like the ones we've been talking about today, inject them into the tail vein, of a mouse, what's likely to happen? well from the diagram on page twenty-four you know from the veins you get eventually pumped into the, uh right side of the heart, from the right side of the heart you get uh, the cel- the cells are pumped into the lungs where it breaks up into the capillary bed, so we do expect these cells initially to get hung up in the lung. and in fact if you look, one or two days after you've injected those cancer cells in the lungs, you will find lots of cancer cells, lodged in the lungs. if you wait two to three, weeks for actual metastases not just individual cells but, metastases to grow up solid tumor modules that you can see and, actual tumors, in fact you get lots of these tumors in the lung. as you would expect, from the rules that i just gave you. but you're gonna see now when we looked at the right side of this, experiment that those rules, weren't the whole explanation for what just happened. let's do another experiment with the same exact cell type, but inject them into the left, ventricle of the heart. as you see from the diagram on twenty-four if you inject cells or cells cancer cells that are in the left, chamber of the heart, from there they are pumped out into the aorta, which means that they are pumped out from the arteries all over the body, and so like lung cancer cells themselves you would expect those to get lodged all over the body. and in fact within the first day or two if you look under the microscope at various tissues, you can find cancer cells lodged, in various tissues throughout the body. but if you come back two or three weeks later to see well where are tumors actually growing, where are metastases actually occurring, they're occurring almost predominately almost entirely in the lung. so this tells us it's not just the anatomy of the circulatory system. there's something else going on here. okay not just the, circulatory system there seems to be something, about these mouse melanoma cells, don't overgeneralize there seems to be something about these mouse melanoma cells, that allows them, to preferentially grow in the lung even though, they've lodged everywhere in the body, they only seem to grow well, in the lung. there's some kind of affinity, some kind of hospitality going on there. these melanoma cells like the lung. it's not just that the_ it was first place they stopped, it wasn't just the first motel they checked into, there was something, nice about that place, so whether it was the first place or not they end up, occupying the lung. so this gives us, a second, principle, that determines, the sites at which cancer cells are gonna grow in terms of metastases. the second principle, which we can conclude from that experiment, is that some cancer cells... prefer to grow... at specific sites... that's why i put the specific sites in this third stage. some cancer cells prefer to grow at specific sites certain sites. some kind of, we think biochemical affinity there's just something about the molecules and environment there, in certain tissues that certain kinds of tumor cells like to grow there. now if that's true it raises a very interesting question. is this the same for all cancer cells? i've only shown you experiments thus far for melanoma, and we haven't even looked at all melanoma cells, we've just looked at melanomas in in this gross sense and found that, as a, total population they seem to, preferentially like the lung. but is that true for all melanoma cells do all melanoma cells prefer the lung? or do some, melanoma cells like other places? now there's been some very clever experiments carried out to address this question if you'll turn to page twenty-six, in the course pack <*PAUSE:06*> you'll see an experiment here which is a variation, on the theme of the experiment described on page twenty-one. i urge you to make a note in your notes not to confuse, experiments on page twenty-one and twenty-six. they look very similar to each other they both involve these sequential, selections and transplantations but the conclusions that are drawn are significantly different, because the experiment is done in a somewhat different way. so make sure you, don't confuse, the experiment on twenty-one, the experiment i'm now

about to go over. in this experiment, they took_ some some mouse melanoma cells were taken and injected into the tail vein of the mice, just (male) mice just like, the experiment on twenty-one, on twenty-one we focused on the lung. we saw that, metastases occurred, initially few in the lung and we selected those and enriched for those. here we're gonna do something a little different. it's true, that metastases occur, mainly in the lung. remember, when you start off, i said you only maybe got one or two of those. so, you get many many more metastases in the lung than you will in the brain and ovary. and you may have to look at dozens and dozens of mice, before you'll find one where there's a single metastasis in the brain, or a single metastasis in the ovary. so these metastases to these organs are much less frequent, than to the lung. is this just a random fluke, did they just happen to get, stuck occasionally in the brain and and it ended up growing there, or was there something special about those cells? well let's do now our sequential transplantation with, cancer cells from these two different organs. start out with the brain. we'll take that one brain metastasis that we found by scouring, you know through thirty or forty mice. take that one brain metastasis remove it, inject it into another mouse. into the tail vein. again you'll find metastases predominantly in the lung, but every once in a while you'll find one in the brain, or the ovary. this time you might find them a little more frequently in the brain than you had the first time. let's take the metastases from the brain again take those cancer cells isolate them inject them into another mouse. and repeat this over, ten times in a row. you repeat it ten times in a row and you will end up with a cell population, that, metastasizes mainly to the brain. metastasizes more frequently to the brain than to the lungs... you do the parallel set of experiments with the ovarian metastases you get a comparable result. eventually you can select for cells that metastasize mainly to the ovary. they metastasize more frequently to the ovary than they do to the lung, and certainly way more frequently to the ovary these would metastasize to the brain. so this tells you that cancer cells vary, in the sites to which they preferentially metastasize. <> again not all cancer cells are not the same. we saw earlier that all cancer cells were not the same in terms of their, ability to metastasize the frequency with which they metastasize. now we see that they're not all the same in terms of the sites to which they like to metastasize. so from a single, cancer cell population, you can isolate, subsets subpopulations of cells that preferentially metastasize, to different organs. now remember when we talked about the question of why some cells metastasize more frequently than others, uh i described to you some experiments which indicated that properties of the plasma membranes, appear to be involved in determining the frequency or influencing the frequency with which cancer cells metastasize. although the evidence isn't quite as good, in this case, there's also some evidence suggesting that differences in the plasma membrane, help to determine, which organ a cancer cell likes, to metastasize to. so we've seen with the melanoma we can isolate, uh, cancer cells that metastasize to the lung those that like the liver uh those that like the ovary those that like the brain, and if you look at the plasma membranes, of those three different subsets, of cancer cells in that population, their plasma membranes look somewhat different not under the microscope in terms of their biochemical makeup. so it appears to be interactions between the plasma membrane, and components of these different target tissues be it brain ovary or lung, that causes cancer cells of different types to preferentially metastasize, to one, organ or another. so the plasma membrane appears to influence not just the frequency, with which cells metastasize, but also where <*AUDIO DISTURBANCE*> (generally.) now once cancer cells, have picked the appropriate site, either from, just being lodged there, to the anatomy of the circulatory system, or through some more preferential mechanism some kind of biochemical affinity which, helps them to grow at a certain site, once this has happened, uh there are again some other factors, that come into play that determine whether or not extensive growth will take place... extensive growth of metastases requires a couple things <*:09 PAUSE WHILE WRITING*> the first thing it requires is angiogenesis. the process we talked last time. just like primary tumors which must, somehow trigger, switch the balance between angiogenesis stimulators and inhibitors, tip that balance towards stimulation to trigger the formation of blood supply, in order to grow beyond a millimeter or two in diameter, the same thing is true of distant metastases. they must tip that balance in the favoring of angiogenesis at the distant site, before they can grow, beyond a millimeter or two in diameter. and remember you have this complicating factor that if there's a large primary tumor somewhere else in the body, if that large primary tumor, is producing large amounts of angiostatin which can spill over into the bloodstream, unlike these other regulators of angiogenesis, that angiostatin can circulate throughout the body, and show up at one of these sites where there's a little tiny tumor nodule, and that little tumor nodule with this massive amount of angiostatin, in the cirulat- circulation will not necessarily be able to overpower that inhibition, and so you may have these silent nodules of a millimeter or two, for long periods of time, until something triggers, that balance for net angiogenesis... so the molecules that regu- regu- regulate angiogenesis clearly play a role. another family of molecule, that appear to play a role, are growth factors. i'm not saying that_ unlike angiogenesis, which is always required, i'm not implying that growth factors are always required but they may play a role. they may play a role. now just to make sure that you're clear on what a growth factor is if you'll turn to page twenty-seven... of the coursepack... i've briefly outlined for

you here, what a growth factor is, uh, first of all, these are non-nutritional. you think, think of things making you grow often you'll think of nutrients these are not nutrients. these are not nutrients. these are, protein molecules, but very specific kinds of protein molecules, that stimulate the growth and division of particular cell types. they don't non-descriminately just stimulate the growth of any cell type but there are different growth factors, which selectively, stimulate the growth, of certain kinds of cells. for example, there's a growth factor called epidermal growth factor or E-G-F, protein that stimulates the growth of epithelial cells. there's a growth factor called platelet-derived growth factor, P-P-G-F, plays an important role in wound healing. uh stimulates predominantly the growth, growth of fibroblasts. uh you've not heard of either of those before, i'm not gonna hold you responsible for those two yet at this part of the course later in the semester later we're gonna come back, talk about these in some detail so at that point i'll hold you responsible for them, but notice that there are a couple, fibroblast growth factor F-G-F vascular endothelial growth factor V-E-G-F, these are ones you already know about. these are ones i will hold you responsible for knowing about, in this part of the course, because those are ones, that stimulate the growth of blood vessels, amongst other things, and therefore these are the growth factors that regulate the process, of angiogenesis. so the only two you read in this list that you really, have to know about right now are these two that regulate, angiogenesis. how do different growth factors manage to selectively, turn on the growth and division of specific cell types? the answer is that different cell types have different kinds of receptor molecules, on their plasma membrane surfaces. these are proteins expressed on the surface of cells, depending on the receptor, that you have on a cell it will bind to different kinds of growth factors so i've tried to illustrate this, by having a complementary shape, between a surface on this growth factor, and this receptor on the surface of this cell. so this growth factor will only bind to cells that contain this particular kind of receptor. the receptor for that specific growth factor. and the binding, of the growth factor to that receptor will, send a signal through the cell, and by the way later in the semester we're gonna talk an awful lot about all the steps involved in transmitting that signal, because the transmission of that signal is intimately associated, with the loss of control of growth in cancer cells so, we're gonna come back to this later in the semester and talk a lot about this pathway, in detail. but for the moment, it'll be just be sort of a black box we won't talk about the individual steps. growth factor binds to a specific receptor, it sends a signal into the cell which ultimately triggers, the division, of that cell. now, if you read the, coursepack article closely, you will, perhaps recall or maybe not but, you should go- be able to go back and find it, the D-one-twenty-two cell line, behaves a little different regarding growth factor from the A-nine. remember the D-one-twenty-two that was the one, that metastasized more frequently than the A-nine, remember although the immune system played some role, that when we, injected D-one-twenty-two and A-nine cells into immunosuppressed animals to get rid of the immune response as a variable, even though we got rid of the immune response as a variable, D-one-twenty-two still metastasized somewhat better, than A-nine. uh there wasn't as great a difference as before, but it still metastasized somewhat better. so there must be an additional difference, between D-one-twenty-two and A-nine, other than the difference in the M-H-C which enhances the immune response. i can now tell you that this other difference is the fact, that there's a gene called the FMS gene F-M-S *<:06 PAUSE WHILE WRITING>* which has become activated in the D-one-twenty-two cells, but not in the A-nine cells. now what is the significance of this gene? this particular gene *<:05 PAUSE WHILE WRITING>* codes for a growth factor receptor. *<:05 PAUSE WHILE WRITING>* remember the receptor is the molecule on the surface of the cell to which a growth factor binds, therefore triggering that cell to grow and divide. so the D-one-twenty-two cells, have a higher concentration of growth factor receptor, on the cell surface. this means that, for a given concentration of growth factor in the tissue, the A-nine cell, which has fewer growth factor receptors, isn't gonna be influenced as much, isn't gonna have its growth stimulated as much, as the D-one-twenty-two cells, because they have more, a higher concentration of growth factor receptor, therefore their growth will be stimulated more, in the presence of the same concentration of growth factor. any question on, this set of observations? *<PAUSE:07>* uh-uh, okay? okay, then, i wanna remind you, that there are three steps, as we've seen in the process of angiogenes- uh, sorry, process of metastasis, uh there's penetration, uh, of surrounding triss- tissue infiltration invasion of surrounding tissues and penetration of the blood vessels, was step one, uh, transport by the circulatory system to distant sites influenced by the immune system and other factors are step two, reinvasion and growth, finally of the distant site was step three. what i want to emphasize to you about the existence of these multiple steps not only do we have multiple steps, but remember we have multiple variables, influencing, each one of those steps impacting each one of those steps. so we have a very complex cascade of events taking place in the process of metastasis, and, only a small number of cancer cells can successfully go through every one of those stages and exhibit all of those properties, that you need in order to get through all the steps in the process of metastasis. and that's why very very few cancer cells actually successfully metastasize. a very very tiny fraction, of a can- cancer cell population successfully metastasizes. i mean if a person, had, you know a hundred metastases that would be considered to be an enormous number. but

you've got tumors that have millions and millions if not billions of cells in them. so only a very very tiny fraction of cells, successfully gets through that complex, series of events. the important point, the important take-home lesson here, is that if we could successfully interfere with just one of these steps, just one of these things that i talked about today, if you could stop the motility of the cancer cell in stage one, if you could inhibit the production of proteases or interfere with their action, uh if could promote the interaction of the immune system, if you could influence any one of those steps, to the detriment of the cancer cell to the detriment of the cascade, then metastasis would not, take place, and if metastasis didn't pla- take place, cancer wouldn't be a disease that we'd have to worry about. okay that concludes what i wanted to say about metastases and i promised you i would occasionally let you go early this semester and today's one such day, but next time we'll talk about more properties, of cancer.

{END OF TRANSCRIPT}

6.4.4. SOURCE 4: ENGLISH FOR ENVIRONMENTAL SCIENCE (GLOSSARY)

A
ABIOTIC
ACCESS
ACID RAIN
AERATE
AEROSOL
AGGREGATION
AGRICULTURAL
AGRICULTURAL SECTOR
AGRICULTURE
AGRONOMIST
AIR QUALITY
ALTITUDE
ALUMINIUM
ANALYSIS
ANALYTICAL
ANALYZE
ANIMAL WELFARE
ANTHROPOGENIC
ANTIBIOTIC
AQUIFER
ARABLE
AREA
ARTIFICIAL
ATMOSPHERE
ATMOSPHERIC
ATOM
ATTRIBUTE DATA
B
BACTERIA
BEDROCK
BENZENE
BIODIVERSITY
BIOFUEL
BIOLOGICAL
BIOMASS
BIOSPHERE
BIOTA
BIOTIC
BOTTLE BANK
BREAK DOWN
BREED
BREEDING
BROMIDE
BROMINE
BUSH MEAT
BY-PRODUCT
C
CAPACITY
CARBOHYDRATE
CARBON
CARBON DIOXIDE
CARBON FOOTPRINT
CARBON MONOXIDE
CARCINOGEN
CARNIVORE
CATALYTIC CONVERTER
CATEGORIZE
CHEMICAL
CHLORINE
CHLOROFLUOROCARBONS
CLAY
CLIMATE
CLIMATIC
COAL
COASTAL
COLLECTION
COMMENCIAL FISHING
COMMUNITY
COMPLEX
COMPLEXITY
COMPLIANCE
COMPONENT
COMPOSITION
COMPOUND
COMPUTERIZE
CONGESTION
CONIFEROUS
CONSERVATION
CONSERVE
CONSULTANCY
CONSUME
CONSUMER
CONSUMPTION
CONTAMINANT
CONTAMINATE
CONTINENTAL
CONTINENTALITY
CONVERT
COOLANT
CROP
CROSS-REFERENCE
CULTIVATE
CULTIVATION
CYCLE
D
DAMAGE
DATA
DATABASE
DECOMPOSE
DECOMPOSER
DECOMPOSITION
DEGRADATION
DENITRIFY
DEPLETION
DEPOSIT
DESIGNATION
DESTRUCTION
DETRITIVORE
DETRITUS
DEVELOPED WORLD
DEVELOPING WORLD
DEVELOPMENT
DIGITAL
DIGITIZE/DIGITALIZE
DISPLACEMENT
DISSIPATE
DIVERSITY
DRAIN
DRAINAGE
DROUGHT
E
ECO-FRIENDLY
ECOLOGIST
ECOLOGY
ECOSPHERE
ECOSYSTEM
EDUCATE
EFFICIENCY
ELEVATION
EMISSION
ENCROACH
ENCROACHMENT
ENDANGERED
END-OF-PIPE

ENERGY
ENVIRONMENT
ENVIRONMENTAL
ENVIRONMENTAL CONSULTANT
ENVIRONMENTAL STRATEGY
ENVIRONMENTAL FRIENDLY
ERODE
EROSION
EUTROPHICATION
EVAPORATE
EVAPOTRANSPIRATION
EVOLUTION
EVOLVE
EXCRETE
EXHAUST
EXPLOIT
EXPOSURE
EXTINCTION

F

FACILITY
FALLOW
FAUNA
FERTILIZE
FERTILIZER
FILTRATION
FINITE
FLORA
FOLIAGE
FOOD CHAIN
FOOD SUPPLY
FOOD WEB
FOSSIL FUEL
FUNGUS

G

GARBAGE
GAS
GASEOUS
GENE
GENERATE
GENERATION
GENETIC
GENETICALLY MODIFIED
GEOLOGY
GEOTHERMAL POWER
GLOBAL
GLOBAL WARMING
GORILLA
GRAPHICAL
GRAPHICS
GREEN BELT
GREENHOUSE EFFECT
GREENHOUSE GAS
GROUNDWATER

H

HABITAT
HARDWARE
HARMFUL
HAZARDOUS
HERBIVORE
HIERARCHY
HORMONE
HOUSEHOLD
HUMAN SETTLEMENT
HUNTING
HYDRO POWER
HYDROCARBON
HYDROLOGY
HYDROSPHERE
HYPERLINK

I

IMAGE
IMPACT
IMPERMEABLE
INCINERATE
INCINERATOR
INCORPORATE
INDEX
INDUSTRIALIZED SOCIETY
INEFFICIENT
INFRASTRUCTURE
INPUT
INTENSIVE FARMING
INTERACTION
INTERCONNECTED
INTERRELATED
INTERSECT
INVENTORY
ION
IRRIGATE
IRRIGATION

K

KEYWORD

L

LANDFILL SITE
LAYER
LEACH
LEAN-BURNING
LEGISLATION
LITHOSPHERE
LITTER
LIVESTOCK
LOAM
LOCATION
LOG IN/LOG ON/LOG OFF
LOW-DENSITY

M

MAP
MAPPING
MARITIME
MATTER
MEASURE
MECHANIZATION
MENU
MESOSPHERE
METAMORPHOSIS
METEOROLOGY
METHANE
MICROORGANISM
MIGRATION
MINERAL
MODEL
MODIFY
MOISTURE
MOLECULE
MONOCULTURE
MULTI-MEDIA
MUTATE

N

NATURAL HABITAT
NATURAL RESOURCES
NATURAL RESERVE
NGO (NON-GOVERNMENTAL ORGANIZATION)
NITROGEN
NITROGEN DIOXIDE
NON-RENEWABLE
NUCLEAR
NUCLEAR POWER
NUTRIENT

O

OPERATING SYSTEM
OPERATION
ORGANIC
ORGANISM
OUTPUT
OVERGRAZING
OVERLYING
OXYGEN
OZONE

P

PARTICLE
PARTICULATE
PERMEABLE
PEST
PESTICIDE
PHOTOSYNTHESIS
PHOTOSYNTHESIZE
PHOTOTHERMAL
PHOTOVOLTAIC
PHYTOPLANKTON
PLANNING
PLANT
POACHING
POLLINATOR
POLLUTANT
POLLUTED
POLLUTER
POLLUTING
POLLUTION
POPULATION
POROUS
POWER
POWER PLANT
POWER STATION
PRESERVATION
PRESERVE
PREVENTIVE
PRIMARY
PRIMATE

PRIVATE SECTOR
PROCESS
PRODUCE
PRODUCER
PRODUCTIVITY
PROJECT
PUBLIC
R
RADIATION
RAINFALL
RANGE
RAW MATERIAL
REAR
RECORD
RECOVER
RECOVERY
RECYCLABLE
RECYCLE
RECYCLING
REDUCE
REDUCTION
REFUSE
REGION
REGULATION
RELEASE
RENEWABLE
REPLENISH
REQUIREMENT
RESEARCH
RESERVOIR
RESIDENTIAL
RESIDUE
RESISTANCE
RESOURCE
RESPIRATORY
REUSE
ROOT
RUBBISH
S
SALMONELLA
SAMPLE
SAND
SCHEME
SCRUBBER
SEARCH
SEARCH ENGINE
SECONDARY
SEDIMENTARY ROCK
SEEP
SEPARATION
SILT
SITE
SMOG
SOCIAL COHESION
SOFTWARE
SOIL
SOLAR POWER
SOLVENT
SORT
SPATIAL
SPECIALIZATION
SPECIES
SPHERE
SPRAY
STAKEHOLDER
STANDARD OF LIVING
STATE STATISTICAL
STOCK
STORAGE
STORE
STRAIN
STRATOSPHERE
STRATUM
SUBPOPULATION
SUBSISTENCE FARMING
SUBSOIL
SUBSPECIES
SUBSTANCE
SULPHUR DIOXIDE
SUNLIGHT
SUPPLY
SURFACE WATER
SURVIVAL
SUSTAINABILITY
SUSTAINABLE
SYNTHESIZE
SYSTEM
T
TABLE
TABULAR
TARGET
TECHNIQUE
TECHNOLOGY
TEMPERATURE
TERTIARY
TEXTURE
THERMAL POLLUTION
THERMOSPHERE
THREAT
THREATENED
TIDAL POWER
TOOL
TOPSOIL
TOXIC
TRANSFORMATION
TRANSPORT
TRANSPORTATION
TRASH
TROPICAL
TROPOSPHERE
TROPOSPHERIC OZONE
TURBINE
U
ULTRAVIOLET
UNDERLYING
USAGE
V
VAPOUR
VARIABLE
VEGETATION
VIABILITY
VIABLE
VOLUME
W
WASTE
WASTE MANAGEMENT
WATER CYCLE
WATER TREATMENT
WAVE POWER
WEATHER
WEATHERED
WILDLIFE
WIND FARM
WOODLAND
WORLDWIDE
X
Y
YIELD
Z
ZOOLOGY

6.4.5. SOURCE 5: ENGLISH FOR ICT STUDIES (GLOSSARY)

A
ACCELERATION
ACCESS
ACTION SEQUENCE
ACTIVE
ADDING MACHINE
ADDITION
ADOPT
ADVANCE
AJAX
ANALOGUE
ANALYTICAL
ANALYZE
ANNOTATION TOOLS
ANTIVIRUS
APPLICATION
APPLICATIONS SOFTWARE
APPROACH
ARITHMETIC
ARITHMETIC LOGIC UNIT
ARCHIVE
ASSESSMENT
ASYNCHRONOUS
AUGMENTATION
AUGMENTED REALITY
AURAL DATA
AUTOMATED
AVATAR

B
BACK END
BALANCE
BANDWIDTH
BARRIER
BEHAVIOUR
BENEFICIAL
`BIG BROTHER´
BILLING
BINARY
BIOMETRIC
BLACK HAT (HACKER)
BLOG
BOTNET
BREAKDOWN
`BRICKS AND MORTAR´
BROADBAND
BROWSE
BROWSER
BUG
BULLETIN BOARD
BUSINESS TO BUSINESS
BUSINESS TO CONSUMER

C
CACHE
CALCULATE
CALL CENTRE
CAPABILITY
CAPACITY
CENSORSHIP
CENTRAL PROCESSING UNIT
CHANGE
CHIP
CHILLER
CIVIL LAW
CLIENT
CLIENT CONSENT
CLUSTER
CODE
COG
COGNITIVE PSYCHOLOGY
COGNITIVE SYSTEMS
COGNITIVE WALKTHROUGH
COMMERCIAL
COMMON
COMMUNICATION
COMPONENT
COMPLEX
COMPUTER-ASSISTED
COMPUTERIZE
CONCEPT
CONFIDENTIALITY
CONFLICT
CONNECT
CONNECTIVITY
CONNECTOR
CONSUME
CONSUME PROTECTION
CONSUMER TO CONSUMER
CONTINGENCY PLANNING
CONTROL
CONTROL UNIT
CONVENTION
CONVERT
COPYRIGHT
CORE
COST
CPU BOARD
CREATOR
CRIMINAL LAW
CRYPTOGRAPHY
CURRENT
CYBERCRIME
CYCLE

D
DATA
DATA CENTRE
DATA INTEGRITY
DATA PROCESSING
DATA PROTECTION
DATABASE
DECIMAL
DECODE
DECRYPT
DENIAL OF SERVICE
DEPENDABILITY
DEPENDENT
DESIGNER
DESKTOP PUBLISHING (DTP)
DEVELOPMENT
DEVELOPMENT METHOD
DEVELOPMENT TOOLS
DEVICE
DIGIT
DIGITAL
DIGITAL RIGHTS MANAGEMENT
DISCIPLINARY ACTION
DISK DRIVE
DISPUTE RESOLUTION
DISRUPT
DISTRIBUTE
DIVISION
DOCUMENTATION
DOWNLOAD
DRIVER

E
E-COMMERCE
ELECTRONIC
ELECTRONIC COMMERCE
ELECTRONIC COMMUNICATION
ELECTRONIC POINT OF SALE
EMBEDDED
ENCODE
ENCRYPT
ENERGY PROPORTIONALITY
ENGINE
ENHANCING
ENVIRONMENT
ESSENTIAL
ETHICAL
EVALUATION
EVENTUALITY
EVOLUTIONARY
EXPAND
EXPLOIT
EXPORT
EYE TRACKING

F
FACTOR
FAULTY
FEATURE CREEP
FEATURES
FIXED
FLASH
FLASH DRIVE
FLEXIBLE
FLEXIBILITY
FOCUS GROUP
FORGERY
FRAUD
FUNCTION
FUNCTIONALITY

G
GENERAL PURPOSE SYSTEM
GEOSPATIAL

GIGABYTE
GLOBAL
GLOBALPOSITIONING SYSTEM
GOODS AND SERVICES
GOVERNMENT LEGISLATION
GRAPHICAL
GREY HAT (HACKER)
GROWTH CURVE

H

HACKER
HAPTIC DATA
HARD DRIVE
HARDWARE
HARDWARE SPECIFICATIONS
HARMFUL
HEURISTIC
HEURISTIC EVALUATION
HOST
HOT SWAPPING
HYPERLINK
HYPERTEXT
HYPERTEXT TRANSFER PROTOCOL

I

IDENTITY THEFT
IDLE
ILLEGAL ACTIVITIES
IMMEDIATE ACCESS STORE
IMPACT
IMPORT
INCLUDE
INCORPORATE
INCREASE
INCREMENT
INCREMENTAL
INDEX
INDEXING
INFORMATION
INFORMATION PROCESSING
INFRASTRUCTURE
INITIATIVE
INNOVATION
INPUT
INPUT DEVICE
INSTALL
INSTRUCTION
INTELLECTUAL PROPERTY
INTERACTION
INTERACTIVE
INTERACTIVITY
INTERFACE
INTERFACE DESIGN
INTERNET
INTRANET
INVENTION
INVENTOR
ITERACTIVE
ITERACTIVE MODEL

J

JOB
JOYSTICK
JUNCTION

K

KEY
KEYBOARD
KEYLOGGER
KEYWORD
KILOBIT

L

LAMP (LINUX, APACHE, MySQL, PHP)
LASER PRINTER
LAUNCH
LAYOUT
LEAD TO
LEGAL INFRASTRUCTURE
LEGAL SITUATION
LEGISLATION
LIFELOGGING
LIMITED:
LIMITATION
LINK
LINUX OPERATING SYSTEM
LOCAL
LOG
LOG IN/ON
LOG OFF
LOGGING

M

MACHINE
MAGNETIC TAPE
MAINTENANCE
MALWARE
MASK
MEASURE
MEMORY
MENTAL MODELS
MENU
METAPHOR
MICROCHIP
MICROPROCESSOR
MIGRATION
MILITARY
MIRROR
MIRROR WORLDS
MOCK-UP
MONITOR
MONITORING DEVICE
MOORE´S LAW
MOTHERBOARD
MOTOR
MOTOR SYSTEM
MOUSE
MULTIPLICATION
MUTI-PURPOSE

N

NEGLIGENCE
NETWORK
NEURAL NETWORKS
NON-COMMERCIAL
NUMERICAL

O

OBJECT ORIENTED
OBLIGATION
OBSOLETE
ONLINE
OPEN SOURCE
OPERATING SYSTEM
OPTIMAL
OPTIMUM
OUTAGE
OUT-OF-THE-BOX
OUTCOME
OUTPUT
OUTPUT DEVICE
OUTSOURCING

P

PAYMENT
PAYMENT COLLECTION
PEER-TO-PEER (P2P)
PERSONAL COMPUTER (PC)
PENETRATION
PENETRATION TESTING
PERCEPTION
PERCEPTUAL SYSTEM
PERSISTENT TREND
PHASE
PHISHING
PHYSICAL REALITY
POSITIONING
PORTABLE
PLUG-IN
PREDICTION
PRE-HYPERTEXT PROCESSOR
PREVALENT
PRIVACY LEGISLATION
PRIVATE
PROCEDURE
PROCESS
PROCESSOR
PROCUREMENT
PRODUCT
PROGRAM
PROGRAMMER
PROJECT MANAGEMENT
PROJECTOR
PROMOTION
PROPRIETARY
PROTECTION
PROTOCOL
PROTOTYPE
PROTOTYPING MODEL
PUBLIC
PUNCHCARD
PURCHASE

Q

QUALITATIVE
QUALITY ASSURANCE

R

RADICAL
REDUCE
REFLECT
REFRESH

REGULATORY FRAMEWORK
RELAY
RELEASE
RELIABILITY
RELOAD
REPUTATION
REQUIREMENTS
RESOURCES
RESPONSIBLE
RESTART
RESULT IN
REVERSE ENGINEERING
REVOLUTIONARY
REVOLUTIONIZE
ROBOT
ROLE-PLAY
ROOT ACCESS
RULE

S

SCALE UP
SCAN
SCANNER
SCHEDULING
SCREEN
SEARCH
SEARCH ENGINE
SEARCH RESULTS
SECURELY
SECURITY
SELECT
SENSOR
SERVER
SERVER FARM
SHARE
SKILLS
SOCIAL ENGINEERING
SOCIAL NETWORKING SERVICES
SOFTWARE
SOFTWARE ENGINEER
SOFTWARE EXPLOIT
SOFTWARE PACKAGE
SPECIFIC
SPECIFICATION
DOCUMENT
SPECIALIZED
SPIDER
SPIRAL MODEL
STABLE
STACK
STAGE
STATIC
STATUS QUO
STORAGE
STORE
SUBMIT
SUBTRACTION
SUCCESSFUL OUTCOME
SURVEILLANCE
SWITCH
SYSTEM
SYSTEM DEVELOPMENT
SYSTEM LOGGING
SYSTEMS SOFTWARE

T

TARGET SYSTEM
TASK
TASK ANALYSIS
TECHNICAL INFRASTRUCTURE
TECHNOLOGICAL
TECHNOLOGY
TELECOMMUNICATIONS
TELEWORKING
TESTING
TEXT
THEFT
THINKING ALOUD
TIMESCALE
TOLERANCE
TRACKING
TRANSACTION
TRANSFORM
TRANSITION
TRANSMIT
TRANSISTOR
TROJAN
TROUBLESHOOTING
TRUST

U

UBIQUITOUS COMPUTING
UNINTERRUPTIBLE POWER SUPPLIERS
UNPATCHED EXPLOIT
UPGRADE
USABILITY
USABILITY TESTING
USER ANALYSIS
USER INPUT
USER INTERFACE
UTILIZATION

V

VACUUM TUBE
VALVE
VARIABLE
VERSION
VIDEO-CONFERENCING
VIRTUAL
VIRTUAL LEARNING ENVIRONMENT
VIRTUAL STOREFRONT
VIRTUAL WORLDS
VIRTUALIZATION
VIRTUALLY ENHANCED
VIRUS
VISUAL DATA
VISUAL DISPLAY
VOICE OVER IP (VoIP)
VOLTAGE REGULATOR CIRCUITRY

W

WATERFALL MODEL
WEARABLE COMPUTER
WEB PAGE
WEBSITE
WHITE HAT (HACKER)
WIRELESS
WIRELESS APPLICATION PROTOCOL
WORK OUT
WORLD WIDE WEB (WWW)
WP (WORD PROCESSING)