SCIENTIA PLENA
www.scientiaplena.org.br
VOL. 18, NUM. 8 2022
doi: 10.14808/sci.plena.2022.084811

A random walk model to qualify echolalias

Um modelo de passeios aleatórios para qualificar ecolalias

T. R. S. Moura[1,2*]

[1]*Faculdade de Física, Laboratório de Inovação Interdisciplinar, Campus Universitário de Salinópolis, Universidade Federal do Pará, 68721-000, Salinópolis-Pará, Brasil*
[2] *Programa de Pós-Graduação em Propriedade Intelectual e Transferência de Tecnologia para Inovação, Universidade Federal do Sul e Sudeste do Pará, 68507-590, Marabá-Pará, Brasil*

**trsmoura@yahoo.com.br*
(Recebido em 30 de novembro de 2021; aceito em 13 de maio de 2022)

Some neurodevelopmental disorders such as Tourette syndrome, Asperger syndrome, autistic spectrum disorders (ASD), persistent vocal tic disorder, transient vocal tic disorder, psychotic disorder due to another medical condition, catatonia associated with another mental disorder, etc; may represent speech fluency problems characterized by the presence of pathological repetition of speech, prolongation of consonants or vowels, broken words, symptoms of communicative disorders called echolalia. Observing this context, this work presents a model of random walks to qualify echolalia using the representation of information entropy. The model presents results consistent with the different types of echolalia, namely, interactive, non-interactive, delayed and non-delayed, including combinations of these echolalia that characterize comorbidities in some patients, such as non-delayed and non-interactive echolalia, non-delayed and interactive echolalia, delayed and non-interactive echolalia, and delayed and interactive echolalia. Each of these types of echolalia was quantified using random walks, measuring the information entropy for each one of the cases which can range from mild, moderate to severe.
Keywords: echolalia, random walks, entropy.

Alguns transtornos do neurodesenvolvimento como a síndrome de Tourette, a síndrome de Asperger, transtornos do espectro autista (TEA), transtorno de tique vocal persistente, transtorno de tique vocal transitório, transtorno psicótico devido a outra condição médica, catatonia associada a outro transtorno mental, etc; podem resultar em problemas na fluência da fala caracterizados pela presença de repetição patológica da fala, prolongação de consoantes ou vogais, palavras quebradas no início da vida, sintomas de desordens comunicativas denominadas ecolalias. Observando este contexto, este trabalho apresenta um modelo de caminhadas aleatórias para qualificar ecolalias usando a representação da entropia da informação. O modelo apresenta resultados consistentes com diversos tipos de ecolalias, a saber, interativa, não-interativa, atrasada e não atrasada, incluindo combinações dessas ecolalias que caracterizam comorbidades em alguns pacientes, tais como a ecolalia não-atrasada e não-interativa, a ecolalia não-atrasada e interativa, a ecolalia atrasada e não-interativa e a ecolalia atrasada e interativa. Cada um desses tipos de ecolalia foi quantificada usando caminhadas aleatórias, medindo a entropia da informação para cada um dos casos que podem variar de leve, moderado a severo.
Palavras-chave: ecolalia, caminhadas aleatórias, entropia.

1. INTRODUCTION

This work is the result of the combination of two general topics of scientific interest: random walks and developmental disorders. In this proposal, random walks are the modeling tool and mental disorders are the modeled object.

Random walking is a topic of common interest in several fields such as hydraulic engineering, meteorology, physics, chemistry, mathematics, economics, materials science, medicine, etc. [1-5]. In medicine, random walks are used as a tool to model memory loss and amnesia, modeling diseases such as Alzheimer's and Autistic Spectrum Disorders (ASD), are related to building computational models for brain tissue, developing new algorithms for software applications. and hardware, new capacitance calculation methods, image analysis and the development of innovative technologies for mobile devices [6-21].

According to the World Health Organization (WHO) mental disorders are varied, have different representations, and can be characterized by a combination of emotions, perceptions,

behaviors and abnormal relationships. For the WHO, depression, bipolar disorder, schizophrenia and other psychoses, dementia and developmental disorders, including ASDs, are classified as mental disorders [22, 23].

Developmental disorders are classified as specific and invasive. The specific disorder is characterized by developmental delays in a specific area or specific areas, whereas the invasive disorder presents deficiencies in basic functions in multiple contexts that include socialization and communication. Pervasive Developmental Disorders (PDD) are part of the group of autism spectrum disorders (ASD). Autism spectrum disorders are prevalent on average, by country, in 1% of the world population, ranging from 0.8% to 1.1% among countries. As for gender, boys are more likely to be diagnosed with ASD than girls. The odds are between 4 to 5 times greater for boys than for girls diagnosed with ASD. In 2016, the global estimate was that 62 million people have some type of ASD, with approximately 50 million males and 18 million females [24].

ASDs are classified as non-degenerative neurodevelopmental disorders, accompanied by symptoms such as delay in verbal and non-verbal communication; resistance to routine change; restricted and persistent interests in relation to an activity, topic, object, speech, idiosyncratic phrases, etc.; abnormalities in eye contact and body expression; difficulties in initiating and maintaining social relationships. Symptoms can range from mild, moderate or severe [25].

Problems in speech fluency characterized by the presence of pathological repetition of speech (sounds or syllables), prolongation of consonants or vowels, broken words in early life. These are symptoms of communication disorders called echolalias. The Diagnostic and Statistical Manual of Mental Disorders (DSM5) describes a significant spectrum of neurodevelopmental disorders that share communicative disorders such as echolalia as a common feature. Examples of neurodevelopmental disorders that present these symptoms are: Tourette's syndrome, Asperger's syndrome, autistic spectrum disorders (ASD), persistent vocal tic disorder, transient vocal tic disorder, psychotic disorder due to another medical condition, catatonia associated with another mental disorder, etc. [25].

There are two main characteristics for the classification of echolalias: social interaction and time for speech execution. Another important aspect is comorbidity. As for social interaction, the first characteristic highlighted, there may or may not exist social interaction. When there is social interaction, this symptom is classified as interactive echolalia (or functional echolalia), i.e., there is an attempt to communicate with the aim of interaction. Therefore, interactive echolalia is the pathological, parodic and apparently meaningless (echoing) repetition of a word or phrase that has just been spoken by another person [25]. When there is no attempt at communication for social interaction, this symptom is termed non-interactive echolalia, i.e., the communication is for personal use only. Non-interactive echolalia is the repetition or imitation of an echo made by a person in relation to words just uttered by that same person [25]. Second, the time-to-speech characteristics are divided into two categories, namely, immediate echolalia and delayed echolalia. Immediate echolalia occurs in the repetition of speech immediately after the speaker's emission. Delayed echolalia is characterized by speech reproduction after a long time of emission of words by the interlocutor [26]. As for the third aspect, comorbidity, it occurs when two or more diseases occur in the same individual. Looking at interactive echolalia and non-interactive echolalia as two distinct diseases. When a patient is observed with these two diseases simultaneously, he presents comorbidity associated with these communicative disorders [25].

There are several studies that seek to understand the causes, the correlations between neurodevelopmental disorders and pathological repetition of speech, providing treatment manuals and specialized training for health professionals and society in general [27-33].

In this work, heuristically incorporating memory effects of non-Markovian processes, a stochastic physical model is proposed to evaluate echolalias. To achieve the goal, a model that allows an appropriate analogy between pathological speech repetitions and stochastic dynamics. The chosen model was the so-called Elephant Random Walk (ERW), a non-Markovian random walk model with memory. The reasons for choosing this model are that the ERW: (a) presents well-known diffusive regimes, (b) the propagator is known, (c) has a well-known analytical solution [4], (d) is a model that is being widely used as a basis for constructing other non-Markovian random walk models [26-32] and, (e) there are analogies between this model and other neurological problems characterized by memory loss [6-10]. In these terms, I propose the

model of random walks with memory inspired by echolalias. In this context, the first approach the stochastic dynamics of non-Markovian random walks at the lattice scale, modeling the different types of echolalia, namely, interactive, non-interactive, delayed and non-delayed, including combinations of these echolalia such as non-delayed and non-interactive echolalia, non-delayed and interactive echolalia, delayed and non-interactive echolalia, and delayed and interactive echolalia. Second, information entropy measurements to qualify the echolalias is performed.

2. MATERIAL AND METHODS

In this section, the random walk model to qualify echolalias is constructed. To achieve the goal, the ERW model in N-dimensions is introduced, highlighting relevant features of the ERW model combined with the standard symptoms of echolalias to build the model, and to finalize the analysis, information entropy measurements are shown.

2.1 ERW Model

According to Schütz and Trimper (2004) [4], for reasons of simplicity they consider a one-dimensional random walk $X_t^{(i)} \in Z$ in infinite lattice. The i-th random walker starts the walk at the origin ($X_0^{(i)} = 0$) at time $t = 0$. The superscript (i) was not introduced in the ERW model, its insertion in the notation is appropriate to capture important characteristics of echolalias. The explanation of this fact will be left for the appropriate time. Returning to the construction, the random walker has a memory of its entire trajectory. At every discrete instant of time the random walker moves one step to the left or right. Its trajectory is quantified by the stochastic equation

$$X_{t+1}^{(i)} = X_t^{(i)} + \sigma_{t+1}^{(i)} \tag{1}$$

And $\sigma_{t+1}^{(i)}$ is a random variable that can take values of ± 1. The memory is constituted by the set of variables $\sigma_{t'}^{(i)}$ of steps taken at previous instants of time that the random walker remembers as follows:

1. in the time: $t = 1$, the i-th walker, initially in the position:$X_0^{(i)}$, where $\sigma_1^{(i)} = +1$ ($\sigma_1^{(i)} = -1$) with probability $q^{(i)}$ $(1 - q^{(i)})$. The probability of the first step is

$$P\left[\sigma_1^{(i)}\right] = \frac{1}{2}\left[1 + \left(2q^{(i)} - 1\right)\sigma_1^{(i)}\right] \tag{2}$$

2. in the time: $t + 1$, for the walker (i) a time t' is uniformly chosen from the set $\{1,2,3, \ldots, t\}$;
3. $\sigma_{t+1}^{(i)}$ is chosen stochastic by rule $\sigma_{t+1}^{(i)} = +\sigma_{t'}^{(k)}$ $(\sigma_{t+1}^{(i)} = -\sigma_{t'}^{(k)})$ with probability $p^{(i\to k)}$ $(1 - p^{(i\to k)})$;

$$P\left[\sigma_{t'}^{(k)}\right] = \frac{1}{2}\left[1 + \left(2p^{(i\to k)} - 1\right)\sigma_{t+1}\sigma_{t'}^{(k)}\right] \tag{3}$$

4. in the time: $t + 1$, walker i chooses one of the walkers with index $k = 1,2,\ldots,M$ with probability $\gamma^{(i\to k)}$. This probability must satisfy the relationship $\sum_{k=1}^{M} \gamma^{(i\to k)} = 1$.
5. according to rules (1) and (4), the probability of the first step is

$$P\left[\sigma_1^{(i)} = \pm 1\right] = \frac{1}{2}\sum_{k=1}^{M}\left[1 + \left(2q^{(i)} - 1\right)\sigma\right]\gamma^{(i\to k)} \tag{4}$$

6. according to the rules (2)-(4), the probability of the step of the i-th walker, in relation to the k-th walker, in time $t+1$, $\sigma_{t+1}^{(i)} = \sigma$, where σ comes from the spectrum of possibilities of the whole $\{\sigma_{t'}^{(k)}\}$, with $k = 1,2,\ldots,M$, is

$$P\left[\sigma_{t'}^{(1,\ldots,M)}\right] = \frac{1}{2t}\sum_{k=1}^{M}\left[1 + \left(2p^{(i\to k)} - 1\right)\sigma\sigma_{t'}^{(k)}\right]\gamma^{(i\to k)} \quad (5)$$

where $\gamma^{(i\to k)}$ is the coupling probability of the i-th walker in relation to the k-th walker. It, ($\gamma^{(i\to k)}$), quantifies the extent to which the microscopic decisions of the i-th walker are influenced by the microscopic decisions of the k-th.

Solving Equation (5), it calculated the conditioned probability $P\left[\{\sigma_{1,2,\ldots,t}^{(1,2,\ldots,M)}\}\right]$, i.e.,

$$P\left[\{\sigma_{1,2,\ldots,t}^{(1,2,\ldots,M)}\}\right] = \frac{1}{2} + \frac{\sigma}{2}\sum_{k=1}^{M}\alpha^{(i\to k)}\,\gamma^{(i\to k)}x_t^{(k)} \quad (6)$$

where $\alpha^{(i\to k)} = 2p^{(i\to k)} - 1$ e $x_t^{(i)} = X_t^{(i)} - X_0^{(i)}$, being the displacement of the i-th walker. The conditional mean displacement of the i-th walker is quantified therefore

$$\langle\{\sigma_{1,2,\ldots,t}^{1,2,\ldots,M}\}\rangle = \sum_{\sigma=\pm1}\sigma P\left[\{\sigma_{1,2,\ldots,t}^{1,2,\ldots,M}\}\right] = \sum_{k=1}^{M}\frac{\alpha^{(i\to k)}\gamma^{(i\to k)}x_t^{(k)}}{t} \quad (7)$$

Solving Equation (7), the found the recursive equation for the first moment of the position:

$$\langle x_{t+1}^{(i)}\rangle = \sum_{k=1}^{M}\left(\delta_{ki} + \frac{\alpha^{(i\to k)}\gamma^{(i\to k)}}{t}\right)\langle x_t^{(k)}\rangle \quad (8)$$

The offset parameter for the first step is defined as:$\beta^{(i)} = 2q^{(i)} - 1$. We found the initial displacement for the i-th walker.

$$\langle x_1^{(i)}\rangle = \sum_{k=1}^{M}\beta^{(i)}\,\gamma^{(i\to k)} \quad (9)$$

2.2 Random Walk to Qualify Echolalias

The build of the random walks model is centered on major characteristics: social interaction, time for speech execution and the presence of comorbidities. Echolalia is divided into two types: non-interactive echolalia and interactive echolalia. To distinguish the echolalias, we used the coupling parameter $\gamma^{(i\to k)}$. The i-th walker chooses one of the index walkers $k = 1,2,\ldots,M$ with probability $\gamma^{(i\to k)}$, must satisfy the relationship $\sum_{k=1}^{M}\gamma^{(i\to k)} = 1$. There are two different cases: $i = k$and $i \neq k$.

i. For the case where $i = k$, the walker mimics its own movement with probability of coupling: $\gamma^{(i\to k)}$. Quantifying the type of echolalia called non-interactive echolalia, a situation in which there is no attempt at communication for social interaction, communication is intended solely for personal use [25]. Greater (smaller) intensities of non-interactive echolalia are obtained the greater (smaller) the coupling: $\gamma^{(i\to k)}$.
ii. For the case where: $\neq k$, implies that the walker mimics the movement of the k-th walker with coupling probability $\gamma^{(i\to k)}$. Therefore, quantifying the echolalia called interactive echolalia, i.e., the pathological, parodic and apparently meaningless repetition (echoing) of a word or phrase that has just been spoken by another person. Greater (smaller) interactive echolalia intensities are obtained the greater (smaller) the coupling: $\gamma^{(i\to k)}$.

Second, the time-to-speech characteristics are divided into two categories: non-delayed echolalia and delayed echolalia. Non-delayed echolalia occurs in speech repetition immediately

after the speaker's emission. Non-delayed echolalia is the reproduction of speech by the individual after a certain period (not immediately) of emission of words by the interlocutor [26].

The modeling of speech delay, starts from the fact that the instant of time that the interlocutor speaks, at time: $t^{(k)}$, repetition by the listener occurs in time: $t^{(i)} > t^{(k)}$, i.e., i-th random walker repeats the movement of the k-th random walker in the time interval: $t^{(i)} - t^{(k)}$. Extreme cases occur when:

iii. Limiting case for non-delayed echolalia: $t^{(i)} - t^{(k)} \to 0$;
iv. Limiting case for a delayed echolalia: $t^{(i)} - t^{(k)} \to \infty$.

Synthesizing points i- iv, combining the different symptoms, there are subtypes of echolalias, namely:

v. To $t^{(i)} - t^{(k)} = 0$ and $i = k$, is the case of the non-delayed and non-interactive echolalia;
vi. To $t^{(i)} - t^{(k)} = 0$ and $i \neq k$, is the case of the non-delayed and interactive echolalia;
vii. To $t^{(i)} - t^{(k)} \neq 0$ and $i = k$, is the case of the delayed and non-interactive echolalia;
viii. To $t^{(i)} - t^{(k)} \neq 0$ and $i \neq k$, is the case of the delayed and interactive echolalia.

According to the above description, the phenomenon of delayed echolalia can be quantified by the probability: $\tau_A^{(i\to k)} = \frac{(t^{(i)} - t^{(k)})}{t}$ of the i-th random walker repeat the movement of the k-th random walker in the time interval: $t^{(i)} - t^{(k)}$, where t is the total time of the walk. The letter (A) receives the following nomenclature: $(A = I)$ for non-delayed echolalia and $(A = D)$ for delayed echolalia. In this way, the above cases v-viii are reformulated as follows:

ix. To $\tau_D^{(i\to k)} = 0$ and $i = k$, is the case of the non-delayed and non-interactive echolalia;
x. To $\tau_D^{(i\to k)} = 0$ and $i \neq k$, is the case of the non-delayed and interactive echolalia;
xi. To $\tau_D^{(i\to k)} \neq 0$ and $i = k$, is the case of the delayed and non-interactive echolalia;
xii. To $\tau_D^{(i\to k)} \neq 0$ and $i \neq k$, is the case of the delayed and interactive echolalia.

Objectively addressing the next aspect involved in echolalias: comorbidity. Comorbidity occurs when two or more diseases occur in the same individual. Considering echolalia, self echolalia and speech delay as distinct pathologies. So, if the characteristic symptoms of interactive, non-interactive and speech delay are observed in a patient simultaneously, he presents comorbidities. We quantified the extreme case of comorbidities when, for coupling probabilities $(\gamma^{(i\to k)})$ and speech delay $(\tau_A^{(i\to k)})$, the indexes: $i \neq k$ and $i = k$, $(A = I)$ and $(A = D)$ occur simultaneously.

In Figure 1, we presented an illustration of the types of echolalias with the diagram of stochastic states, summarizing the characteristics of social interaction, time for speech execution and comorbidities.

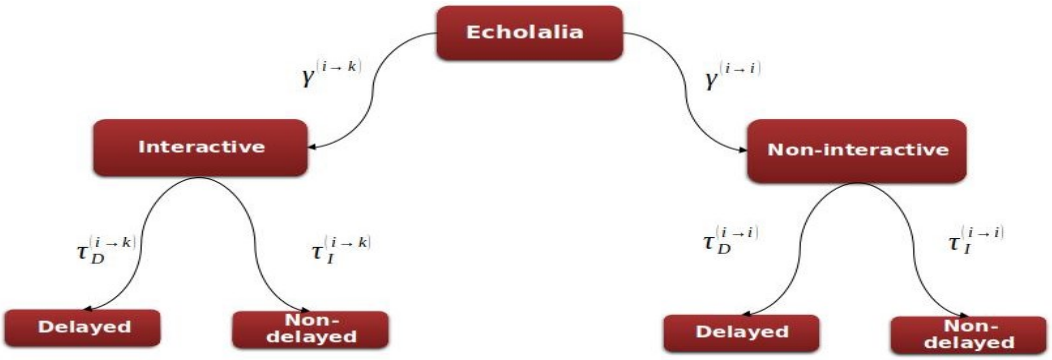

Figure 1: Stochastic states diagram of echolalias.

To introduce the interaction and delay effects in random walks, the memory needs to be defined. The memory is formed by two variable sets $\sigma_{t\prime}^{(1)}$ for $i = 1$, a random variable that can take the values ± 1 and the variable $\rho_{t\prime}^{(j)}$ for $j = 2$, a random variable that can take the value 0. To account for these effects, we introduced a third random walker with index $k = 3$. The third random walker access the values of the variable $\sigma_{t\prime}^{(1)}$ with probability $\tau_I^{(3\to1)}$ and the value $\rho_{t\prime}^{(2)}$ with probability $\tau_D^{(3\to2)}$. The slightest (severe) case of delay occurs for $\tau_D^{(3\to2)} = 0$ ($\tau_D^{(3\to2)} = 1$), intermediate cases occur for the range: $0 < \tau_D^{(3\to2)} < 1$ between these two extreme cases. In this context, using Equation (1), we found the initial displacement for the third walker

$$X_{t+1}^{(3)} = X_t^{(3)} + \tau_I^{(3\to1)}\sigma_{t+1}^{(1)} + \tau_D^{(3\to2)}\rho_{t+1}^{(2)} \tag{10}$$

Writing $x_t^{(3)} = X_t^{(3)} - X_0^{(3)}$ e $x_{t+1}^{(3)} = X_{t+1}^{(3)} - X_0^{(3)}$, we found that

$$x_{t+1}^{(3)} = x_t^{(3)} + \tau_I^{(3\to1)}\sigma_{t+1}^{(1)} + \tau_D^{(3\to2)}\rho_{t+1}^{(2)} \tag{11}$$

Continuing, using Equation (6), the average displacement is

$$\langle x_{t+1}^{(3)}\rangle = \sum_{k=1}^{2}\left(\delta_{k3} + \frac{\alpha^{(3\to k)}\gamma^3\tau_I^{(3\to k)}}{t}\right)\langle x_t^{(k)}\rangle + \tau_D^{(3\to k)}\langle\rho_t^{(k)}\rangle \tag{12}$$

where $\tau_I^{(3\to2)} = \tau_D^{(3\to1)} = 0$.

The displacement parameter for the first step is set to $\beta^{(3)} = 2q^{(3)} - 1$. The initial displacement for the third walker is

$$\langle x_1^{(3)}\rangle = \sum_{k=1}^{2}\beta^{(3)}\gamma^{(3\to k)}\left(1 - \tau_D^{(3\to k)}\right) \tag{13}$$

Notice that $\tau_D^{(3\to k)} = 0$, we retrieved the ordinary ERW model, observed when in this limit equations (12) and (13) are generalizations of equations (8) and (9), respectively.

To mediate the effects of the characteristics of echolalia in random walks, social interaction, time for speech execution and the presence of comorbidities, information entropy measurements for each position is performed, calculating the average probability of visiting this position, we obtained the following formula for the average entropy production of random walks

$$S = -\sum_{k=1}^{\mu} W(x_k)ln\left(W(x_k)\right) \tag{14}$$

where $W(x_k)$ is the probability distribution of finding the random walkers in the position. x_k from the origin, where the sum effectively traverses all allowable length paths μ. The unit of the chosen entropy is the nips because the neperian logarithm of eq(14). For example, if base were 2, the measure of entropy would be in bits.

3. RESULTS AND DISCUSSION

Numerical experiments of finite-sized random walks to 10^4 walkers and 10^7 length. Other parameters taken from equations (1)-(14), for simplicity, were rewritten as $\tau_D^{(3\to2)} = \tau_D$, $p^{(3\to1)} = p$ and $\gamma^{(3\to1)} = \gamma$. These parameters were controlled in the interval of [0,1]. In this way, we can reformulate cases ix-xii as follows:

xiii. To $\tau_D = 0$ and $\gamma = 0$, this is the case of non-delayed and non-interactive echolalia;
xiv. To $\tau_D = 0$ and $\gamma \neq 0$, this is the case of non-delayed and interactive echolalia;
xv. To $\tau_D \neq 0$ and $\gamma = 0$, this is the case of delayed and non-interactive echolalia;

xvi. To $\tau_D \neq 0$ and $\gamma \neq 0$, this is the case of delayed and interactive echolalia.

In accordance with the numerical experiments, the results in the representation of the entropy of information are shown. To display the general picture of the results of the entropy changes as a function of τ_D, p and γ, points xiii-xvi were followed.

In Figure 2 we presented typical measures of information entropy for numerically controlled variations of τ_D, p in the range of [0,1] and fixed γ for the values of 0, 0.1, 0.2, 0.3, 0.5, 0.7 and 1. There were observed two distinct behaviors: one abrupt and one mild. The abrupt behavior occurs in the range of $0 \leqslant \tau_D \leqslant 0.1$, where entropy decays rapidly due to variations in τ_D. Above this range, $0.1 \leqslant \tau_D \leqslant 1$, entropy decays more slowly with variations of τ_D.

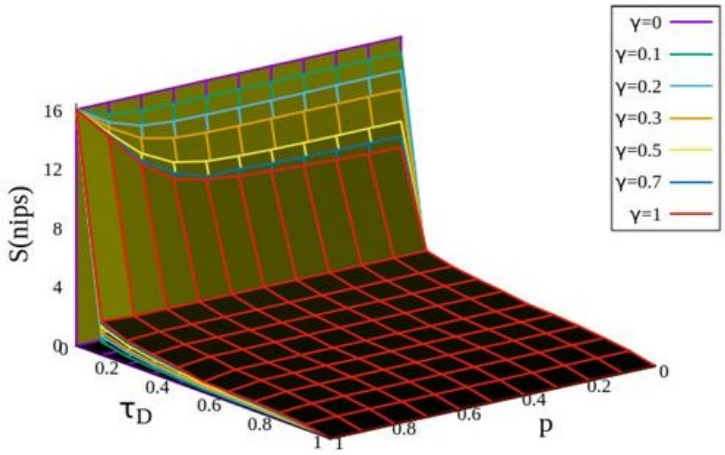

Figure 2: 3D graph of information entropy as a function of the parameters p *and* τ_D *each one in the range of* [0,1]. *The different surfaces are related to the different values of* γ *displayed in the right frame. The first surface, which does not show entropy variations, is at the base (purple lines) for the coupling probability* $\gamma = 0$. *The last surface, with the largest entropy variations, is at the top (red lines) for the coupling probability* $\gamma = 1$ *Between these two extremes we present intermediate entropy variations for* $0 < \gamma < 1$.

For the presentation of the results, it was divided into two parts according to τ_D. First in range:$0 \leqslant \tau_D \leqslant 0.1$ and the second in the range of $0.1 \leqslant \tau_D \leqslant 1$.

To begin by analyzing two cases, the first the case of non-delayed and non-interactive echolalia, at the ends of $\tau_D = 0$ e $\gamma = 0$, and the second case of non-delayed and interactive echolalia, where $\tau_D = 0$ and $\gamma \neq 0$. The results of this region are shown in Figure 3. To highlight them in order to perform better analysis, the curves were reconstructed using, for comparison terms, the same colors as in Figure 2. Specifically, entropy curves are shown for values of $\tau_D = 0$ and various values of the coupling strength γ. Fixing γ and varying p in the interval [0,1], we identified that greater entropy variations are observed for greater coupling intensities. For the case of non-delayed and non-interactive echolalia, $\gamma = 0$, typical measurements of entropy exhibit constant values in relation to changes in p of the order of 16 nips. Therefore, when there is no interaction, larger entropy variations are displayed. For the case of non-delayed and interactive echolalia, $\gamma \neq 0$, at $\gamma = 0.1$, the entropy varies in the range of approximately $15 < S < 16$ nips; for $\gamma = 0.2$, the entropy varies in the range of approximately $14 < S < 16$ nips, following this trend for the other intermediate values of coupling. Similarly, for the highest coupling intensity $\gamma = 1$, entropy exhibits its greatest variation in the range of approximately $8 < S < 16$ nips. Therefore, non-delayed and interactive echolalia is more sensitive to p variations, while non-delayed and non-interactive echolalia does not have this sensitivity.

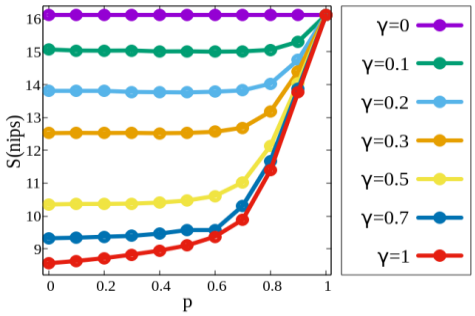

Figure 3: Information entropy variation as a function of the feedback parameter p. The various curves are related to the different γ values displayed in the right frame and delay probability: $\tau_D = 0$. For the parameters $\tau_D = 0$ e $\gamma = 0$, is the case of non-delayed and non-interactive echolalia. However, for $\tau_D = 0$ and $\gamma \neq 0$, is the case of non-delayed and interactive echolalia.

Continuing, we present the second part of our results, namely, for the delay probability interval: $0.1 \leqslant \tau_D \leqslant 1$. In this region of temporal delay, we have the emergence of two cases of echolalia, namely, for $\tau_D \neq 0$ e $\gamma = 0$, we have the case of delayed and non-interactive echolalia and for $\tau_D \neq 0$ and $\gamma \neq 0$, we have the case of delayed and interactive echolalia.

In Figure 4, is presented a 3D graph of the information entropy as a function of the parameters p and τ_D. In it, several surfaces associated with different intensities of coupling probability γ are presented. Also, a color table is available to the right of the surfaces with a different color for each value of γ. For the first surface, in the base, i.e., for the case of delayed and non-interactive echolalia ($\gamma = 0$), no entropy variations are displayed. Therefore, entropy changes are invariant to changes in p and γ. In Figure 4, for the case of delayed and interactive echolalia ($\gamma \neq 0$), we observed that typical measurements of entropy are greater than zero. As we increase the values of $\gamma > 0$ greater entropy variations are observed. The last surface, for the coupling probability $\gamma = 1$, exhibits the greatest entropy variations, bordering region for the entropy variations for the case of delayed and interactive echolalia.

Now, analyzing the impact to probability τ_D, which quantifies walker response delays in entropy measurements. Observing Figure 4, we noticed that the smaller (larger) the values of τ_D larger (smaller) are the variations of the entropy information. This trend is observed for any values of the coupling strength γ. For these results, greater entropy variations are associated with shorter walker response delays. For $\tau_D = 1$, the greatest delay intensity of delayed and interactive echolalia, the entropy variations are null for any values of the coupling strength γ and the feedback parameter p.

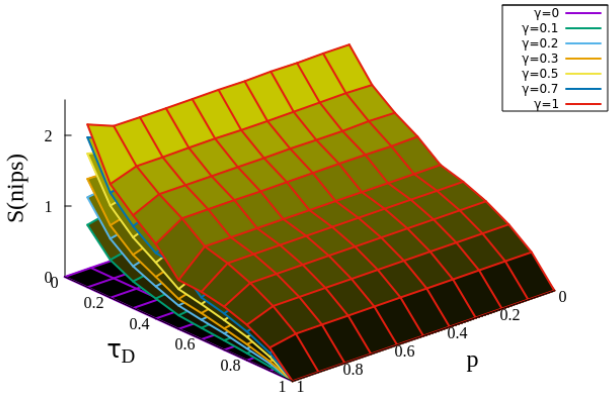

Figure 4: 3D graph of information entropy as a function of the parameters p and τ_D: p in the interval $[0,1]$ and τ_D in the interval $[0.1,1]$. The different surfaces are related to the different values of γ displayed in the right frame. The first surface, which does not show entropy variations, is at the base (purple lines) for the coupling probability $\gamma = 0$. The last surface, with the largest entropy variations, is at the top (red lines) for the coupling probability $\gamma = 1$. Between these two extremes we presented intermediate entropy variations for $0 < \gamma < 1$.

4. CONCLUSION

This work emphasized that problems in speech fluency characterized by the presence of pathological repetition of speech, prolongation of consonants or vowels, and broken words in early life are symptoms of communicative disorders called echolalia. Echolalia are symptoms present in some neurodevelopmental disorders such as Tourette syndrome, Asperger syndrome, autistic spectrum disorders (ASD), persistent vocal tic disorder, transient vocal tic disorder, psychotic disorder due to another medical condition, catatonia associated with another mental disorder, etc.

Then, scrutinizing this context, a random walk model to qualify echolalia in the representation of information entropy was reported. Such a model presents results consistent with the different types of echolalia, namely, interactive, non-interactive, delayed and non-delayed, including combinations of these echolalias that characterize comorbidities in some patients, such as non-delayed and non-interactive echolalia, non-delayed and interactive echolalia, delayed and non-interactive echolalia, and delayed and interactive echolalia.

For the case of non-delayed and non-interactive echolalia, for the lowest coupling strength, we found the largest entropy variations and they are invariant to changes in the feedback probability. For non-delayed and interactive echolalia, when the coupling strength is maximum, entropy exhibits its greatest variation as a function of the variations in the feedback parameter. Therefore, non-delayed and interactive echolalia is more sensitive to variations in the feedback parameter.

In the case of delayed and non-interactive echolalia, there are no entropy changes, i.e., the entropy changes are invariant to the changes in the feedback parameter and the coupling strength.

For the case of delayed and interactive echolalia, for non-null values of the coupling strength, we observed that typical entropy measures are greater than zero. As we increase the coupling strength, greater entropy variations are displayed. Coupling intensity is accompanied by greater entropy variations, characterizing entropy variations in the border region of delayed and interactive echolalia.

As for the impact of delay probability, the data shows that the smaller (greater) the delays, the greater (smaller) the entropy variations. This behavior is observed for any values of the coupling strength. When the delay probability is maximum, characterizes the greatest delay intensity of delayed and interactive echolalia, the entropy variations are null for any values of the coupling strength and feedback parameter.

This theoretical-computational work is not claiming that these cases of echolalia exist, but neither is it saying that they cannot be observed in routine examinations with the help of technically trained professionals. The clinical observation of these cases is beyond the scope of this work. We left proof or rebuttal of the consequences of our theoretical results to healthcare professionals.

5. ACKNOWLEDGMENTS

Thanks to my wife Daniele Moura for the design in Figure 1.

6. REFERENCES

1. Hurst HE, Black RP, Simaika YM. Long-term storage: An experimental study. Constable. 1966;129(4):591-3. doi: 10.2307/2982267
2. Carbonea A, Castellia G, Stanley HE. Time-dependent Hurst exponent in financial time series. Physica A. 2004 Jul;344(1-2):267-71. doi: 10.1016/j.physa.2004.06.130
3. Serletis A, Rosenberg AA. The Hurst exponent in energy futures prices. Physica A. 2007 Jul;380:325-32. doi: 10.1016/j.physa.2007.02.055
4. Schütz GM, Trimper S. Elephants can always remember: Exact long-range memory effects in a non-Markovian random walk. Phys Rev E. 2004 Jun;70:045101. doi: 10.1103/PhysRevE.70.045101
5. Scher H, Montroll EW. Anomalous transit-time dispersion in amorphous solids. Phys Rev B. 1975 Sep;12(6):2455-77. doi: 10.1103/PhysRevB.12.2455

6. Cressoni JC, da Silva MAA, Viswanathan GM. Amnestically induced persistence in random walks. PRL. 2007 Feb;98:070603. doi: 10.1103/PhysRevLett.98.070603
7. Felisberto ML, Passos ES, Ferreira AS, da Silva MAA, Cressoni JC, Viswanathan GM. Sudden onset of log-periodicity and superdiffusion in non-Markovian random walks with amnestically induced persistence: Exact results. Eur Phys J B. 2009 Oct;72:427. doi: 10.1140/epjb/e2009-00361-6
8. da Silva MAA, Viswanathan GM, Ferreira AS, Cressoni JC. Spontaneous symmetry breaking in amnestically induced persistence. Phy Rev E. 2008 Apr;77:040101R. doi: 10.1103/PhysRevE.77.040101
9. Cressoni JC, Viswanathan GM, Ferreira AS, da Silva MAA, Alzheimer random walk model: Two previously overlooked diffusion regimes. Phy Rev E. 2012 Oct;86:042101. doi: 10.1103/physreve.86.042101
10. Borges GM, Ferreira AS, da Silva MAA, Cressoni JC, Viswanathan GM, Mariz AM. Superdiffusion in a non-Markovian random walk model with a Gaussian memory profile. Eur Phys J B. 2012 Sep;85:310. doi: 10.1140/epjb/e2012-30378-5
11. Alves GA, de Araùjo JM, Cressoni JC, da Silva LR, da Silva MAA, Viswanathan GM. Superdiffusion driven by exponentially decaying memory. J Stat Mech. 2014 Apr;(2014):P04026. doi: 10.1088/1742-5468/2014/04/p04026
12. Diniz RMB, Cressoni JC, da Silva MAA, Mariz AM, de Araújo JM. Narrow log-periodic modulations in non-Markovian in random walks. Phy Rev E. 2017Apr;96:062143. doi: 10.1088/1742-5468/2014/04/p04026
13. Moura TRS, Fulco UL, Albuquerque EL. A random walk model to evaluate autism. Physica A. 2018 Dec;492:1694-9. doi: 10.1016/j.physa.2017.11.090
14. Grinberg F, Farrher E, Oros-Peusquens AM, Shah N.J. Random walks in model brain tissue. AIP Conf Proc. 2011 Mar;1330(31):31-34. doi: 10.1063/1.3562226
15. Sotero RC, Sanchez-Rodriguez LM, Moradi N, Dousty M. Estimation of global and local complexities of brain networks: A random walks approach. Netw Neurosci. 2020;4(3):575-94. doi: 10.1162/netn_a_00138
16. Gerstein GL, Mandelbrot B. Random Walk Models for the spike of a single neuron. Biophysical Journal. 1964 Jan;4(1 Pt 1):41-68. doi: 10.1016/s0006-3495(64)86768-0
17. Severa, W, Lehoucq R, Parekh O, Aimone JB. Spiking Neural algorithms for markov process random walk. In: 2018 International Joint Conference on Neural Networks (IJCNN). Rio de Janeiro: IEEE; 2018. p. 1-8. doi: 10.1109/ijcnn.2018.848962
18. Mascagni M, Simonov NA. The random walk on the boundary method for calculating capacitance. J Comput Physics. 2004 Apr;195(2):465-73. doi: 10.1016/j.jcp.2003.10.005
19. Calimera A, Mach E, Poncino M. The human brain project and neuromorphic computing. Funct Neurol. 2013 Oct;28:191-6. doi: 10.11138/FNeur/2013.28.3.191
20. Zeller RC, Pohl RO. Thermal conductivity and specific heat of noncrystalline solids. Phys Rev B. 1971 May;4(6):2029-41. doi: 10.1103/PhysRevB.4.2029
21. Cena F, Rapp A, Mattutino C, Mauro N, Ardissono L, Cuccurullo S, et al. A Personalised Interactive Mobile app for people with Autism Spectrum Disorder. In: Ardito C, Lanzilotti R, Malizia A, Petrie H, Piccinno A, Desolda G, editors. Human-Computer Interaction – INTERACT 2021. Bari (IT): Springer; 2021. p. 313-7. doi: 10.1007/978-3-030-85607-6_28
22. Wang PS, Aguilar-Gaxiola S, Alonso J, Angermeyer MC, Borges G, Bromet EJ, et al. Use of mental health services for anxiety, mood, and substance disorders in 17 countries in the WHO world mental health surveys. The Lancet. 2007 Sep;370(9590):841-50. doi: 10.1016/s0140-6736(07)61414-7
23. James SL, Abate D, Abate KH, Abay SM, Abbafati C, Abbasi N, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet. 2018 Nov;392(10159):1789-858. doi: 10.1016/S0140-6736(18)32279-7
24. Baio J, Wiggins L, Christensen DL, Maenner MJ, Daniels J, Warren Z, et al. Prevalence of autism spectrum disorder among children aged 8 years— Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. MMWR Surveill Summ. 2018 Apr;67(6):1-23. doi: 10.15585/mmwr.ss6706a1
25. Diagnostic and Statistical Manual of Mental Disorders: DSM-5. 5th ed. Washington (US): New School Library; 2013.
26. Mergl M, Azoni CAS. Echolalias's types in children with autism spectrum disorder. Rev CEFAC. 2015 Nov-Dez;17(6):2072-80. doi: 10.1590/1982-021620151763015
27. Howlin P, Asgharian A. The diagnosis of autism and Asperger syndrome: findings from a survey of 770 families. Dev Med Child Neurol. 1999 Dec;41(12):834-9. doi: 10.1017/s0012162299001656

28. Johnson CP, Myers SM. Council on children with disabilities. Identification and evaluation of children with autism spectrum disorders. Pediatrics. 2007 Nov;120(5):1183-215. doi: 10.1542/peds.2007-2361
29. Mandell DS, Maytali MN, Zubritsky CD. Factors associated with age of diagnosis among children with autism spectrum disorder. Pediatrics. 2007 Oct;116(6);1480-6. doi: 10.1542/peds.2007-2361
30. Martinez-Pedraza FL, Carter A. Autism spectrum disorders in young children. Child Adolesc Psychiatr Clin N Am. 2009 Jul;18:645-63. doi: 10.1016/j.chc.2009.02.002
31. Ozonoff S, Young GS, Steinfeld MB, Hill MM, Cook I, Hutman T, et al. How early do parent concerns predict later autism diagnosis? J Develop Behav Pediatrics. 2009 Oct;30(5):367-75. doi: 10.1097/DBP.0b013e3181ba0fcf
32. Pickles A, Simonoff E, Conti-Ramsden G, Flacaro M, Simkin Z, Charman T, et al. Loss of language in early development of autism and specific language impairment. J Child Psychol Psych. 2009 Jul;50(7):843-52. doi: 10.1111/j.1469-7610.2008.02032.x
33. Rogers SJ. Developmental regression in autism spectrum disorders. Ment Retard Dev Disabil Res Rev. 2004;10(2):139-43. doi: 10.1002/mrdd.20027