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Research paper

Modeling of dielectric permittivity in the aggregation of erythrocyte molecules: application to deoxy-hemoglobin S

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Abstract

In this paper, study of the impact of a new treatment approach in the aggregation kinetics of deoxy-hemoglobin S molecules is developed. New approach exploring mathematical model of dielectric permittivity of Davidson-Cole and takes into account several relaxation times of the dielectrics substances like blood. Results are such that, (i) only a fine analysis of intrinsic energy dissipation curves allow sufficient precision to be obtained to describe chronic hemolytic anemia due to sickle cell anemia based on the frequency of relaxation, (ii) hematocrit calculation by the formula $Q = \omega_\circ / \Delta\omega$ allowed to show the uniqueness of the relaxation time of deoxy-hemoglobin S, (iii) hematocrit levels are low and the dispersion coefficient is the parameter most influential in the aggregation of hemoglobin S. From the analysis of all these results, it appears that, the mathematical model of Davidson-Cole is a good model interpretation and knowledge of sickle cell disease. The interest of this work is the understanding of the molecular mechanism that can help develop new treatment approaches capable of preventing or limiting the risk of complications of red blood cell diseases.

Keywords: *Aggregation; Hematocrit Level; Single Relaxation Time; Permittivity Dielectric*.

1. Introduction

Dielectric properties of materials have been extensively studied for several years by physicists and chemists. Recently, dielectric measurements have been used to characterize materials in several fields such as: medicine, engineering sciences and earth sciences. Since several decades, studying the transformation of hemoglobin S gel-solution was described by researchers such as Hofrichter et al., in 1974 [1], then Harris and Bensusan in 1980 [2]. The problem essential that still arises is the relationship between relaxation spectra and microscopic properties of dielectric materials. This description is little developed and so far there is no definitive approach. The goal in this work is to determine by calculation of hematocrit, the sickle cell nature of the deoxy-hemoglobin S. First, the relaxation spectra of the erythrocyte solutions, then the characteristic parameters of which the hematocrit level by the formula $Q = \omega_\circ / \Delta\omega$. The goal is to understand the mechanism molecular aggregation kinetics of deoxy-hemoglobin S molecules using Davidson-Cole empirical models [3, 4] in order to extract information from characteristic parameters of deoxy-hemoglobin S and be able to prevent aggregation of red blood cells.

2. Description of empirical mathematical theories

2.1. Cole-Cole theory

The empirical Cole-Cole model [3,4] can be written in form of the relation (1):

$$\varepsilon^*(\omega) = \varepsilon_\infty + \frac{\varepsilon_0 - \varepsilon_\infty}{1 + (j\omega\tau_0)^\beta} \tag{1}$$

Where, $\varepsilon^*(\omega)$ is the dielectric constant which in general is a complex quantity dependent of frequency ω. The constant ε_0 is the maximum permittivity in a static field ($\omega = 0$), the constant ε_∞ is the minimum permittivity for optical frequencies ($\omega = \infty$), parameter τ_0 is the main relaxation time and the empirical exponent β is the dispersion coefficient whose value is such that $0 \prec \beta \prec 1$. If $\beta = 1$, this Cole-Cole model is reduced to the Debye model [3]. The peculiarity of Debye's model is that, it omits molecular interactions and only

considers a single relaxation time τ_0 . Thus Davidson-Cole and other researchers proposed modifications to Debye's formula by introducing empirical exponents leading to an increase in the number of relaxation times τ .

2.2. Davidson-Cole theory

The model is described by the functional relation (2) [4]:

$$\varepsilon^*(\tau,\omega)=\varepsilon_\infty+\frac{\varepsilon_0-\varepsilon_\infty}{(1+j\,\omega\tau)^\beta} \tag{2}$$

The dispersion parameter β keeps the same physical meaning as before. The complex quantity (2) can be written in the form (3):

$$\varepsilon^*(\tau,\omega)=\varepsilon'(\tau,\omega)+\varepsilon''(\tau,\omega) \tag{3}$$

Where $\varepsilon'(\tau,\omega)$ represents the real permittivity and $\varepsilon''(\tau,\omega)$ represents the intrinsic dissipation power of the dielectric. After a few transformations, we get:

$$\varepsilon'(\tau,\omega)=\varepsilon_\infty+\frac{(\varepsilon_0-\varepsilon_\infty)\cos(\beta\arctan(\omega\tau))}{(1+\omega^2\tau^2)^{\beta/2}} \tag{4}$$

And

$$\varepsilon''(\tau,\omega)=\varepsilon_\infty+\frac{(\varepsilon_0-\varepsilon_\infty)\sin(\beta\arctan(\omega\tau))}{(1+\omega^2\tau^2)^{\beta/2}} \tag{5}$$

The empirical models of Cole-Cole and Davidson-Cole are very powerful and are stable up to 5% of Gaussian white noise. They are used to adjust the data experiments of a wide variety of materials with only four parameters at namely (ε_0 , ε_∞ , τ , β). In the following, we consider the Davidson-Cole model due to the goal of this paper. Hematocrit levels are determined by the formula $Q=\omega_\circ/\Delta\omega$, where $\omega_\circ$ is the frequency at resonance and $\Delta\omega$ is the width of the frequency band intrinsic power dissipation spectra. Reference values for hematocrit levels are between 40% and 52% for men; between 35% and 47% for women and between 32% and 45% for children. A low hematocrit can have as originates from sickle cell anemia, a genetic disease characterized by an alteration of the composite membranes. High hematocrit can be caused by severe dehydration on the body. The maximum permittivity ε_0 and the minimum ε_∞ hemoglobin S gel solutions are well defined in [5]. In our paper, we consider four solution-gel of hemoglobin S. The methods used have led to results.

3. Results

3.1. Dielectric permittivity of deoxy-hemoglobin solutions S

Figs. 1 and 2 respectively show the curves of the relative permittivity and loss factors of the Davidson-Cole model for relaxation times $9{,}3.10^{-13}s$; $3{,}336.10^{-11}s$; $6{,}698.10^{-11}s$ with the dispersion coefficient $\beta=0{,}987$ (Fig.1.a and Fig.2.a) and $\beta=0{,}5$ (Fig.1.b and Fig.2.b).

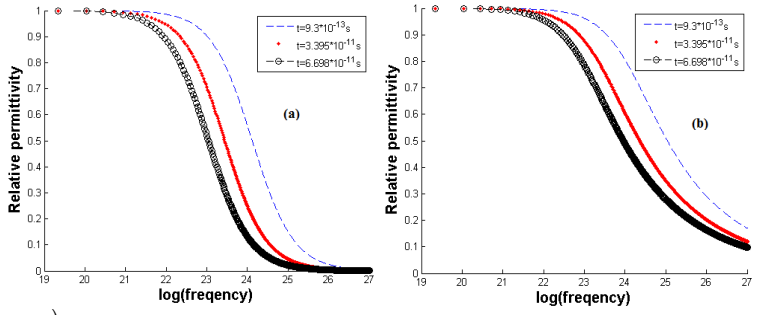

Fig.1: Relative permittivity $(\varepsilon'-\varepsilon_\infty)/(\varepsilon_0-\varepsilon_\infty)$ of the model of Davidson-Cole for three different relaxation times with the coefficient of dispersion $\beta=0{,}987$ (a) and $\beta=0{,}5$ (b).

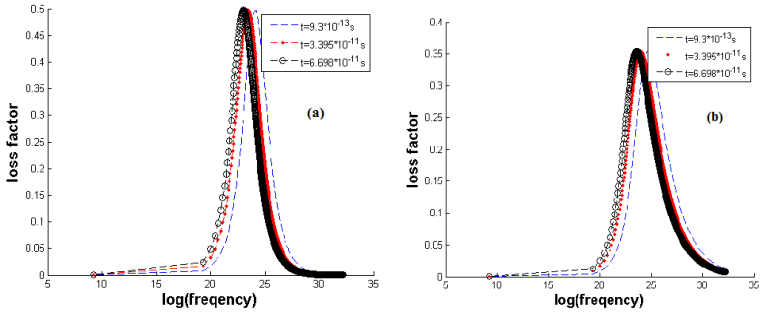

Fig.2: Loss factors $\varepsilon^{"}/(\varepsilon_0-\varepsilon_\infty)$ of the Davidson-Cole model for three different relaxation times with the dispersion coefficient $\beta=0{,}987$ (a) and $\beta=0{,}5$ (b).

The relaxation spectra (Fig.1 and Fig.2) of the Davidson-Cole model show that the deformation during the flow of a material generates loss factor energy. The Gaussian curves of the power loss factors are varied according to the dispersion coefficient β . The characteristic times are in the reference space relaxation times of erythrocyte solutions $\left[10^{-14}s\,,10^{-10}s\right]$

3.1.1. Numerical models: solution 1($\varepsilon_0=114\,;\,\varepsilon_\infty=56$)

Figs. 3 and 4 respectively shows the curves of the real permittivity and the intrinsic power dissipation of deoxy-hemoglobin S (solution 1) for relaxation times $3{,}4.10^{-13}s\,;\,6{,}7.10^{-13}s\,;10^{-12}s$ by varying the dispersion coefficient β .

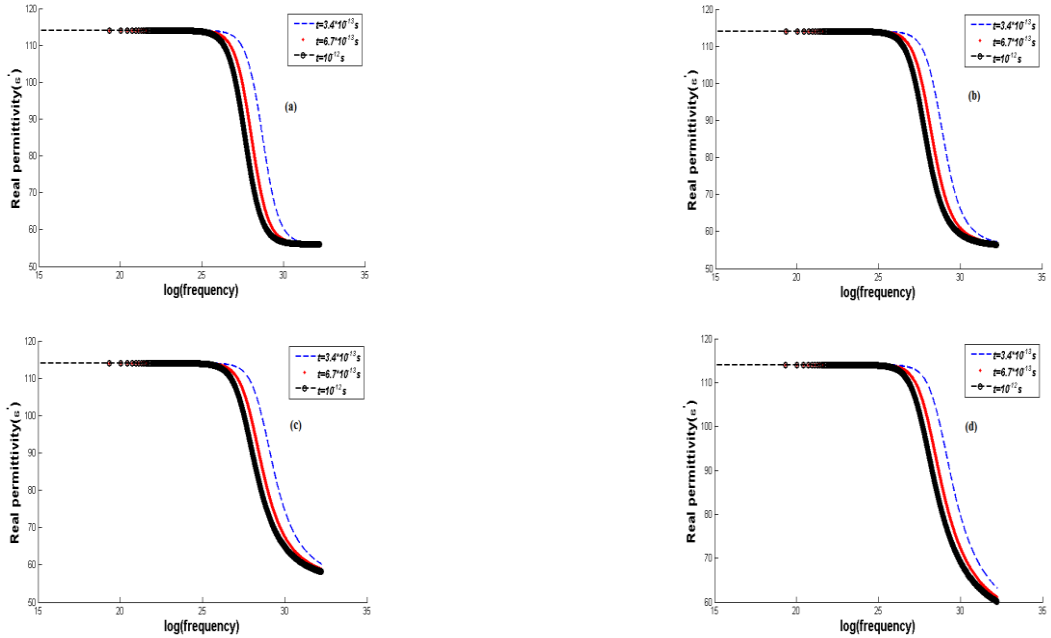

Fig. 3: Real permittivity of solution 1 ($\varepsilon_0=114\,;\,\varepsilon_\infty=56$) for $\beta=0{,}987$ (a); $\beta=0{,}8$ (b); $\beta=0{,}6$ (c); $\beta=0{,}5$ (d).

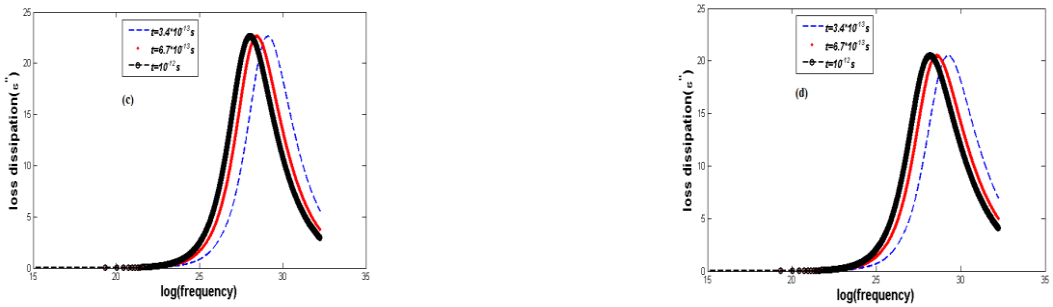

Fig.4: Evolution of the intrinsic power dissipation of the solution 1 ($\varepsilon_0 = 114$; $\varepsilon_\infty = 56$), for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

Tables 1 to 4 present the determination of the hematocrit level of the solution 1 ($\varepsilon_0 = 114$; $\varepsilon_\infty = 56$), for three different relaxation times by varying the dispersion coefficient β .

Table 1: Parameter values $(\tau\,;\omega_0\ ;Q)$ for $\beta = 0.987$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}4.10^{-13}$	28,8335	$2{,}9715.10^{12}$	$5{,}994.10^{12}$	49,57%
$6{,}7.10^{-13}$	28,8335	$1{,}5080.10^{12}$	$3{,}0418.10^{12}$	49,57%
10^{-12}	28,8335	$1{,}0103.10^{12}$	$2{,}038.10^{12}$	49,57%

Table 2: Parameter values $(\tau\,;\omega_0\ ;Q)$ for $\beta = 0.8$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}4.10^{-13}$	26,1788	$3{,}5053.10^{12}$	$8{,}1942.10^{12}$	42,77%
$6{,}7.10^{-13}$	26,1788	$1{,}7788.10^{12}$	$4{,}1585.10^{12}$	42,77%
10^{-12}	26,1788	$1{,}1918.10^{12}$	$2{,}7863.10^{12}$	42,77%

Table 3: Parameter values $(\tau\,;\omega_0\ ;Q)$ for $\beta = 0.6$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}4.10^{-13}$	22,6470	$4{,}4018.10^{12}$	$13{,}079.10^{12}$	33,65%
$6{,}7.10^{-13}$	22,6470	$2{,}2338.10^{12}$	$6{,}6375.10^{12}$	33,65%
10^{-12}	22,6470	$1{,}4965.10^{12}$	$4{,}4472.10^{12}$	33,65%

Table 4: Parameter values $(\tau\,;\omega_0\ ;Q)$ for $\beta = 0.5$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}4.10^{-13}$	20,5061	$5{,}0943.10^{12}$	$18{,}072.10^{12}$	28,18%
$6{,}7.10^{-13}$	20,5061	$2{,}5853.10^{12}$	$9{,}171.10^{12}$	28,18%
10^{-12}	20,5061	$1{,}7320.10^{12}$	$6{,}1447.10^{12}$	28,18%

3.1.2. Numerical models: solution 2 ($\varepsilon_0 = 104$; $\varepsilon_\infty = 54$)

Figs. 5 and 6 respectively shows the curves of the real permittivity and the intrinsic power dissipation of deoxy-hemoglobin S (solution 2) for relaxation times $3{,}101.10^{-11}s$; $6{,}2.10^{-11}s$; $9{,}3.10^{-11}s$ by varying the dispersion coefficient β .

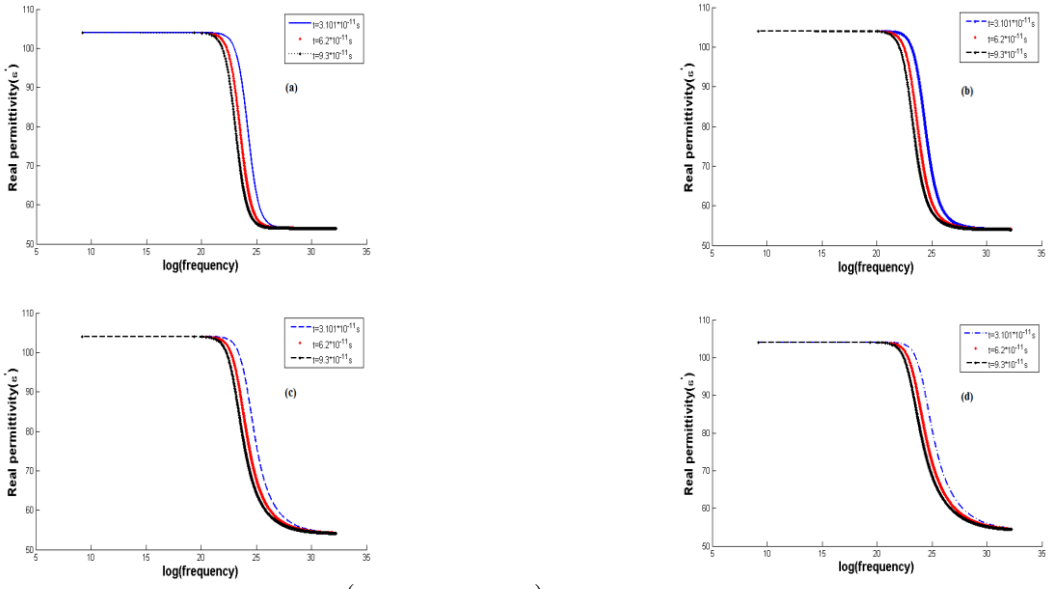

Fig. 5: Real permittivity of solution 2 $(\varepsilon_0 = 104\,;\ \varepsilon_\infty = 54)$ for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

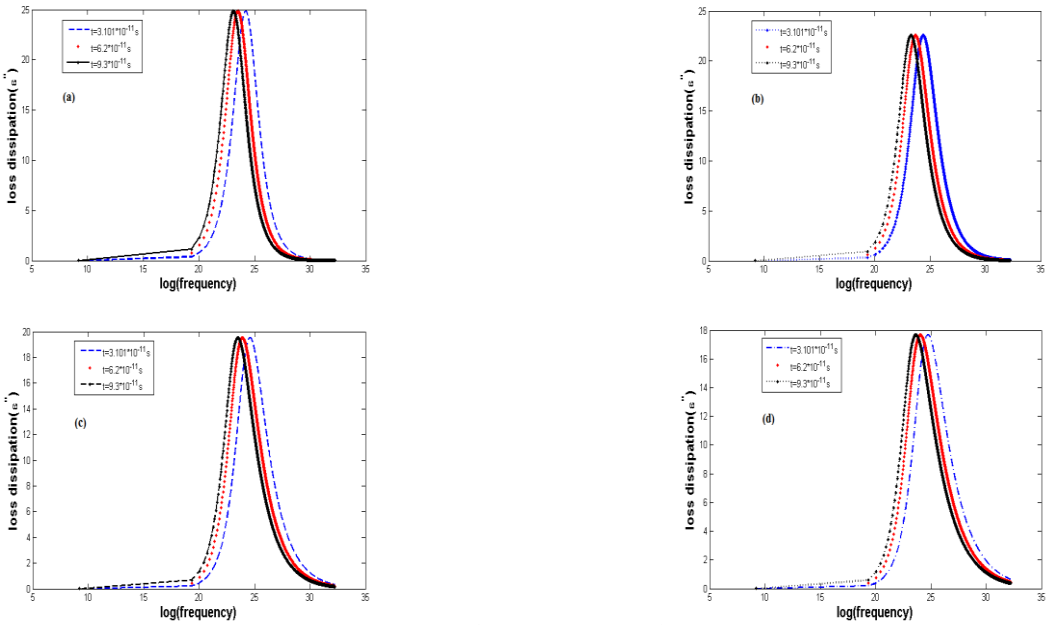

Fig.6: Evolution of the intrinsic power dissipation of the solution 2 $(\varepsilon_0 = 104\,;\ \varepsilon_\infty = 54)$ for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

Tables 5 to 8 present the determination of the hematocrit level of the solution 2 $(\varepsilon_0 = 104\,;\ \varepsilon_\infty = 54)$, for three different relaxation times by varying the dispersion coefficient β .

Table 5: Parameter values $(\tau\,;\ \omega_0\ ;\ Q)$ for $\beta = 0.987$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}101.10^{-11}$	24,8564	$3{,}2500.10^{10}$	$6{,}6000.10^{10}$	49,24%
$6{,}2.10^{-11}$	24,8564	$1{,}6250.10^{10}$	$3{,}3250.10^{10}$	48,87%
$9{,}3.10^{-11}$	24,8551	$1{,}0750.10^{10}$	$2{,}2250.10^{10}$	48,31%

Table 6: Parameter values $(\tau\,;\ \omega_0\ ;\ Q)$ for $\beta = 0.8$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}101.10^{-11}$	22,5679	$3{,}8500.10^{10}$	$9{,}0250.10^{10}$	42,65%
$6{,}2.10^{-11}$	22,5679	$1{,}9250.10^{10}$	$4{,}5250.10^{10}$	42,54%
$9{,}3.10^{-11}$	22,5677	$1{,}2750.10^{10}$	$3{,}0250.10^{10}$	42,14%

Table 7: Parameter values $(\tau\,;\ \omega_0\ ;\ Q)$ for $\beta = 0.6$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}101.10^{-11}$	19,5233	$4{,}8250.10^{10}$	$14{,}3750.10^{10}$	33,56%
$6{,}2.10^{-11}$	19,5231	$2{,}4250.10^{10}$	$7{,}2000.10^{10}$	33,68%
$9{,}3.10^{-11}$	19,5230	$1{,}6000.10^{10}$	$4{,}8250.10^{10}$	33,16%

Table 8: Parameter values $(\tau\,;\ \omega_0\ ;\ Q)$ for $\beta = 0.5$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}101.10^{-11}$	17,6776	$5{,}5750.10^{10}$	$19{,}8500.10^{10}$	28,08%
$6{,}2.10^{-11}$	17,6776	$2{,}8000.10^{10}$	$9{,}9250.10^{10}$	28,21%
$9{,}3.10^{-11}$	17,6774	$1{,}8500.10^{10}$	$6{,}6250.10^{10}$	27,92%

3.1.3. Numerical models: solution 3 ($\varepsilon_0 = 99\,;\ \varepsilon_\infty = 52$)

Figs. 7 and 8 respectively shows the curves of the real permittivity and the intrinsic power dissipation of deoxy-hemoglobin S (solution 3) for relaxation times $3{,}11.10^{-12}\,s\,;\ 6{,}21.10^{-12}\,s\ ;\ 9{,}31.10^{-12}\,s$ by varying the dispersion coefficient β .

Fig.7: Real permittivity of solution 3 $(\varepsilon_0 = 99\,;\, \varepsilon_\infty = 52)$ for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

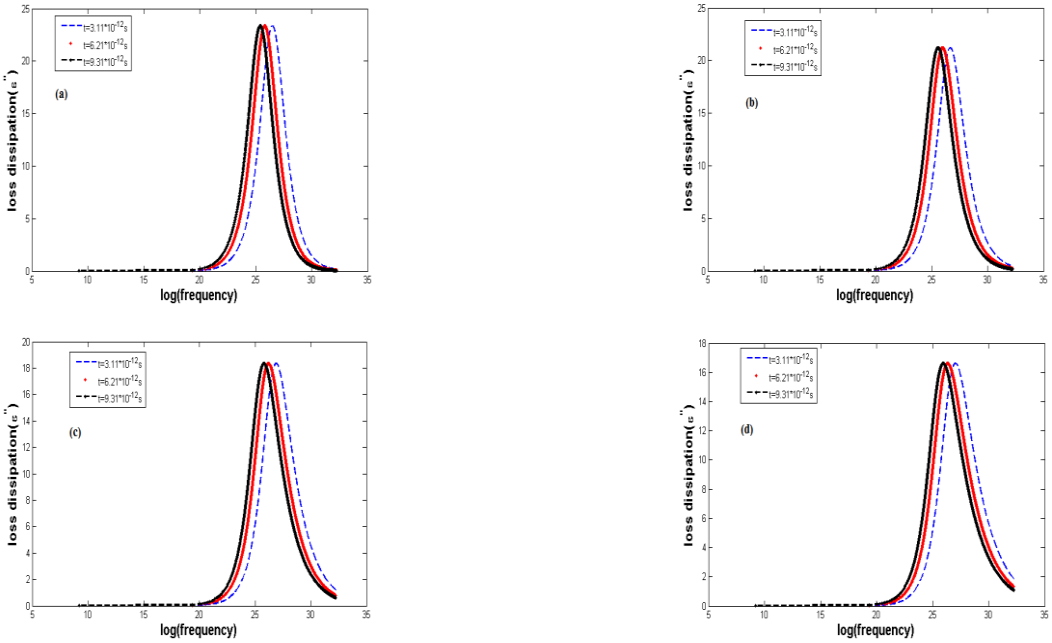

Fig. 8: Evolution of the intrinsic power dissipation of the solution 3 $(\varepsilon_0 = 99\,;\, \varepsilon_\infty = 52)$ for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

Tables 9 to 12 present the determination of the hematocrit level of the solution 3 $(\varepsilon_0 = 99\,;\, \varepsilon_\infty = 52)$, for three different relaxation times by varying the dispersion coefficient β .

Table 9: Parameter values $(\tau\,;\, \omega_0\;;\, Q)$ for $\beta = 0.987$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}11.10^{-12}$	23,3651	$3{,}2475.10^{11}$	$6{,}5550.10^{11}$	49,54%
$6{,}21.10^{-12}$	23,3651	$1{,}6275.10^{11}$	$3{,}2850.10^{11}$	49,54%
$9{,}31.10^{-12}$	23,3651	$1{,}0850.10^{11}$	$2{,}1900.10^{11}$	49,54%

Table 10: Parameter values $(\tau\,;\, \omega_0\;;\, Q)$ for $\beta = 0.8$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}11.10^{-12}$	21,2138	$3{,}8325.10^{11}$	$8{,}9600.10^{11}$	42,77%
$6{,}21.10^{-12}$	21,2138	$1{,}9200.10^{11}$	$4{,}4900.10^{11}$	42,76%
$9{,}31.10^{-12}$	21,2138	$1{,}2800.10^{11}$	$2{,}9950.10^{11}$	42,73%

Table 11: Parameter values $(\tau\,;\, \omega_0\;;\, Q)$ for $\beta = 0.6$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}11.10^{-12}$	18,3519	$4{,}8125.10^{11}$	$14{,}3020.10^{11}$	33,64%
$6{,}21.10^{-12}$	18,3519	$2{,}4100.10^{11}$	$7{,}1920.10^{11}$	33,50%
$9{,}31.10^{-12}$	18,3519	$1{,}6075.10^{11}$	$4{,}7800.10^{11}$	33,62%

Table 12: Parameter values $(\tau\,;\, \omega_0\;;\, Q)$ for $\beta = 0.5$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$3{,}11.10^{-12}$	16,6170	$5{,}5700.10^{11}$	$19{,}7570.10^{11}$	28,19%
$6{,}21.10^{-12}$	16,6170	$2{,}7900.10^{11}$	$9{,}8950.10^{11}$	28,19%
$9{,}31.10^{-12}$	16,6170	$1{,}8600.10^{11}$	$6{,}6025.10^{11}$	28,17%

3.1.4. Numerical models: solution 4($\varepsilon_0 = 92$; $\varepsilon_\infty = 47$)

Figs. 9 and 10 respectively shows the curves of the real permittivity and the intrinsic power dissipation of deoxy-hemoglobin S (solution 4) for relaxation times $2{,}167.10^{-13}s$; $4{,}233.10^{-13}s$; $6{,}3.10^{-13}s$ by varying the dispersion coefficient β .

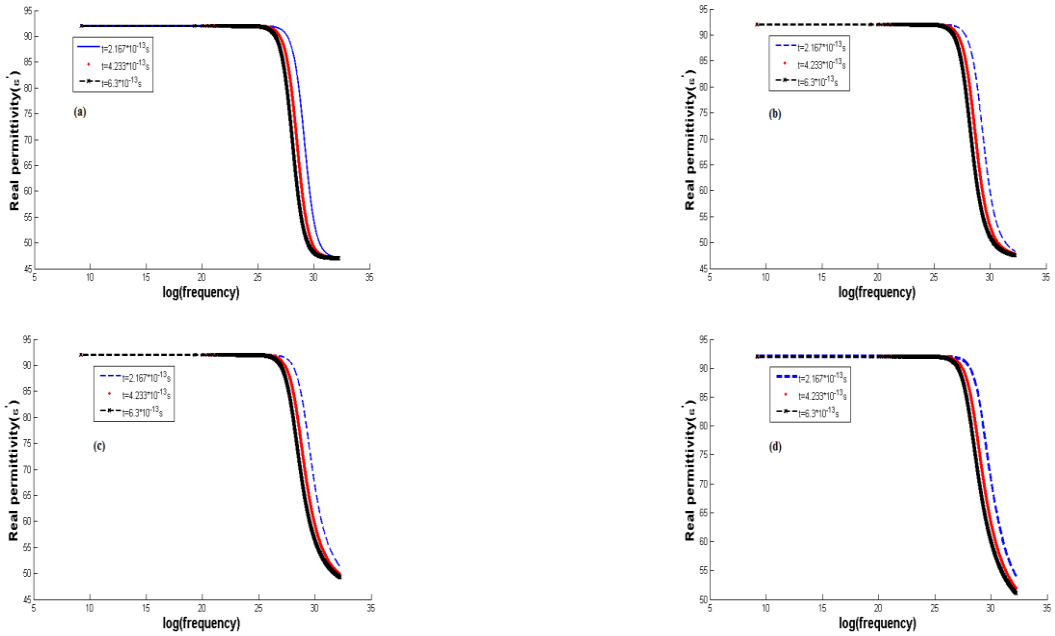

Fig. 9: Real permittivity of solution 4 $(\varepsilon_0 = 92\ ;\ \varepsilon_\infty = 47)$ for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

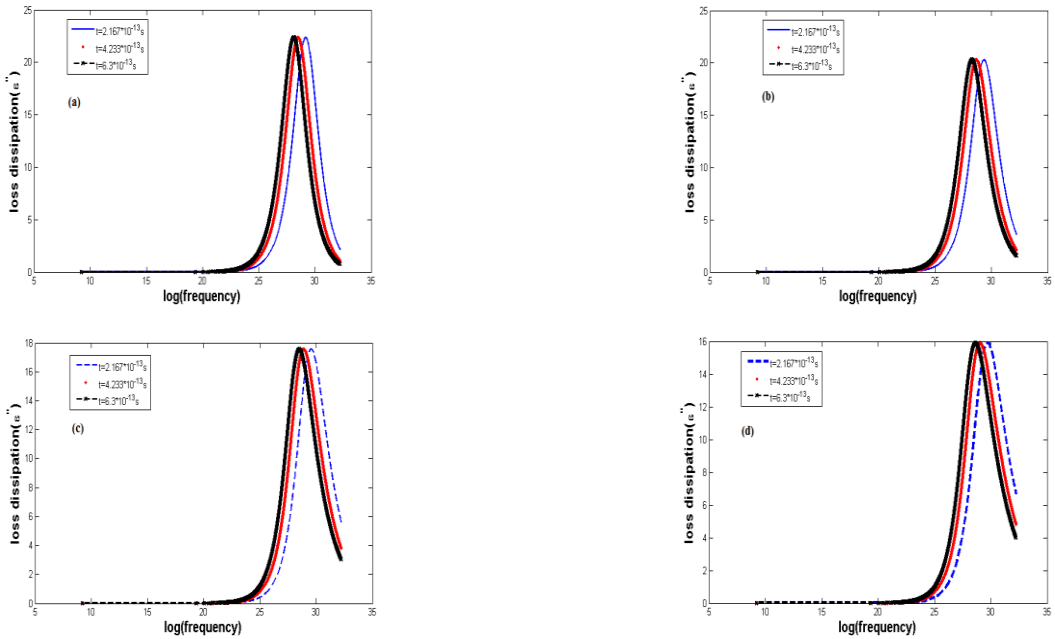

Fig. 10: Evolution of the intrinsic power dissipation of the solution 4 $(\varepsilon_0 = 92\ ;\ \varepsilon_\infty = 47)$ for $\beta = 0{,}987$ (a); $\beta = 0{,}8$ (b); $\beta = 0{,}6$ (c); $\beta = 0{,}5$ (d).

Tables 13 to 16 present the determination of the hematocrit level of the solution 4 $(\varepsilon_0 = 92\ ;\ \varepsilon_\infty = 47)$, for three different relaxation times by varying the dispersion coefficient β .

Table 13: Parameter values $(\tau\ ;\ \omega_0\ ;\ Q)$ for $\beta = 0.987$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$2{,}167.10^{-13}$	22,3708	$4{,}6630.10^{12}$	$9{,}4050.10^{12}$	49,58%
$4{,}233.10^{-13}$	22,3708	$2{,}3865.10^{12}$	$4{,}8140.10^{12}$	49,57%
$6{,}3.10^{-13}$	22,3708	$16038{,}.10^{12}$	$3{,}2350.10^{12}$	49,57%

Table 14: Parameter values $(\tau ; \omega_0 ; Q)$ for $\beta = 0.8$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$2{,}167.10^{-13}$	20,3111	$5{,}5005.10^{12}$	$12{,}8580.10^{12}$	42,77%
$4{,}233.10^{-13}$	20,3111	$2{,}8153.10^{12}$	$6{,}5815.10^{12}$	42,77%
$6{,}3.10^{-13}$	20,3111	$1{,}8918.10^{12}$	$4{,}4225.10^{12}$	42,77%

Table 15: Parameter values $(\tau ; \omega_0 ; Q)$ for $\beta = 0.6$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$2{,}167.10^{-13}$	17,5709	$6{,}9075.10^{12}$	$20{,}5260.10^{12}$	33,65%
$4{,}233.10^{-13}$	17,5709	$3{,}5353.10^{12}$	$10{,}5050.10^{12}$	33,65%
$6{,}3.10^{-13}$	17,5709	$2{,}3755.10^{12}$	$7{,}0545.10^{12}$	33,67%

Table 16: Parameter values $(\tau ; \omega_0 ; Q)$ for $\beta = 0.5$

Relaxation time τ in (s)	Powers atresonance	Frequencies atresonance in (Hz)	Frequency bandwidths $\Delta\omega$ in (Hz)	Hematocrit level
$2{,}167.10^{-13}$	15,9099	$7{,}9940.10^{12}$	$28{,}2590.10^{12}$	28,28%
$4{,}233.10^{-13}$	15,9099	$4{,}0915.10^{12}$	$14{,}5510.10^{12}$	28,11%
$6{,}3.10^{-13}$	15,9099	$2{,}7493.10^{12}$	$9{,}7860.10^{12}$	28,09%

3.2. Interpretation and validity of the Davidson-Cole model

The nature of sickle cell disease has been clarified in molecular terms. Indeed, when frequency ω of the erythrocyte solution is between 10^4 Hz and 10^8 Hz, all deoxy-hemoglobin S molecules are in the solution phase (disaggregated state). During the first phase of the aggregation process called the lag phase, the stresses increase and the loss of energy is observed when the frequency ω is greater than 10^8 Hz. This transformation generates an energy loss of exponentially. The molecules of deoxy-hemoglobin S clump together them, the molecular space is gradually reduced, giving rise to a steric effect increasing (polymerization mechanism) up to the critical frequency ω_0 called frequency of resonance. The rate of amortization of red blood cells is high, i.e. the hematocrit level is low and this is explained by the fact that the energy loss of the hemoglobin is large. The exponential growth of this energy ends up tearing the membrane of red blood cells, which is then destroyed. Fixing and transport respiratory gases in the blood become complicated. On the other hand, the subjects said SS homozygotes develop chronic hemolytic anemia and suffer from seizures often painful vaso-occlusives that can affect all organs. At the end of the second phase of the transformation process called the phase of relaxation, stresses decrease, energy loss drops exponentially up to frequency $\omega = 10^{14}$, all molecules of deoxy-hemoglobin S are in the polymer or solid phase (completely aggregated state) (Fig.4, Fig.6, Fig.8 and Fig.10). So the permittivity dielectric of the solution decreases considerably and reaches its minimum value (Fig.3, Fig.5, Fig.7 and Fig.9). Moussiliou et al. found similar results [5]. The maximum value of the real permittivity remains unchanged whatever degree dispersion of solutions of deoxy-hemoglobin S (Fig.3, Fig.5, Fig.7 and Fig.9). On the other hand the value of the intrinsic power dissipation at resonance as well as hematocrit levels decrease dramatically as the dispersion coefficient decreases (Fig.4, Fig.6, Fig.8 and Fig.10). Figs.11 presents respectively the intrinsic energy dissipation for the relaxation time $\tau = 3{,}4.10^{-13}$s (Fig.11a) and $\tau = 10^{-12}$s (Fig.11b). We take note that the frequency ω_0 at resonance varies according to the relaxation time and the coefficient of dispersion (Fig.11). In addition, the hematocrit level is one of the determining factors of the sickle cell nature of the red blood cells; we note that the calculated hematocrit levels are low. In addition, an important point is that the hematocrit level is independent of characteristic relaxation times but rather depends on coefficient of dispersion (table.1 to 16). The solutions of deoxy-hemoglobin S are therefore characterized by a unique time of relaxation. This result is in agreement with Debye's theory whose fundamental hypothesis is the unique relaxation time [4]. The coefficient dispersion is the most influential parameter in the aggregation dynamics of the deoxy-hemoglobin S. From these results, it appears that the relaxation spectra developed are good interpretation results that can adjust the experimental data of the deoxy-hemoglobin S solutions to check for blood abnormalities and diagnose certain abnormalities related to sickle cell disease.

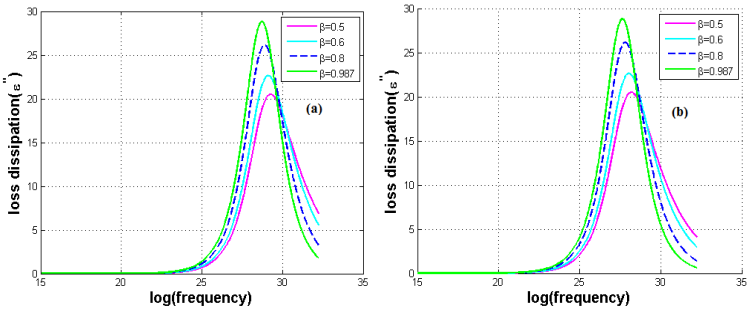

Fig. 11: Intrinsic power dissipation curves varying the dispersion coefficient β for relaxation times $\tau = 3{,}4.10^{-13}s$ (Fig. a) and $\tau = 10^{-12}s$ (Fig. b).

4. Conclusion

Sickle cell disease has been identified by determining hematocrit levels of four solutions of deoxy-hemoglobin S. We have shown that the coefficient dispersion is the most influential parameter in the aggregation of erythrocytes. We also showed a unique relaxation time of the hemoglobin gel-solution S. The behavior of deoxy-hemoglobin S is well interpreted by developing the dielectric permittivity as a function of the relaxation frequency. This development also provides that the hematocrit levels are decisive and explain very well chronic hemolytic anemia, the origin of which may be sickle cell disease. This study suggests that the numerical models developed are good interpretive models which can adjust the experimental data of solutions of deoxy-hemoglobin S.

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