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Phagocytic properties and organelle motility of pulmonary macrophages from smokers and nonsmokers estimated *in vitro* by magnetometric means

V. Im Hof*, M. Klauser**, P. Gehr**

Phagocytic properties and organelle motility of pulmonary macrophages from smokers and nonsmokers estimated in vitro *by magnetometric means. V. Im Hof, M. Klauser, P. Gehr.*

ABSTRACT: Monolayer cultures of pulmonary macrophages (PM) from 11 smokers (S) and 9 nonsmokers (NS) were incubated with submicrometric ferromagnetic Fe_3O_4 particles for one hour. After magnetization by an externally applied pulse magnetic field, the aligned Fe_3O_4 particles, located in the phagosomes and secondary lysosomes of the PM, emanated a remanent magnetic field which decayed with time. The initial field strength, B_0, which is proportional to the amount of phagocytosed Fe_3O_4 particles, was 13.24 nT (SE 0.79 nT) in S and 11.74 nT (SE 1.39 nT) in NS. Ten nT correspond to roughly 4 μg Fe_3O_4. The initial rate of decay of the remanent field (during the first 60 s after magnetization), λ_0, is proportional to the rate of particle misalignment. Therefore, λ_0, is hypothesized to be an estimate of organelle motility. It was found to be the same in S and in NS, being $3.14\times10^{-3}\cdot s^{-1}$ (SE $0.18\times10^{-3}\cdot s^{-1}$) in S and $3.17\times10^{-3}\cdot s^{-1}$) (SE $0.22\times10^{-3}\cdot s^{-1}$) in NS. These results suggest that, *in vitro*, there is no difference in phagocytic activity and organelle motility in PM from S and NS. *Eur Respir J., 1990, 3, 157–162.*

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The pulmonary macrophages (PM) with their locomotory, phagocytic and lytic potentials are an essential line of defence in the lungs. By phagocytosing bacteria, viruses, other organic and inorganic inhaled particles, red cells, cell debris and surfactant, the PM keep the airway and alveolar surfaces clean and sterile [1]. The same cells, however, also participate in the pathogenesis of several lung diseases. Secreting lysosomal proteolytic enzymes capable of degrading connective tissue (collagenase, elastase), the PM are involved in the development of emphysema [2]. Particles which have been phagocytosed by the PM are thought to stimulate the release of factors which cause fibroblast migration and replication as well as the local production of collagen by fibroblasts, a basic mechanism of fibrosis [2, 3].

Inhalation of large amounts of particles causes the number of PM to increase. In chronic smokers the number of alveolar macrophages has been found to be increased two to thirteenfold as compared to nonsmokers [4]. Moreover, the cells from smokers have more and larger secondary lysosomes [2, 5, 6] and appear to be more active than those in nonsmokers, if we consider both surface morphology and cell function [7, 8]. Thus, we expected PM from smokers to have greater phagocytic activity and organelle motility than PM from nonsmokers. To test this hypothesis, we harvested PM from smokers and nonsmokers by bronchoalveolar lavage (BAL), and analysed them *in vitro* by magnetometric means. Magnetometry utilizes measurements of the remanent magnetic fields produced by ferromagnetic particles (Fe_3O_4) after magnetization. Fe_3O_4 particles phagocytosed by macrophages are located in phagosomes and secondary lysosomes. The particles can, therefore, be used as probes for non-invasive studies of organelle motility *in vivo* [9–13] and *in vitro* [14–18].

Methods

Study population

Adult volunteers were recruited for the study. The criteria for inclusion were: 1) age 20–40 yrs; 2) current good health; 3) complete absence of any history of pulmonary disease; 4) no disease, especially no viral infection, for ten weeks prior to the investigation; 5) no drugs at the present time; 6) normal physical examination of heart and lungs; and 7) smokers: 20 cigarettes per day for at least 5 yrs prior to the study (≥5 pack-years); nonsmokers: never smoked. Since no correlation has been reported between BAL results (percentage of volume recovery, number of harvested cells) and any lung volume [17], we performed no pulmonary function tests.

Performance of BAL in these subjects was approved by the Committee for the Use of Human Subjects at the University Hospital (University of Berne, Switzerland) and written informed consent was obtained from all subjects prior to the study. Twenty three subjects underwent BAL. In three cases no magnetometric measurements were performed for the following reasons: 1) fungal growth after 24 h in the cell culture dishes in two cases; and 2) an unexplained high percentage (>50%) of lymphocytes in the recovered cells of one subject. Therefore, we present the results of 20 lavages only, namely 11 smokers (subjects no. 1–11; 3 women and 8 men) and 9 nonsmokers (subjects no. 12–20; 3 women and 6 men). The average age of smokers and nonsmokers was 27.3 yrs (SE 1.2 yrs) and 27.8 yrs (SE 1.3 yrs), respectively (table 1). Since no gender-related differences in BAL data have been shown [17], we included both sexes in our study and did not investigate them separately.

Table 1. – Lavage data

		Sex f/m	Age yrs	HbCO %	Volume recovery ml	Cell yield millions
Smokers	Mean value	3/8	27.3	4.8	191.3	52.4
	SE		1.2	0.6	5.6	12.1
Nonsmokers	Mean value	3/6	27.8	1.2	222.3	15.1
	SE		1.3	0.1	7.3	1.2
t-test	2p		>0.7	<0.001	<0.005	<0.02

Bronchoalveolar lavage (BAL)

Subjects were instructed not to eat or drink for 6 h prior to the bronchoscopy. Smoking was allowed. All BAL were performed between 8:00 and 9:00 a.m. by one of the investigators. In order to get an estimation of the actual smoking habits, 5 ml of blood was drawn by means of an antecubital intravenous puncture, and the carboxyhaemoglobin (HbCO) was measured in a blood gas-analyser (CO-Oxymeter IL-282, Instrumentation Laboratory, Lexington, MA, USA). Following premedication with 10 mg of morphine and 0.5 mg of atropine by intramuscular injection, the subjects underwent local anaesthesia (pharynx, larynx, trachea and main bronchi) with 1% lidocaine. No lidocaine was used in the lobar and segmental/subsegmental bronchi. The subjects lay in the supine position throughout the subsequent lavage procedure. A flexible fibreoptic bronchoscope (Olympus BF P-10) was introduced perorally and the tip of the instrument (outer diameter: 4.8 mm) was gently wedged in a segmental/subsegmental bronchus of the right middle lobe. The lavage procedure consisted of six washes. A wash was carried out by: 1) instillation of 50 ml of sterile isotonic 0.9% sodium chloride solution (free of divalent cations) at 37°C; and 2) the immediate aspiration of the fluid. The entire lavage procedure took approximately 10 min. In order to remove mucous strands, the recovered fluid, kept in a siliconized glass suction trap, was filtered through one layer of sterile cotton gauze into a polystyrene tube. Subsequently, the recovered volume was estimated, the cells were counted (Neubauer chamber) and their viability was tested (trypan blue exclusion test).

Cytomagnetometry

The magnetometric method, as described earlier [18], was slightly modified. The lavage fluid from each subject was centrifuged at 200 g for 6 min. The supernatant was discarded and the resulting cell pellet was resuspended in culture medium (approximately 10^6 cells·ml^{-1} medium). The culture medium used was Eagle's minimal essential medium (MEM: H_2CO_3 0.85 g·l^{-1}, without glutamine) supplemented with 10% newborn calf serum, gentamycin (50 µg·ml^{-1}) and mycostatin (50 µg·ml^{-1}).

Eight to ten sterile Petri dishes per subject, each containing 2.5 ml of cell suspension, were incubated for 1 h at 37°C and 5% CO_2. Then, non-adherent cells (approximately 50% of the cells) and cell debris were thoroughly rinsed off and new culture medium (MEM) was added. The adherent cells (non-confluent monolayer, approximately 2–3×10^3 PM·mm^{-2}) were incubated for 24 h at 37°C and 5% CO_2. Afterwards the cell cultures were washed again with MEM. One droplet (0.1–0.15 ml) of a solution of unopsonized submicrometric Fe_3O_4 particles (magnetite, Pfizer, NY, type MO-7029; average particle diameter: 0.3 µm) was then added to the monolayer cultures. The magnetite suspension (1.33 mg Fe_3O_4·ml^{-1} of 0.9% NaCl), was homogenized by sonification immediately before being added to the cultures. After 1 h of incubation the cell medium was changed again, thus discarding the non-phagocytosed, free Fe_3O_4 particles (approximately 50% of the added particles). A single culture dish was then placed in the temperature controlled well. Applying a pulse magnetic field of 0.5–1 T (Tesla; 1 T=1 WB·m^{-2}) for 1–2 ms to this dish, the Fe_3O_4 particles were magnetized, *i.e.* became magnetic dipoles and aligned according to the field lines of the pulse field. The dish was then instantaneously moved into a cylindrical moly-permalloy shield to reduce external magnetic field interference and the culture was presented to a fluxgate magnetometer probe (Förster, Magnetoscope 1.067, D-7410 Reutlingen, Germany). In order to reduce noise,

this probe was mounted in gradiometer mode. The remanent magnetic field, collectively produced by the aligned Fe_3O_4 particles in the cells of the monolayer culture (the magnetometer probe measures the field below about 80,000 cells), was recorded every second by a computer (DEC, PDP-11/13) for 60 s after magnetization and simultaneously plotted by a chart recorder. The data were subsequently transferred to a computer (Apple, Macintosh II) for calculations, statistical analyses (t-test) and printing of tables and graphs. Every cell culture (*i.e.* 8–10 Petri dishes on average per subject) was measured identically. Consequently, the results of the magnetometric measurements per subject always represent the mean values of these 8–10 measurements. The viability of the cells has been tested again after the magnetometric measurements.

The remanent magnetic field of the monolayer culture begins to decay (relaxation) as soon as the magnetizing field is removed. Since it takes a few seconds to move the culture dish to the shielded magnetometer probe, the initial magnetic field strength, B_0, which is proportional to the amount of Fe_3O_4 particles present [11, 14], cannot be measured. Therefore, B_0 has to be calculated using the recorded remanent field data of the first 60 s according to the single exponential equation:

$$B_{(t)} = B_0 \times e^{-\lambda_0 \cdot (t)} \qquad (1)$$

where $B_{(t)}$ is the magnetic field strength in Nanotesla (nT) at time t after magnetization [18]. The initial relaxation rate, λ_0 (unit: s^{-1}), is the exponential decay coefficient of the remanent field, calculated from the values during the first 60 s after magnetization. Strictly speaking, the relaxation curve of the first 60 s is not a clear single exponential curve. In order to obtain a more accurate value for the initial strength of the remanent field, we therefore calculated B_0' which is the field strength with regard to the first 10 s of relaxation only. The decay during this time period is strictly single exponential. B_0' is calculated according to equation 1 and is used for normalizing the relaxation curves (table 2 and fig. 1).

The relaxation phenomenon is a reflection of the misalignment of the magnetic particles. It is due to random progressive rotation of the particle containing phagosomes and secondary lysosomes, probably caused by forces exerted on them by contractile elements of the cytoskeleton [11, 14]. It is not caused by passive processes such as Brownian movement or elastic recoil [15]. λ_0 is, therefore, considered to be an estimate of organelle motility [11, 14].

Results

Bronchoalveolar lavage (BAL)

The lavage data are summarized in table 1. The total lavage fluid recovered from smokers was significantly lower than that from nonsmokers, being 191.3 ml (SE 5.6 ml) in smokers and 222.3 ml (SE 7.3 ml) in nonsmokers. Over 90% of the recovered cells were found to be PM, the rest being neutrophils, lymphocytes and eosinophils. The viability of the PM was >85% in all subjects. There was no difference between smokers and nonsmokers. The total yield of PM (table 1) was 52.4×10^6 (SE 12.1×10^6) in smokers and 15.1×10^6 (SE 1.2×10^6) in nonsmokers. PM from smokers showed a greatly expanded lysosomal compartment as compared to that of PM from nonsmokers (fig. 2). The carboxyhaemoglobin (HbCO) concentration was significantly higher in smokers (4.8%; SD 2.0%) than in nonsmokers (1.2%; SD 0.2%). Normal nonsmoker values in our laboratory are ≤2%.

Cytomagnetometry

Within 1 h of incubation the PM phagocytosed most of the submicrometric Fe_3O_4 particles (fig. 2). The initial remanent field strength, B_0 (table 2), was 13.24 nT (SE 0.79 nT) in the smokers and 11.74 nT (SE 1.39 nT) in the nonsmokers. Thus, the cells of both groups ingested

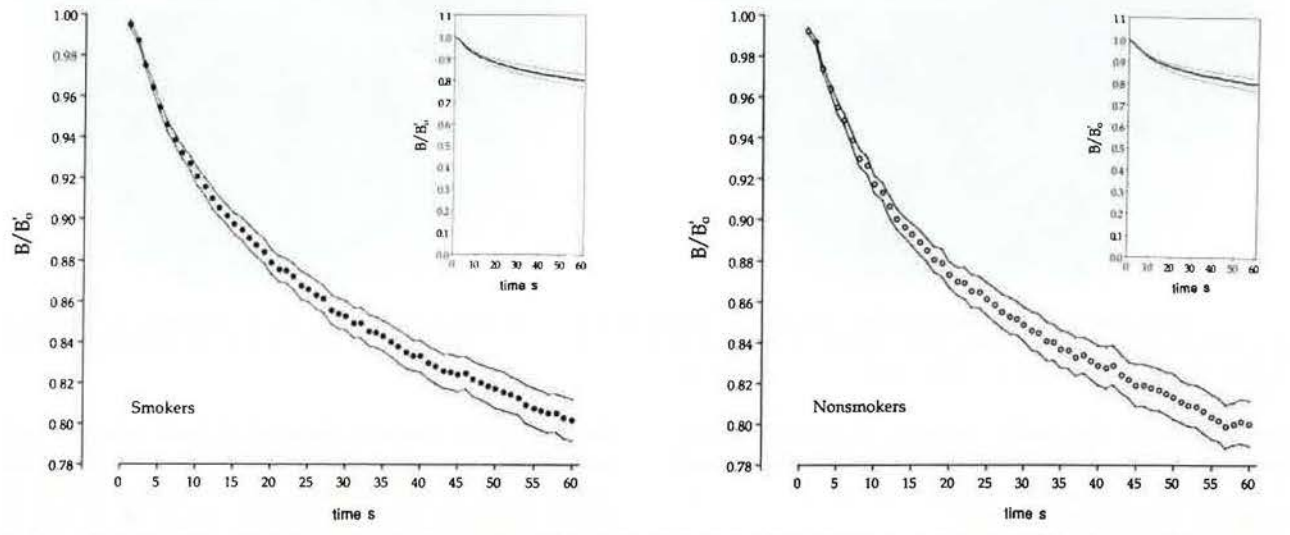

Fig. 1. – Normalized relaxation curves (mean values, range of SE) of smokers (left) and nonsmokers (right). Insets: full scale for B/B_0'.

Table 2. – Cytomagnetometric data

	no.	B_0 nT	B_0' nT	B_{60} nT	λ_0 $\times 10^{-3} \cdot s^{-1}$
Smokers	1	14.13	14.66	12.56	2.35
	2	16.89	17.78	14.28	3.15
	3	14.84	15.54	12.72	2.88
	4	8.79	9.30	7.10	3.87
	5	10.29	11.03	8.16	4.13
	6	9.23	9.72	7.93	2.89
	7	15.13	16.19	12.20	3.96
	8	14.13	14.85	12.21	2.81
	9	14.61	15.39	12.62	2.75
	10	13.58	14.21	11.58	2.90
	11	13.98	14.69	11.95	2.85
	Mean	13.24	13.94	11.21	3.14
	SE	0.79	0.82	0.71	0.18
Nonsmokers	12	12.89	13.50	10.96	2.95
	13	10.80	11.45	9.31	2.79
	14	18.00	18.96	15.30	3.07
	15	8.48	8.94	7.64	2.08
	16	6.02	6.36	4.98	3.76
	17	8.34	9.00	5.74	4.42
	18	11.18	11.95	9.39	3.46
	19	18.31	19.25	15.47	3.13
	20	11.64	12.27	9.94	2.89
	Mean	11.74	12.41	9.86	3.17
	SE	1.39	1.45	1.23	0.22
t-test	2p	>0.3	>0.3	>0.3	>0.9

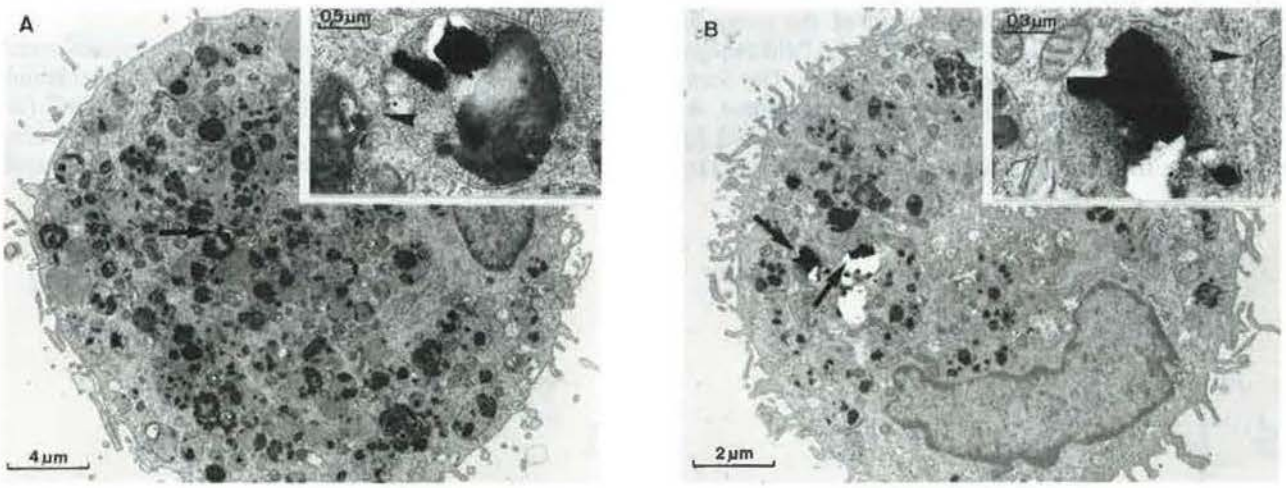

Fig. 2. – Human pulmonary macrophages (PM), recovered by bronchoalveolar lavage from a smoker (A) and a nonsmoker (B), containing phagocytosed Fe_3O_4 particles (arrows). Note increased density or organelles in PM from the smoker. Insets: Fe_3O_4 particles in secondary lysosomes in close association with filaments (arrow heads) of the cytoskeleton.

approximately the same amount of ferromagnetic particles, namely about 60 µg $Fe_3O_4 \cdot 10^{-6}$ cells. The amount 4 µg Fe_3O_4 corresponds roughly to 10 nT (see also Methods: Cytomagnetometry).

The remanent magnetic field strength measured below the monolayer cultures, decayed in both smokers and nonsmokers in a very similar manner (fig. 1). The mean values of λ_0 of both groups (table 2) were found to be nearly the same, being $3.14 \times 10^{-3} \cdot s^{-1}$ (SE $0.18 \times 10^{-3} \cdot s^{-1}$) in smokers and $3.17 \times 10^{-3} \cdot s^{-1}$ (SE $0.22 \times 10^{-3} \cdot s^{-1}$) in

nonsmokers. The viability tested after the measurements was still good, and there was no difference between cells from smokers and nonsmokers.

Discussion

Two distinct populations of human PM were obtained by BAL: 1) PM from healthy nonsmokers; and 2) PM from healthy smokers chronically exposed to a cigarette smoke burden. The HbCO concentration of the smokers was, as expected, significantly higher than that of the nonsmokers. Our lavage data (*i.e.* recovered fluid volume per lavage, PM recovery per lavage, viability of the recovered PM, inter-individual variability of the lavage data) were all in accordance with data given in the literature [17, 20–22]. With the lavage procedure used in this study (six washes of 50 ml NaCl per lavage) we achieved a large recovery of PM in nonsmokers, allowing enough cytomagnetometric measurements per subject. The reason for the significant difference in the total volume recovered between smokers and nonsmokers could perhaps be the narrowing of the small airways caused by an inflammatory oedema as a consequence of chronic bronchitis in smokers.

Cytomagnetometry is a useful approach for estimating the phagocytic properties and organelle motility of cells which have phagocytosed magnetic particles [9, 10, 15, 16, 18]. The initial magnetic field strength, B_0, is proportional to the amout of phagocytosed Fe_3O_4 particles. The relaxation phenomenon (decay of the remanent field) is caused by the misalignment of the magnetic particles due to random progressive rotations of the particle containing phagosomes and secondary lysosomes. These rotations are attributed to forces exerted on the organelles by contractile elements of the cytoskeleton, probably by actin-myosin interactions [2, 11, 14, 18].

Despite the burden of phagosomes and/or secondary lysosomes we observed to be increased in number and volume, due to the increased amount of foreign material phagocytosed *in situ* (fig. 2), B_0 of PM from smokers did not differ significantly from that of nonsmokers (table 2). That is, PM from smokers were able to continue phagocytosis *in vitro* at the same rate as PM from nonsmokers. These findings are similar to data presented in earlier studies [6, 7, 23, 24], where PM from nonsmokers did not reveal a higher phagocytic capacity than PM from tobacco or marijuana smokers.

Suprisingly, no significant difference was found in the relaxation ratio, λ_0, between smokers and nonsmokers (fig. 1). We conclude that the PM of both groups exhibited *in vitro*, *i.e.* at least 26 h after BAL, the same organelle motility. The increase in number, size, and density of the phagosomes in PM from smokers seems not to impair the movements of the organelles caused by the contractile elements of the cytoskeleton.

The results of our magnetometric measurements with PM from nonsmokers are similar to those recently reported by VALBERG *et al.* [25] who also investigated the organelle motility of PM from both smokers and nonsmokers *in vitro*. λ_0 of PM from nonsmokers was $3.5\times10^{-3}\cdot s^{-1}$ (SE $0.62\times10^{-3}\cdot s^{-1}$) in the study of VALBERG *et al.* as compared to $3.17\times10^{-3}\cdot s^{-1}$ (SE $0.22\times10^{-3}\cdot s^{-1}$) in our study. Disagreement exists, however, in regard to PM from smokers: λ_0 of PM from smokers was found to be $8.5\times10^{-3}\cdot s^{-1}$ (SE $1.3\times10^{-3}\cdot s^{-1}$) by VALBERG and co-workers as compared to $3.14\times10^{-3}\cdot s^{-1}$ (SE $0.18\times10^{-3}\cdot s^{-1}$) in our study. The following considerations may account for the differences: 1) VALBERG *et al.* did not give age or smoking history of the lavaged subjects; 2) their cytomagnetometric measurements were performed with pelleted cells; and 3) they incubated their harvested PM with Fe_3O_4 particles "overnight".

In a very recent study, YAMAYA *et al.* [13] analysed the influence of smoking on the cytoplasmic motility of PM *in vivo* in dogs by cytomagnetometric means. They showed that the relaxation rate λ_0, increased during acute cigarette smoke inhalation but returned to pre-exposure values within 15 min. This suggests that cigarette smoke can cause a transitory increase of the cytoplasmic motility of the alveolar macrophages. In our *in vitro* experiments, the PM from smokers were analysed at least 26 h after the last *in vivo* exposure to cigarette smoke. Thus, perhaps, we compared baseline conditions in macrophages from smokers and nonsmokers. It may be, that the *in situ* conditions of our young smokers (average age: 27 yrs, ≥5 pack-years) did not induce a long-term change of the phagocytic activity and cytomotility of the PM.

In summary, our findings provide evidence that PM from young smokers and nonsmokers have, *in vitro*, the same phagocytic properties and the same organelle motility, at least under baseline conditions.

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Propriétés phagocytaires et motilité des organelles des macrophages pulmonaires de fumeurs et de nonfumeurs: estimation in vitro par methode magnétométrique. V. Im Hof, M. Klauser, P. Gehr.

RÉSUMÉ: Des cultures en couche unique de macrophages pulmonaires (PM) provenant de 11 fumeurs et 9 nonfumeurs ont été incubées pendant 1 heure avec des particules submicroniques ferromagnétiques de Fe_3O_4. Après magnétisation sous l'effet d'un champ magnétique pulsé d'application extérieure, les particules alignées de Fe_3O_4, situées dans les phagosomes et les lysosomes secondaires du PM, ont émis un champ magnétique rémanant, diminuant avec le temps. La puissance initiale du champ (B_0), proportionelle à la quantité de particules de Fe_3O_4 phagocytées s'élève à 13.24 nT (D.S. 0.79 nT) chez les fumeurs et à 11.74 nT (D.S. 1.39 nT) chez les nonfumeurs. Dix nT correspondent à environ 4 µg de Fe_3O_4. Le taux initial de décroissance du champ rémanent (pendant les 60 s faisant suite à la magnétisation) est proportionnel au taux de mauvais alignement particulaire. C'est la raison pour laquelle on émet l'hypothèse que λ_0 est une estimation de la motilité des organelles. Elle s'avère similaire chez les fumeurs ($3.14\times10^{-3}\cdot s^{-1}$ [SE $0.18\times10^{-3}\cdot s^{-1}$]) et chez les nonfumeurs ($3.17\times10^{-3}\cdot s^{-1}$ [SE $0.22\times10^{-3}\cdot s^{-1}$]). Ces résultats suggèrent qu' il n'y a pas de différences dans l'activité phagocytaire et la motilité des organelles dans les macrophages des fumeurs par rapport à ceux des nonfumeurs.
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