



## Jornadas do **CENTRO DE NEUROCIÊNCIAS** CUF Porto



### **PUBLICAÇÃO CIENTÍFICA: MOTIVAÇÃO E FORMAÇÃO** 09 de abril de 2016 | Auditório do Hospital CUF Porto

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#### **SECRETARIADO INSCRIÇÕES E INFORMAÇÕES**

secretariado.academiacuf@jmellosaude.pt

**08h45** Abertura do secretariado

**09h15** **Sessão de abertura e introdução**  
João Massano | Rui Vaz

**09h30** **Pesquisa bibliográfica – dicas práticas para o mundo contemporâneo**  
Palestrante: Helena Donato  
Moderação: José Lopes Lima

**10h00** **Tipos de artigos científicos e sua estrutura**  
Palestrante: Manuel Gonçalves Pinho  
Moderação: Altamiro da Costa Pereira

**10h30** **Como escrever um artigo científico**  
Palestrante: Margarida Lima  
Moderação: José Pereira Monteiro

**11h00** Coffee break

**11h30** **Escolher autores e revista para publicar; indexação e factores de impacto**  
Palestrante: Adelino Leite Moreira  
Moderação: Elsa Azevedo

**12h00** **Investigação e publicação científica em Medicina Geral e Familiar**  
Palestrante: Raquel Braga  
Moderação: Luís Cavadas

**12h30** **Novas tendências da divulgação científica: ORCID, ResearchGate, LinkedIn, outras redes sociais**  
Palestrante: José Carona Carvalho  
Moderação: José Castanheira

**13h00** Almoço

**14h30** **O trajecto do artigo submetido a uma revista científica e o papel do Editor-Chefe**  
Palestrante: Rui Marinho  
Moderação: João Massano

**15h00** **Análise crítica de um artigo científico**  
Palestrante: António Morais  
Moderação: Paulo Pereira

**15h30** **Educação médica e publicação científica**  
Palestrante: Maria Amélia Ferreira  
Moderação: Rui Vaz

**16h00** **A visão dos alunos de medicina**  
Palestrante: André Fernandes  
Moderação: Rui Coelho

**16h30** **Questões finais da audiência, discussão geral e conclusões**  
Moderação: João Massano | André Fernandes | Rui Vaz

**17h00** Encerramento

## Análise crítica de um artigo científico

## António Morais Revista Portuguesa de Pneumologia

Relevância do objetivo da investigação ?  
(questão formulada)

Mediante todos os dados publicados sobre determinado tema:

- Questão original ?
- Questão que questiona um conceito estabelecido?
- Questão que sugere adicionar algo a um conceito estabelecido ?
- Questão que pretende confirmar uma hipótese previamente colocada ?



revista portuguesa de  
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[www.revportpneumol.org](http://www.revportpneumol.org)



**Como avaliar um artigo**

How to write a scientific paper – and win the game scientists play!

How to write a scientific paper—Writing the methods section

How to write a scientific paper - Searching and Managing Biomedical information

To publish or perish: How to review a manuscript

**Como avaliar um artigo**

Título

Abstract

Introdução

Metodos

Resultados

Discussão

Referencias

Tabelas

Suplementos

Um bom título é curto, informativo e específico

**Effective titles:**

- identify the main issue of your paper
- begin with the subject of your paper
- are accurate, unambiguous, specific, and complete
- do not contain abbreviations
- attract readers

## Abstracts

- ✓ objectivo e racional do estudo (porque é que o estudo foi feito)
- ✓ metodologia (como foi feito)
- ✓ resultados (o que é que foi descoberto)
- ✓ conclusões (o que é que representa)

## THE CHROMOSOME NUMBER OF MAN

By JOE HIN TJIO and ALBERT LEVAN

ESTACION EXPERIMENTAL DE AULA DEI, ZARAGOZA, SPAIN, AND CANCER CHROMOSOME LABORATORY, INSTITUTE OF GENETICS, LUND, SWEDEN



**Background.** It is generally accepted that the chromosome number in humans is 48. But to count chromosomes has been difficult, as they clump and partially cover each other. In this study, cultured cells were treated with solutions that spread the chromosomes and made them easier to count.

**Methods.** Cultured cells from human embryonic lung were treated with both colchicine and hypotonic solution.

**Results.** Among 265 mitoses counted, all but 4 had a chromosome number of 46.

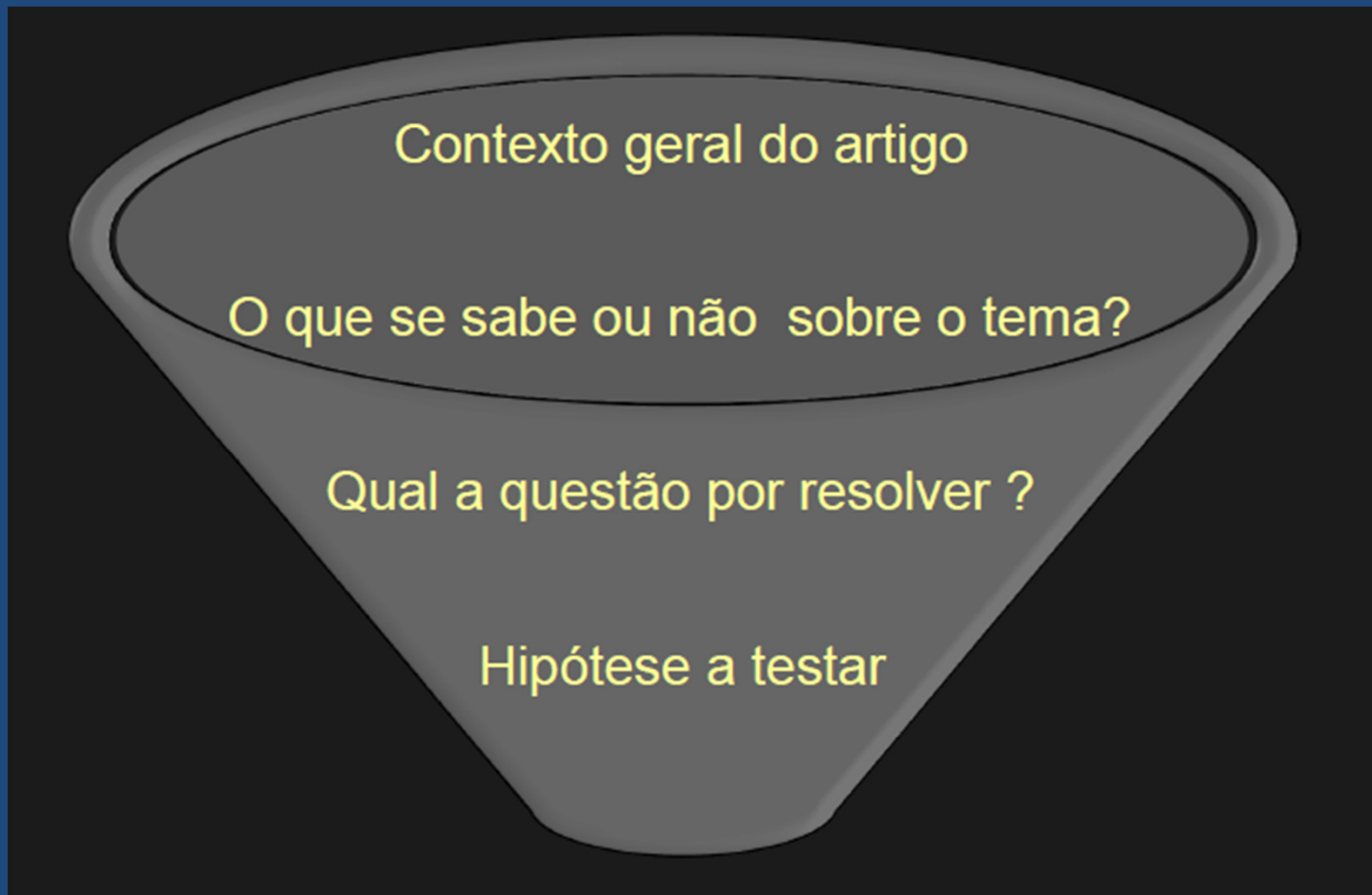
**Conclusion.** The results suggest that the chromosome number in humans is 46, not 48.

A introdução tem dois objectivos:

- Captar o interesse do leitor para o tema do artigo
- Fornecer a informação necessária para entender o artigo

- ✓ Informação sobre o que é sabido
- ✓ O que não é conhecido
- ✓ O foco do trabalho

**Como avaliar um artigo**





Contents lists available at ScienceDirect

Respiratory Investigation

journal homepage: [www.elsevier.com/locate/resinv](http://www.elsevier.com/locate/resinv)



Original article

## All-case post-marketing surveillance of 1371 patients treated with pirfenidone for idiopathic pulmonary fibrosis

Takashi Ogura<sup>a,\*</sup>, Arata Azuma<sup>b</sup>, Yoshikazu Inoue<sup>c</sup>, Hiroyuki Taniguchi<sup>d</sup>, Kingo Chida<sup>e</sup>, Masashi Bando<sup>f</sup>, Yuka Niimi<sup>g</sup>, Shinichi Kakutani<sup>h</sup>, Moritaka Suga<sup>i</sup>, Yukihiko Sugiyama<sup>f</sup>, Shoji Kudoh<sup>l</sup>, Toshihiro Nukiwa<sup>j</sup>

Como avaliar um artigo

### 1. Introduction

Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic lung disease. The median survival of patients with IPF is 2–5 years because of limited treatment options [1–3]. Pirfenidone, an oral drug with anti-fibrotic, anti-inflammatory, and anti-oxidant activities, is the first drug approved for IPF treatment, which was approved in 2008 in Japan, in 2011 in the European Union, and in 2014 in the United States. The clinical efficacy and tolerability of pirfenidone in mild-to-moderate IPF were confirmed in two placebo-controlled, randomized clinical trials in Japan [4,5] and two concurrent multinational randomized trials (the CAPACITY program) [6]. A Cochrane meta-analysis of pirfenidone, including these three key clinical trials, demonstrated that pirfenidone reduced the risk of disease progression by 30% [7]. In addition, the ASCEND trial demonstrated that pirfenidone therapy significantly retarded disease progression [8].

To date, there is little information regarding the tolerability and efficacy in patients with severe IPF, although two small studies have been conducted with patients in advanced stages [9,10]. In Japan, pirfenidone is approved for mild-to-severe IPF treatment. Moreover, the medical expenses of patients with severe IPF are covered by the Japanese government according to the Japanese Respiratory Society (JRS) severity grading criteria (i.e., IPF staging) (Table S1 in the Supplementary Material) [5]; subsidies are only provided to patients with disease stages III–IV. Therefore, we anticipated that more patients with severe IPF would receive pirfenidone in Japan, presenting an unique opportunity to investigate the tolerability and efficacy of pirfenidone in patients with severe IPF.

Under the direction of the Japanese regulatory authority, we conducted a post-marketing surveillance (PMS) of all patients who initiated pirfenidone treatment in the first year after its launch in Japan. The primary objective of this study was to analyze pirfenidone clinical safety. Furthermore, the clinical courses of patients on pirfenidone in a real-world observational setting were assessed.

✓ A secção dos métodos é a parte mais importante na avaliação de um artigo, pois contém a informação que permite ao leitor inferir sobre a validade dos resultados e das conclusões descritas.

- ✓ descrição clara e rigorosa de como o estudo foi realizado
- ✓ explicação das escolhas dos materiais e métodos
- ✓ características do desenho do estudo

Embora os métodos devam ser descritos de forma clara e concisa, deverá no entanto conter a informação suficiente que permita:

- (1) Replição do estudo, permitindo aferir a reproductabilidade dos resultados
- (2) Avaliação dos resultados e conclusões por parte dos leitores

1. Desenho do estudo
2. Selecção dos participantes — critérios e métodos de selecção
3. Colecta de dados — vareáveis, métodos e instrumentos
4. Análise de dados

2. Selecção dos participantes — critérios e métodos de selecção
  - a. Critérios de selecção
  - b. Métodos de selecção dos participantes (amostra)
  - c. Processo de recrutamento

## Tiotropium Respimat Inhaler and the Risk of Death in COPD

### METHODS

#### STUDY DESIGN AND OVERSIGHT

The study methods have been described in detail previously,<sup>19</sup> and the complete study protocol is available with the full text of this article at NEJM.org. The trial was performed in accordance with the provisions of the Declaration of Helsinki, and the study protocol and procedures were approved by relevant institutional review boards and ethics committees. All the patients provided written informed consent.

#### STUDY PATIENTS

Full inclusion and exclusion criteria have been reported previously<sup>19</sup> and are included in the protocol. In brief, we enrolled patients who were 40 years of age or older and who had received a clinical diagnosis of COPD, had at least 10 pack-years of smoking history, had a postbronchodilator ratio of the forced expiratory volume in 1 second (FEV<sub>1</sub>) to the forced vital capacity (FVC) of 0.70 or less, and had an FEV<sub>1</sub> of 70% or less of the predicted value. Patients with concomitant

### PROCEDURES

Randomization was based on permuted blocks of nine, stratified according to center. Each patient received one of two possible Respimat inhalers: either 1.25 µg or 2.5 µg per inhalation, and a HandiHaler device; in each case, one of the inhalers held active medication and the other one contained placebo. Thus, patients received tio-

### OUTCOME MEASURES

The primary safety outcome was the time to death from any cause, which was used to calculate the proportional-hazards ratio, or relative risk of death, between groups. The primary efficacy outcome was the risk of the first COPD exacerbation. COPD exacerbations were defined as the worsening of two or more major respiratory symptoms (dyspnea, cough, sputum, chest tightness, or wheezing) with a duration of at least 3 days requiring specified

### STATISTICAL ANALYSIS

We used a Cox proportional-hazard model (with no covariate adjustment) to perform the primary analyses, using a hierarchical analysis plan. The comparisons were tested in the following order

We estimated that a target sample of 16,800 patients was required in order to observe 1266 deaths within 3.5 years of follow-up, assuming a power of 90% with a one-sided P value of 0.025

**Annexin A11 gene polymorphism (R230C variant)  
and sarcoidosis in a Portuguese population**

**Genotyping**

Genomic DNA from patients and controls was extracted from peripheral whole blood samples and collected in acid citrate dextrose (ACD) anticoagulant tubes, using either QIASymphony or QIAmp DNA kits (Qiagen, Venlo, The Netherlands). DNA samples were genotyped for rs1049550 C/T (R230C) using a TaqMan SNPs genotyping assay (Applied Biosystems, Foster City, CA) according to the manufacturer's instructions. Minor groove binding probes were labeled with the fluorescent dyes fluorescein amidite (FAM) and VIC. Polymerase chain reaction was performed in a total reaction volume of 12.5  $\mu$ l with TaqMan Genotyping Master Mix 1 $\times$ , 20 ng of genomic DNA and the SNP genotyping assay 1 $\times$ . The amplification protocol began with a denaturation step at 95°C for 10 min, followed by 40 cycles of denaturation at 92°C for 15 s and annealing and extension at 60°C for 1 min. After polymerase chain reaction, the genotype of each sample was automatically determined by measuring allelic-specific fluorescence on the ABI PRISM 7000 Sequence Detection Systems using sds 1.2 software for allelic discrimination (Applied Biosystems).

**Statistical analysis**

Allele and genotype distributions were statistically analyzed with the chi-squared test, or the Fisher exact test when appropriate. Relative risks or ORs with their 95% confidence intervals (CIs) were calculated as association measures. The software used was the STATCALC program (EpiInfo 2002, Centers for Disease Control and Prevention, Atlanta, GA). *P*-values of less than 0.05 were considered statistically significant. Annexin A11 allele frequencies in patients and controls were determined for each allele using the following formula: allele frequency (%) =  $(n/2N) \times 100$ , where *n* indicates the sum of a particular allele and *N* indicates the total number of individuals. Deviations from the Hardy–Weinberg equilibrium were tested with ARLEQUIN v3 software (23) using the method described by Guo and Thompson (24).

## Resultados

Descrição clara, concisa e objetiva dos achados

- “Espelho dos métodos”
- Estrutura:
  - População incluída
  - Análise primária
  - Análise secundária
  - Achados inesperados e adicionais

### Results

The distribution of *rs1049550* genotypes did not show any significant departure from the Hardy–Weinberg equilibrium, *P*-values for the exact test exceeded 0.05 in both patients and healthy controls groups. The frequency of the *rs1049550*\*T allele was significantly lower in sarcoidosis patients (33.2%) than in controls (44.9%) (*P* < 0.001, OR = 0.61, 95% CI = 0.45–0.82). The frequencies of annexin A11 alleles are summarized in Table 1. An OR of 0.52 and 0.44 for sarcoidosis was obtained, respectively, for the carriers of one (genotype CT) and two (genotype TT) copies of the *rs1049550*\*T allele, when compared with the CC wild-type genotype (*P* < 0.001). When the patients were divided into subgroups with (*n* = 55) and without (*n* = 145) Löfgren syndrome, we observed no significant differences in the frequency of the *rs1049550*\*T allele (*P* = 0.37). However, when these subgroups were compared with controls, only those without Löfgren syndrome had a significant decreased frequency of the allele (Table 2). No significant differences were observed between frequencies of *rs1049550*\*T when patients were compared in terms of lung function impairment patterns and radiological stages (Scadding criteria). We did, however, observe a significant increase in the frequency of the T allele

in patients with neutrophilia in BAL fluid (>3%; *n* = 30) (45 vs 31%, *P* = 0.04, OR = 1.58, 95%CI = 1.02–2.47) and this association persisted after adjustment to smoking habits and the presence of lung fibrosis. No significant associations were found between the presence of the *rs1049550*\*T allele and disease progression (resolution or chronicity) over a period of either 2 or 5 years (Table 3) or when the chronic stable (*n* = 41) or chronic unstable (*n* = 42) forms of the disease were compared (data not shown).

**Table 1** Association between annexin A11 *rs1049550 C/T* single nucleotide polymorphism and susceptibility to sarcoidosis<sup>a</sup>

	Sarcoidosis	Controls	OR	95% CI	P-value
Allele					
rs1049550 C	278 (66.8%) <sup>b</sup>	217 (55.1%) <sup>b</sup>	1		
rs1049550 T	138 (33.2%) <sup>b</sup>	177 (44.9%) <sup>b</sup>	0.61	0.45–0.82	<0.001
Total (2n)	416	394			
Genotype					
CC	99 (47.6%)	61 (31.0%)	1		0.001
CT	80 (38.5%)	95 (48.2%)	0.52		
TT	29 (13.9%)	41 (20.8%)	0.44		
Total (n)	208	197			

CI, confidence interval; OR, odds ratio.

<sup>a</sup>Allele frequencies of annexin A11 variants between patients and controls were compared using the chi-squared test; genotype frequencies were compared using the chi-squared test for trend normalized to the CC homozygotes (OR set to 1).

<sup>b</sup>Allele frequencies.

**Table 2** Frequency of annexin A11 *rs1049550 C/T* single nucleotide polymorphism in patients with sarcoidosis according to presence or absence of Löfgren syndrome (LS)<sup>a</sup>

	Sarcoidosis		Controls	Sarcoidosis without LS vs controls			Sarcoidosis with LS vs controls		
	With LS	Without LS		OR	95% CI	P-value	OR	95% CI	P-value
Allele									
rs1049550 C	70 (64%) <sup>b</sup>	198 (68%) <sup>b</sup>	217 (55.1%) <sup>b</sup>	1		0.001	1		0.11
rs1049550 T	40 (36%) <sup>b</sup>	92 (32%) <sup>b</sup>	177 (44.9%) <sup>b</sup>	0.57	0.41–0.79		0.76	0.53–1.07	
Total (2n)	110	290	394						
Genotype									
CC	25 (46%)	71 (49%)	61 (31.0%)	1		0.001	1		0.12
CT	20 (36%)	56 (39%)	95 (48.2%)	0.51			0.51		
TT	10 (18%)	18 (12%)	41 (20.8%)	0.38			0.6		
Total (n)	55	145	197						

CI, confidence interval; OR, odds ratio.

<sup>a</sup>Allele and genotype frequencies of annexin A11 variants between patients and controls were compared using the chi-squared test; genotype frequencies were compared using the chi-squared test for trend normalized to the CC homozygotes (OR set to 1). Eight patients were not included because the presence of Löfgren syndrome could not be confirmed.

<sup>b</sup>Allele frequencies.

**Table 4** Multivariate analysis of prognostic factors.

Factors	OR	%95 CI	<i>p</i> -Value
<i>PFS</i>			
DM	1.83	1.20–2.79	0.005
Stage	1.69	1.25–2.30	0.001
<i>OS</i>			
DM	2.38	1.48–3.81	<0.001
Stage	3.76	2.45–5.77	<0.001
Performance status	1.92	1.32–2.79	0.001

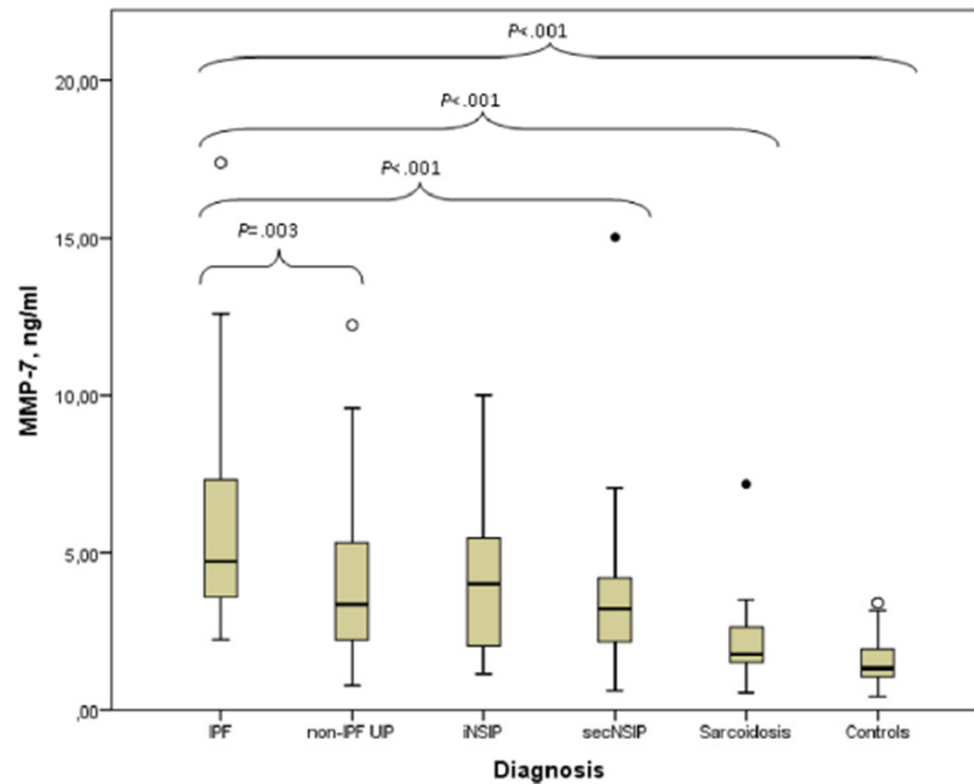
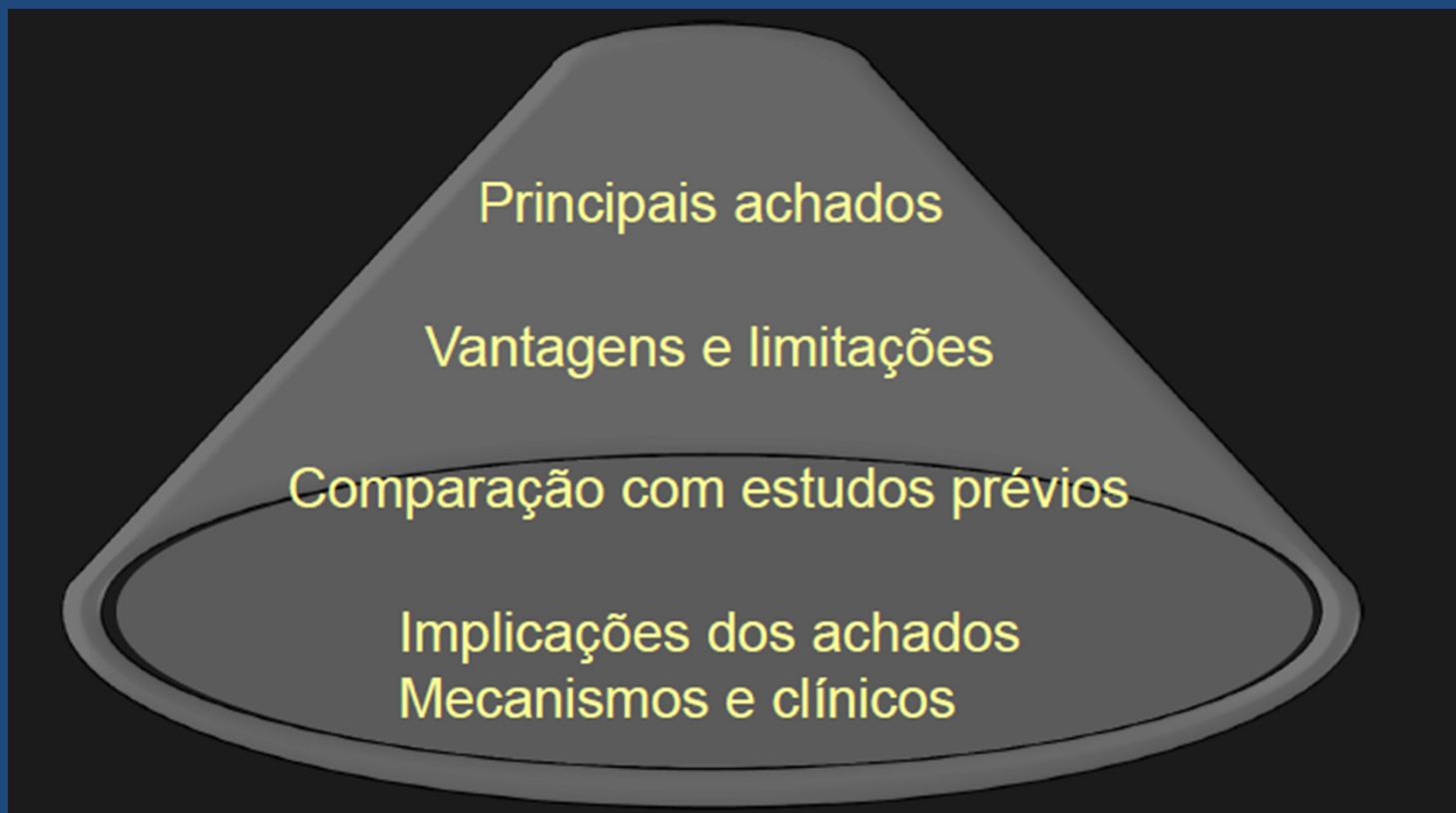
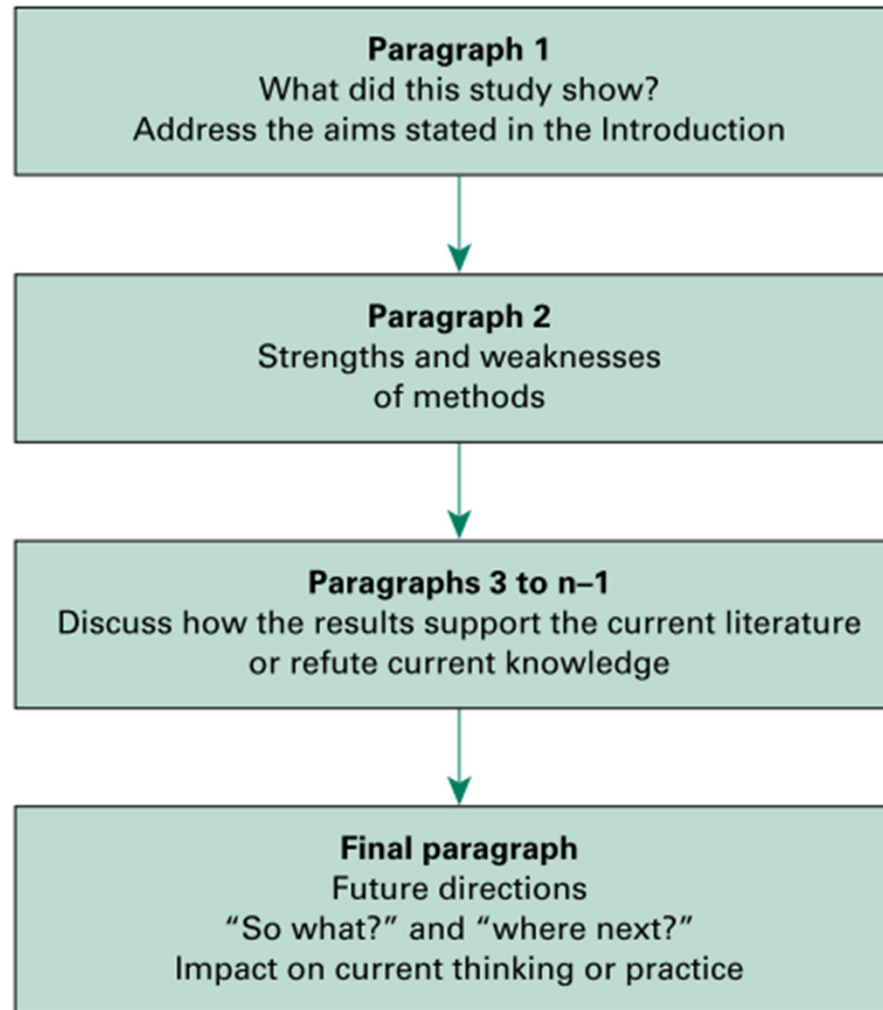


Fig. 2. White circles represent mild outliers and black circles represent extreme outliers. MMP-7 levels in patients and controls. Levels were significantly higher in IPF patients than in controls ( $P < .001$ ), patients with non-IPF UIP ( $P = .003$ ), secondary NSIP ( $P < .001$ ), and sarcoidosis ( $P < .001$ ). MMP indicates matrix metalloproteinase; IPF, idiopathic pulmonary fibrosis; non-IPF UIP, non-IPF usual interstitial pneumonia; iNSIP, idiopathic nonspecific interstitial pneumonia; secNSIP, NSIP secondary to a connective tissue lung disorder; UIP, usual interstitial pneumonia.

## Discussão

Como avaliar um artigo





## Referências:

- Reconhecer fontes
- Suportar a hipótese
- Demonstrar conhecimento do assunto
- Apenas as referências mais importantes e de maior qualidade
- Publicações *peer-review*

Após a avaliação do artigo deverão ser colocadas as seguintes questões:

- What specific problem does this research address? Why is it important?
- Is the method used a good one? The best one?
- What are the specific findings? Am I able to summarize them in one or two sentences?
- Are the findings supported by persuasive evidence?

- Is there an alternative interpretation of the data that the author did not address?
- How are the findings unique/new/unusual or supportive of other work in the field?
- What are some of the specific applications of the ideas presented here? What are some further experiments that would answer remaining questions?