La structure IMRaD : écriture d'un article scientifique

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- I (Introduction)
- M (Methods)
- R (Results)
- a
- D (Discussion)

Section	Contenus-clés
Introduction	Problématique, état des connaissances, justification et objectifs
Méthodes	Population, critères, protocoles, éthique, analyse
Résultats	Description échantillon, résultats, analyses, tableaux/figures
Discussion	Interprétation, comparaison, limites, implications

Adoptée dans la quasi-totalité des articles biomédicaux : clarté, standardisation, rigueur

Pourquoi une telle structure ?

- Permet la lecture rapide et systématique par tous les chercheurs du monde
- Favorise la comparaison entre études
- Guide le processus de rédaction pour les auteurs

Pourquoi une telle structure ?

Contexte du projet ?
Question de recherche ?

Pourquoi ?

Outils pour répondre à la question ?

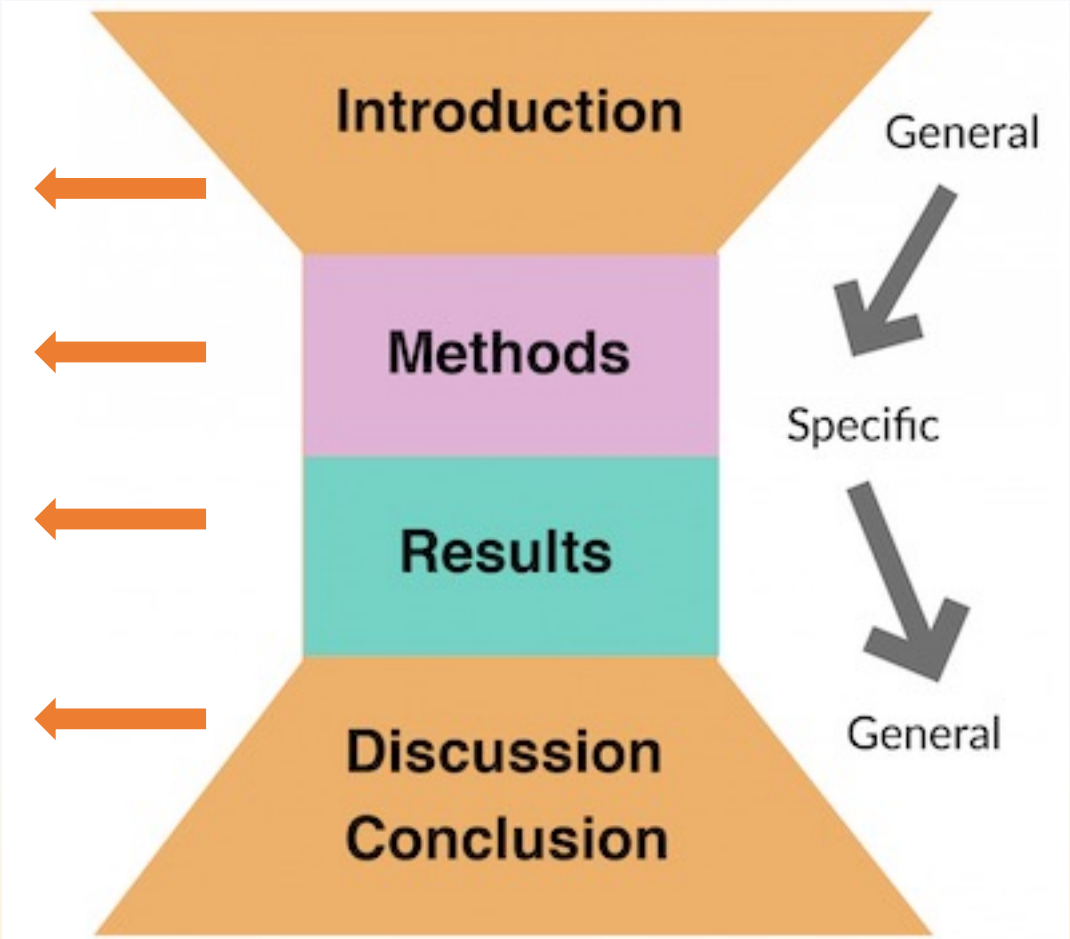
Comment ?

Quelle est la réponse à la question ?

Quoi ?

Que signifie le projet ?
Comment s'intègre-t-il par rapport aux autres études ?

Et donc ?



Introduction

- Objectif : Présenter la problématique, poser l'état des connaissances, justifier l'étude et formuler l'objectif/hypothèse

- Exemple d'accroche : "Malgré les campagnes de prévention, les taux de vaccination stagnent…"

- À faire/À éviter :
  - Faire : préciser contexte, citer les principaux articles, finir sur l'objectif
  - Éviter : surcharger de détails, citer trop de références secondaires

Introduction

Rheumatoid Arthritis (RA) is a chronic autoimmune inflammatory disease that targets articular joints, transforming the synovium into an inflamed, hyperplastic and invasive pannus that results ultimately in cartilage and bone destruction [1]. Such joint destruction in RA and in mouse models of inflammatory arthritis (e.g., collagen-induced arthritis [CIA]), has long been associated with the chronic autoimmune inflammation that results from dysregulated T helper cell responses [2]. Nevertheless, more recently, interest has also focused on the important contributions that synovial fibroblasts (SFs) make to pathogenesis, from the early onset of the disease through to established inflammation and joint destruction [1]. During pathogenesis, SFs adopt a mesenchymal/fibrotic phenotype [3], undergoing epigenetic rewiring in response to the hypoxia and inflammation generated in the microenvironment of the arthritic joint [4,5]. This rewiring is evidenced by changes in their global DNA methylation status, particularly the hypomethylation of promoter regions associated with the de-repression of inflammatory genes driving SF transformation [1,4,6–8]. Collectively, expression of these inflammatory genes (e.g., cytokines and matrix metalloproteinases [MMPs]) drives cellular infiltration, pannus formation and, via the impact of RANKL on osteoclastogenesis, the cartilage and bone damage leading to joint destruction [1,4,5,7]. Indeed, recent studies have now provided direct evidence that such hyper-responsive SFs drive both joint inflammation and bone damage in mouse models of RA [9]. ES-62-mediated protection (and that of small molecule analogues [SMAs] based on its active PC-moiety) against CIA is associated with its ability to resolve the autoimmune IL-17-driven responses that would otherwise promote the pathogenic microenvironment of the arthritic joint: ES-62 achieves this by targeting TLR4 to downregulate aberrant MyD88 signalling and reset homeostatic immunoregulation, primarily by suppressing $CD4^+$ and γδ T cell-derived IL-17 production and restoring levels of regulatory B cells (Bregs) [10–13]. However, ES-62 also acts to suppress the hyper-inflammatory phenotype of CIA-SFs [12] and prevent osteoclastogenesis [14].

Méthodes

Contenus-clés :

- Population, critères d'inclusion/exclusion
- Matériel, protocole détaillé
- Plan statistique, méthode d'analyse
- Aspects éthiques (consentement, comité)

Exemple : "Étude prospective, monocentrique, menée chez 120 étudiants…"

FACE assays

Fast-activated cell-based (FACE) assays were performed as we described previously [92]. SFs were seeded at 10^4 cells/well (96-well plates) and after overnight incubation in 1% FCS DMEM to synchronize the cells, were then stimulated for the indicated time points (from 0 to 120 minutes) before being treated with fixative buffer (Biolegend) according to the manufacturer's recommendations. Once permeabilized, the cells were incubated with antibodies specific for either the activated or whole protein form of the signalling element (e.g., ERK1/2 and the dually phosphorylated active forms of ERK [pERK; Cell Signalling Technology]) for 1 hour before addition of streptavidin-conjugated horseradish peroxidase for 30 minutes (anti-rabbit HRP, Cell Signalling Technology). Following washing and incubation with tetramethylbenzidine (TMB), absorbance was read at 450nm using a Tecan Sunrise plate reader.

Western Blot analysis

SFs were seeded in 6-well plates at $1x10^6$ cells per well and synchronised overnight in 1% FCS DMEM before being treated as indicated. Following washing, cells were lysed by addition of 100 µl of RIPA buffer (50 nM Tris buffer pH 7.4, 150 mM sodium chloride, 2% (v/v) NP40, 0.25% (w/v) sodium deoxycholate, 1 mM EGTA, 1x Halt protease inhibitor and 1x Halt phosphatase inhibitor [Pierce]) and the protein concentration of the solubilised proteins measured by the BCA protein assay kit (Pierce). Proteins (20 µg) were separated by SDS-PAGE on 4–12% Bis-Tris gradient gels, using the NuPAGE Novex system (Invitrogen) in NuPAGE MOPS buffer, with addition of antioxidant. Proteins were then transferred onto nitrocellulose membranes (GE) using the wet transfer NuPAGE system as described previously [93]. Following blocking (5% non-fat milk protein in Tris buffered saline-1% (v/v) Tween-20 [TBS-T]), membranes were incubated with the relevant primary antibody in 5% BSA TBS-T overnight at 4˚C. After washing, secondary antibody conjugated to HRP was added in 5% milk for 1 hour at room temperature and following washing developed using ECL Western blotting substrate (Thermo Fisher Scientific) and X-ray film (Kodak). Membranes were stripped and re-probed using Restore Western Blot stripping buffer (Thermo Fisher Scientific) as described previously [93] and exemplar blots shown (S7 Fig).

mRNA and miRNA analysis

Total RNA and miRNA were isolated and separated using the miRNeasy kit (Qiagen) following the manufacturer's instructions.

For miRNAs, the miScript Reverse Transcription Kit (Qiagen) was used for cDNA preparation. miScript SYBR green qPCR kit (Qiagen) and miScript primer assay (Qiagen) were used for semi-quantitative determination of the expression of mouse miR-19b-1 (MS00005915), miR19b-1* (MS00024493), miR-19b-2* (MS00024500), miR-23b-2 (MS00032606), miR-24-1* (MS00011543), miR-34a (MS00001428), miR-124*(MS00011081), miR-125a (MS00001533) miR-146 (MS00001638), miR-155 (MS00001701), miR-203 (MS00001848), miR-203* (MS00011452) and miR-346_2 (MS00032753). The expression of Hs-RNU6-2_11 (MS00033740) was used as endogenous control [94]. SFs were transfected with the miR-155

À votre avis, pourquoi préciser vos méthodes en détail ?

Résultats

But : Montrer ce qui a été observé/mesuré — pas d'interprétation ici !

Éléments :

- Caractéristiques de la population
- Résultats principaux, secondaires
- Tableaux, figures

À faire/À éviter :

- Faire : chiffres précis, clarté des graphiques
- Éviter : commentaires/interprétations (c'est pour la discussion !)

Results

Exposure to ES-62 *in vivo* induces a stably modulated CIA-SF phenotype

SFs from ES-62-treated CIA mice exhibit reduced spontaneous and IL-17-stimulated IL-6 release *ex vivo*, relative to those from control CIA mice [12]. To investigate whether this reflects stable rewiring of their functional phenotype, SF explant cultures (passaged for 3–4 weeks) from non-arthritic (Naïve; no CIA induction) and PBS- (CIA; disease control) and ES-62-treated mice undergoing CIA (ES-62) were examined for release of proinflammatory cytokines, chemokines and MMPs (Fig 1). We now show that both the increased spontaneous release of IL-6 in CIA- relative to Naïve-SFs and the ES-62 rescue of CIA-SF responses back towards the naïve functional phenotype is evident even after such sustained culture of SFs, *ex vivo* (Fig 1A). Moreover, IL-17-, IL-1β-, LPS- and BLP-stimulated release of IL-6 by SFs from PBS-treated CIA mice was similarly suppressed by *in vivo* ES-62 treatment (Fig 1B–1E), as was the spontaneous and IL-17- and LPS-stimulated CCL2 release by SFs from CIA mice (S1A and S1B Fig). This pattern of suppressed cytokine and chemokine release was found to be mirrored at the mRNA level (S1C and S1D Fig), analysis that also revealed spontaneous and IL-17-stimulated induction of MMP9 and MMP13 to be similarly downregulated in ES-62-CIA-, relative to CIA-, SFs (Fig 1F and 1G). At the same time, whilst the key negative regulators of such proinflammatory signalling, SOCS1 and SOCS3 [15, 16] are downregulated in CIA-SFs, exposure to ES-62 rescues their expression towards the higher levels observed in Naïve-SFs (Fig 1H and 1I). Reflecting this observed differential imprinting of pro-inflammatory and cartilage/extracellular matrix-degrading SF activities, whilst the joint damage observed in CIA-mice (Fig 1J) is associated with increased levels of hypoxia in joint cells (Fig 1K) and induction of vascular leakage (Fig 1L), these pathologies are countered by ES-62 (Fig 1J–1L).

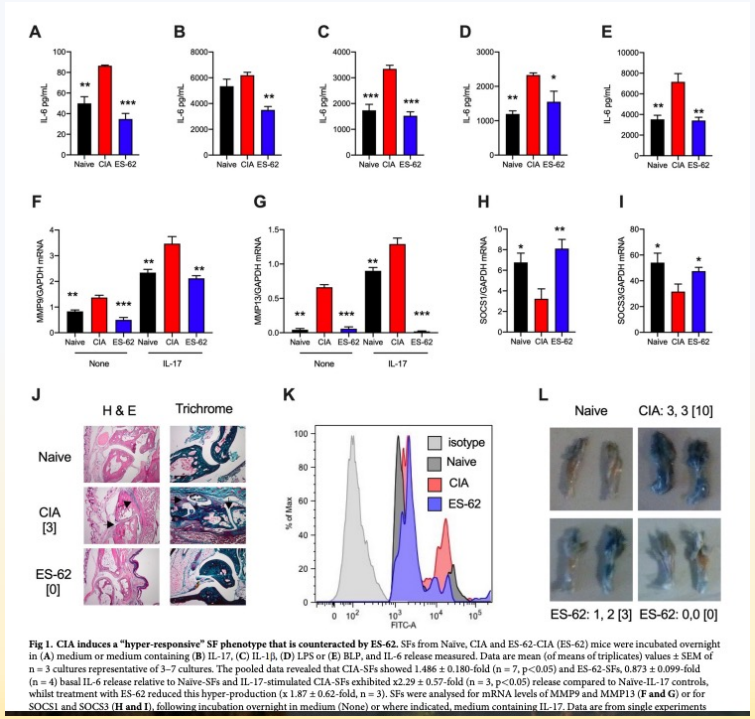


Fig 1. CIA induces a "hyper-responsive" SF phenotype that is counteracted by ES-62. SFs from Naïve, CIA and ES-62-CIA (ES-62) mice were incubated overnight in (**A**) medium or medium containing (**B**) IL-17, (**C**) IL-1β, (**D**) LPS or (**E**) BLP, and IL-6 release measured. Data are mean (of means of triplicates) values ± SEM of n = 3 cultures representative of 3–7 cultures. The pooled data revealed that CIA-SFs showed 1.486 ± 0.180-fold (n = 7, $p<0.05$) and ES-62-SFs, 0.873 ± 0.099-fold (n = 4) basal IL-6 release relative to Naïve-SFs and IL-17-stimulated CIA-SFs exhibited x2.29 ± 0.57-fold (n = 3, $p<0.05$) release compared to Naïve-IL-17 controls, whilst treatment with ES-62 reduced this hyper-production (x 1.87 ± 0.62-fold, n = 3). SFs were analysed for mRNA levels of MMP9 and MMP13 (**F and G**) or for SOCS1 and SOCS3 (**H and I**), following incubation overnight in medium (None) or where indicated, medium containing IL-17. Data are from single experiments

Discussion

Missions :

- Interpréter les résultats
- Comparer avec la littérature existante (« Nos données sont cohérentes avec… »)
- Souligner limites de l'étude
- Ouvrir sur implications/applications
- Conclusion synthétique

Exemple : "Nos résultats suggèrent que… mais l'échantillon reste limité…"

Discussion

Increasing evidence suggests that exposure to pathogens, including parasitic worms, shapes our immune responses and indeed, such "training" has been associated with the epigenetic rewiring of immune system effectors, as well as their progenitors and haemopoietic stem cells. Indeed, although not examined at the level of the methylome, we have previously shown ES-62 (which, based on a protein BLAST search, has homologs in other filarial nematodes including species that parasitise humans, with ~70% identity) to induce stable anti-inflammatory phenotypes of dendritic cells (DCs) and macrophages (from bone marrow-derived progenitors) and immunoregulatory B cells in the MRL/Lpr mouse model of SLE [56–58]. Thus, in this context, there are key novel features arising from the current study. Firstly, that a single, defined parasite-derived molecule possesses the capacity to induce stable epigenetic remodelling of target cells to suppress the pathogenic outcomes of chronic inflammation. Secondly, that ES-62 does this, not by simply suppressing the chronic inflammation and subsequent reprogramming of aggressive inflammatory cells, but rather, by additionally directing further cell differentiation to a novel "protective/repair" phenotype.

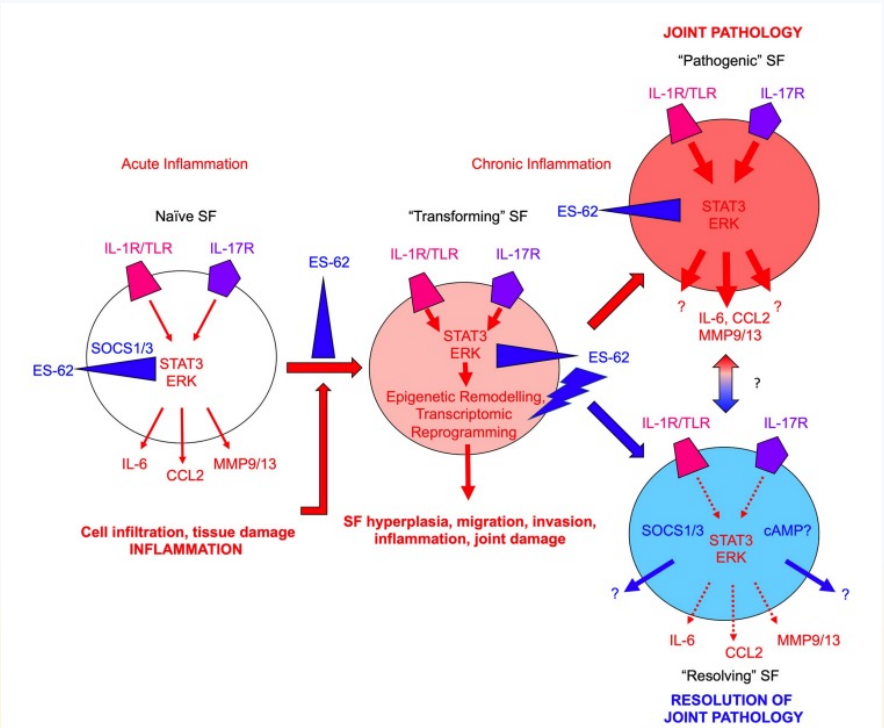


Fig 9. Model of ES-62 action in SFs during CIA. Healthy SFs (Naïve) respond to acute pro-inflammatory signals (e.g., IL-1β/IL-17) in the joint microenvironment resulting in release of pathological mediators such as IL-6, CCL2 and MMP9/13, which cause cell (neutrophils, macrophages, lymphocytes) infiltration and tissue damage, resulting in chronic inflammation. In addition to perpetuating joint inflammation and damage, chronic inflammation drives rewiring of the epigenetic landscape and consequently, reprogramming of the SFs to a "pathogenic" aggressive hyperplastic, migrating and invasive phenotype that, via its hyper-responsiveness to environmental cues, plays a key role in joint pathology and destruction (all pathogenic signals denoted by red arrows with imprinted hyper-responsiveness represented by increased arrow size and redness of SF). ES-62 can disrupt this process at multiple points: thus, it can inhibit (blue blocking symbol) the (i) initial pro-inflammatory signalling to suppress acute inflammation; (ii) inflammatory signals generated by infiltrating cells and tissue damage to suppress induction of chronic inflammation; (iii) signalling associated with chronic inflammation to suppress transformation of SFs to an aggressive hyper-responsive phenotype and (iv) pathogenic signals in aggressive SFs during established disease. Consistent with our findings that ES-62 does not simply maintain/restore the Naïve-SF phenotype, it also exhibits positive actions (blue lightning bolt symbol), specifically inducing epigenetic remodelling (perhaps of an intermediate anti-inflammatory SF phenotype, generated by inhibition of pathogenic ERK and STAT3 signalling) that reprograms the cell to an inflammation-resolving and tissue repair phenotype. The potential ability of the "resolving" ES-62-CIA-SFs to also directly influence the behavior of "pathogenic" SFs in the joint during established disease is represented by the red-blue gradient arrow.

Résumé, titres, références

- Importance du résumé (abstract) : doit pouvoir être compris seul, en 250-300 mots
- Titres précis et hiérarchisés
- Références à jour et de qualité

Suppression of inflammatory arthritis by the parasitic worm product ES-62 is associated with epigenetic changes in synovial fibroblasts

Marlene Corbet [1], Miguel A Pineda [1], Kun Yang [1], Anuradha Tarafdar [1], Sarah McGrath [1], Rinako Nakagawa [2], Felicity E Lumb [3], Colin J Suckling [4], William Harnett [3], Margaret M Harnett [1]

Affiliations + expand

PMID: 34748611 PMCID: PMC8601611 DOI: 10.1371/journal.ppat.1010069

Abstract

ES-62 is the major secreted protein of the parasitic filarial nematode, Acanthocheilonema viteae. The molecule exists as a large tetramer (MW, ~240kD), which possesses immunomodulatory properties by virtue of multiple phosphorylcholine (PC) moieties attached to N-type glycans. By suppressing inflammatory immune responses, ES-62 can prevent disease development in certain mouse models of allergic and autoimmune conditions, including joint pathology in collagen-induced arthritis (CIA), a model of rheumatoid arthritis (RA). Such protection is associated with functional suppression of "pathogenic" hyper-responsive synovial fibroblasts (SFs), which exhibit an aggressive inflammatory and bone-damaging phenotype induced by their epigenetic rewiring in response to the inflammatory microenvironment of the arthritic joint. Critically, exposure to ES-62 in vivo induces a stably-imprinted CIA-SF phenotype that exhibits functional responses more typical of healthy, Naïve-SFs. Consistent with this, ES-62 "rewiring" of SFs away from the hyper-responsive phenotype is associated with suppression of ERK activation, STAT3 activation and miR-155 upregulation, signals widely associated with SF pathogenesis. Surprisingly however, DNA methylome analysis of Naïve-, CIA- and ES-62-CIA-SF cohorts reveals that rather than simply preventing pathogenic rewiring of SFs, ES-62 induces further changes in DNA methylation under the inflammatory conditions pertaining in the inflamed joint, including targeting genes associated with ciliogenesis, to programme a novel "resolving" CIA-SF phenotype. In addition to introducing a previously unsuspected aspect of ES-62's mechanism of action, such unique behaviour signposts the potential for developing DNA methylation signatures predictive of pathogenesis and its resolution and hence, candidate mechanisms by which novel therapeutic interventions could prevent SFs from perpetuating joint inflammation and destruction in RA. Pertinent to these translational aspects of ES-62-behavior, small molecule analogues (SMAs) based on ES-62's active PC-moieties mimic the rewiring of SFs as well as the protection against joint disease in CIA afforded by the parasitic worm product.

Erreurs fréquentes à éviter

- Mélanger méthodes et résultats
- Oublier les aspects éthiques ou la justification scientifique
- Surinterpréter les résultats sans limites
- Jargon excessif ou ambigu

Rédiger efficacement : bonnes pratiques, pièges et éthique

Pourquoi la qualité d’écriture compte-t-elle autant ?

Une bonne recherche mal rédigée, c’est une recherche invisible ou incomprise !

Exemple : Article refusé car résumé confus ; donnée déformée à cause d’une mauvaise phrase

Qui a déjà eu du mal à comprendre un texte scientifique ?

Les principes d’une rédaction claire

- Phrase courte, sujet-verbe-complément
- Structure logique (du général au particulier, « entonnoir »)
- Limiter le jargon, expliquer (même brièvement) les acronymes ou termes techniques
- Soutenir l’apport scientifique par des chiffres, exemples, analogies
- Astuce : Faire relire votre texte à une personne extérieure au domaine

Les principaux pièges à éviter

- Ambiguïtés, approximations, erreurs de syntaxe
- Surcharge d’informations (trop de détails hors sujet)
- Manque de cohérence entre les différentes parties (exemple : l’objectif annoncé n’est pas celui testé)

Éthique et intégrité scientifique

- Définition : L'honnêteté dans la rédaction et la présentation des résultats est une obligation.
- Plagiat :
  - Copier-coller sans citer = FRAUDE
  - Auto-plagiat = aussi à proscrire
  - Conséquences graves : retrait d'articles, perte de diplôme, poursuites

  - Confidentialité, consentement (pour études cliniques : toujours anonymiser et déclarer les démarches éthiques)

Pratiques vertueuses

- Toujours citer ses sources (et éviter le copier-coller non référencé)
- Déclarer toute aide ou conflit d'intérêt
- Relecture croisée : demander à un pair de relire, puis incorporer les corrections
- Utiliser les correcteurs et outils anti-plagiat avant tout dépôt

L'utilisation de l' IA pour produire un texte, puis le présenter comme un travail personnel sans le mentionner est considérée comme une fraude académique : c'est une forme de plagiat

ChatGPT

- Générateur de texte pas une encyclopédie ! Bien pour résumer, reformuler, traduire
- Pas de recherche d'informations sur internet mais prédiction du texte le plus probable

→ **Modèle statistique et non base de données !**

- Il peut déformer des infos, inventer des études, faire des raccourcis

→ **Hallucinations**

- Ne cite pas ses sources
- Et même si demandées elles sont parfois (souvent) inventées

- Pas connecté en temps réel à l'actualité (recommandation de santé)

- Entrainé sur TOUT internet, avec de plus en plus de textes générés par IA

→ **Entshittification**

Utiliser des IA spécialisées selon les besoins

1 ★★★★½ Gratuit

Perplexity AI

« Un moteur de recherche qui donne des réponses précises et fiables à des questions complexes »

EDUCATION / ÉTUDES
IA POPULAIRES +2

VISITER 754

2 ★★★★★ Freemium

WolframAlpha

« Calculez ou demandez ce que vous voulez à cette incroyable IA, experte en science (niveau très avancé) »

EDUCATION / ÉTUDES
RECHERCHE & SCIENCE

VISITER 74

3 ★★★★½ Gratuit

Consensus

« Exploitez en profondeur les résultats tirés de la recherche scientifique depuis un moteur de recherche dopé à l'IA »

EDUCATION / ÉTUDES
RECHERCHE & SCIENCE

VISITER 69

4 ★★★★★ Gratuit

Semantic Scholar

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EDUCATION / ÉTUDES
MOTEUR DE RECHERCHE +2

VISITER 58

5 ★★★★★ Payant

Scite.ai

« Une plateforme de recherche IA pour découvrir et évaluer des articles scientifiques. Recherchez parmi 1,2 milliard de citations et 187..

EDUCATION / ÉTUDES
MOTEUR DE RECHERCHE +1

VISITER 56

6 ★★★★½ Gratuit

Liner AI

« Un copilote IA pour votre espace de travail, propulsé par ChatGPT. Trouvez rapidement des informations et des sources de...

EDUCATION / ÉTUDES
MOTEUR DE RECHERCHE +1

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7 ★★★★½ Freemium

Paperpal

« Votre assistant IA pour l'excellence académique. Profitez de corrections de langue précises, d'un contrôle anti-plagiat intégré, d'une aide à la..

EDUCATION / ÉTUDES
RECHERCHE & SCIENCE +1

VISITER 53

8 ★★★★★ Freemium

SciSpace AI Writer

« Cet assistant de recherche très performant est doté d'un puissant dispositif IA d'aide à la rédaction. Créez du contenu scientifique point...

RECHERCHE & SCIENCE
RÉDACTION & WEB SEO

VISITER 34

Exercice en groupe de 2-4 personnes

En groupe de deux, choisissez une question de recherche

Rédigez :

- 2 phrases d'**Introduction** (problématique + objectif)
- 2 phrases de **Méthodes** (sujet, protocole, durée, mesure)
- 2 phrases de **Résultats** fictifs
- 2 phrases de **Discussion** (interprétation et limite)

Déposer sur Moodle

1. L'écoute de musique améliore-t-elle la concentration chez les étudiants ?
2. Boire un café avant un examen augmente-t-il la vigilance ?
3. Dormir moins de 6 heures par nuit influence-t-il la mémoire ?
4. Marcher 30 minutes par jour améliore-t-il l'humeur ?
5. L'utilisation du téléphone avant de dormir réduit-elle la qualité du sommeil ?
6. Boire de l'eau pendant les cours augmente-t-il la concentration ?
7. Les étudiants qui prennent un petit-déjeuner réussissent-ils mieux aux tests de mémoire ?
8. La couleur des murs d'une salle de classe influence-t-elle la motivation ?
9. Faire une sieste de 20 minutes améliore-t-il la productivité dans l'après-midi ?
10. L'utilisation d'écouteurs avec musique douce réduit-elle le stress avant un oral ?