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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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PSORIASIS PATHOGENESIS: INSIGHTS FROM TRANSCRIPTOMICS AND PROTEOMICS STUDIES OF KERATINOCYTES

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Abstract.

Background: Complex inflammatory skin disease with autoimmune roots is psoriasis. This disease affects various cell types, and the underlying signaling processes are complicated yet not fully understood.

Results: Extensive psoriatic lesions' proteome and transcriptome of several researches were combined to understand disease's underlying biological mechanisms. According to a network-based study, both transcriptomics and proteomics control were comparable. They discovered many pathways of signaling previously undiscovered and possibly involved in overexpression of psoriasis genes. They also found a collection of transcription factors in charge of this process. The functional overlap between the results of transcriptomics and proteomics was also examined.

Conclusion: There created a network-based method for combining the analysis of many high-throughput data sources. Proteomic and transcriptome studies of psoriasis data sets demonstrated regulatory flexibility apparatus underpinning disease and complementary relationships within two cellular organizations.

Key words. Psoriasis, keratinocytes, skin biopsies.

Introduction.

The hyper proliferation of keratinocytes in psoriasis causes the skin to develop thicker, scaly plaques. Psoriasis is a chronic inflammatory skin illness. Psoriasis has a complicated etiology that involves interactions among genetic, environmental, and immunological variables. The fundamental processes behind the onset and progression of psoriasis have become better understood because to research in transcriptomics and proteomics. The disruption of protein profiles and gene expression in psoriatic keratinocytes has been the main subject of these investigations [1]. Pathogenesis of psoriasis involves dysregulated cytokine signaling in a significant way. Small proteins called cytokines function as immune system signaling molecules, controlling inflammatory reactions and immunological responses. An imbalance in the synthesis and signaling of cytokines causes persistent inflammation and aberrant keratinocyte growth in psoriasis. The signaling pathways of these dysregulated cytokines and the constant inflammation and aberrant keratinocyte proliferation they cause in psoriasis are perpetuated via a feedback loop [2]. Immune dysregulation is a significant contributor to a diagnosis of psoriasis. It entails an abnormal immunological response characterized by the malfunctioning of innate and adaptive immune systems. Psoriatic plaque development, aberrant keratinocyte proliferation, and chronic inflammation are all caused by immunological dysregulation. A common chronic inflammatory skin disorder called psoriasis affects around 2% of a world's population and is brought on by an immune response that is dysregulated and manifested in excess hyperproliferation and abnormal keratinocyte differentiation.

The homeostasis of skin milieu depends on a precise and balanced relationship among keratinocyte proliferation and differentiation and immunological defense [3].

The multilayer architecture of human skin is made up of a variety of cell types, primarily keratinocytes and fibroblasts, and immune cells, melanocytes, adipocytes, and endothelial cells that control processes that result in wound healing, pathogenic infection, exposure to ultraviolet radiation, and toxicity. The transcriptome profile of skin can throw light on skin physiology and disease by revealing details on gene expression, non-coding regulatory components, and gene splicing [4]. This pathogenic characteristic develops due to severe inflammatory infiltration, aberrant keratinocyte differentiation, neovascularization, and epidermal hyper proliferation. Psoriasis is a multifaceted aetiology currently poorly understood, but Genetic sensitivity and immunity dysfunction lead to a suggestion as these is the primary cause. Additionally, several environmental variables, including stress, infections, cigarette smoking, and alcohol use, are strongly linked to the onset of psoriasis [5]. Insights into pathogenesis of psoriasis are gained through keratinocyte transcriptomics and proteomics studies; however, to achieve a more complete understanding of condition, that is crucial to supplement these results with other methods, including an entire tissue analysis, animal models, and clinical studies. A more comprehensive understanding of a pathophysiology of psoriasis is possibly achieved by integrating several levels of research to transcend the limitations of individual studies [6].

The paper [7] examined how T lymphocytes, myeloid cells, keratinocytes, innate lymphoid cells, and stromal cells are affected by the skin conditions hepatitis (PSO) and psoriasis-associated arthritic (PSA). Using specific cells connections are translational processes. The paper [8] elevated expression of CD147 in human and animal models of Imiquimod (IMQ)-induced psoriasis into a role of this integral membrane protein in pathogenesis of disease. The paper [9] conducted cell-of-origin enrichment analysis inside Geno dermatoses, identifying the interaction pathways used by dangerous cellular groups, thus suggested a number of potential therapy options. This comprehensive datasets are contained in a publicly available digitized repository to facilitate mechanical and therapeutic

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studies of both typical and damaged skins. The paper described the state of glycolysis in psoriatic skin and evaluated a benefit of a glycolysis score for treatment choice [10]. The paper [11] explored the tissue, cellular, and molecular processes that underlay clinical variability in Psoriatic arthritis (PsA) symptoms and provided an overview of recent tissue-based research, including information obtained by advanced interrogative techniques with single-cell accuracy. The paper [12] investigated ACKR2 expression in PsA skin and compared the transcriptome of Psoriatic Arthritis (PsA) skin to Healthy Control (HC) skin. The paper [13] intended to characterize keratinocyte population-level epidermal changes in psoriasis. A unique cell type-specific method was used to isolate epidermal cell populations from skin biopsies taken people with psoriasis and healthy volunteers. The paper [14] examined single-cell RNA sequencing's technical concerns and accessible technologies and some of its applicability to various skin diseases. The paper [15] illness affecting human skin is psoriasis.

Additionally, autoimmune reactions throughout the body are thought to be responsible for the disease's development, which affects several tissues. The paper [16] validated models that incorporate key psoriasis characteristics in terms of evaluation of the epidermal morphology, gene and protein expression profiles, and limitations of the available models. The paper [17] developed as well as medications that target biomarkers are assessed. The approaches for finding biomarkers using genomes, transcriptomics, proteomics, metabolomics, and microbiome investigation are also covered. The paper [18] discovered that keratinocyte-specific ablation of Glut1 did not impair the development or homeostasis of mouse skin, although the facilitative transporter Glut1 mediates most glucose transport in keratinocytes. The paper [19] determined ex vivo patterns of gene expression of regulatory vs. pathogenic immune cell subsets in psoriasis skin patients to circumvent the constraint mentioned above and apply single-cell transcriptomics to emigration skin cells. The paper [20] provided an in-depth analysis of the state-of-the-art understanding of Given a focus on pathophysiology findings relevant for both therapeutic and diagnostic reasons, the identification of new indicators, and there possible use for precision medicine in Psoriatic Arthritis (PsA), this study examines the role of -omics in PsA.

Investigations by the fields of transcriptomics and proteomics were being conducted to pinpoint the individual genes and proteins that are dysregulated in psoriatic keratinocytes as opposed to normal keratinocytes. Potential diagnostics for psoriasis diagnosis or determining the severity of the illness include these dysregulated molecules. Scientists can identify the signaling pathways and molecular mechanisms that contribute to the emergence and management of psoriasis diseases from examining the gene expression and protein profiles of psoriatic skin cells. Using this information, tailored medicines may be created.

Materials and Methods.

Skin biopsies:

The Vavilov Institute of General Genetics review board approved the acquisition of human tissue, and the study was carried out with the patient's permission and following the Declaration of Helsinki Principles. Three patients with psoriasis provided 6 paired skin biopsies, 3 of which were nonlesional and 2 of which were lesional. Each donor who donated biopsy tissue signed a written informed permission form allowing the study's use of the tissue. Additional file 1 contains a list of all patients' clinical information. All patients had full-thickness punch biopsies performed on healthy skin and the affected edge of a psoriatic plaque.

Preparing a sample:

The solubilization buffer, which contains 8 M urea, 2 M thiourea, 0.5% (w/v) trampolines 4/10, 20 mM Tries base, 1 was mechanically homogenized on ice using a pestle and mortar. Seven times the weight of the tissue in wet conditions was chosen as solubilization buffer per sample. After that, samples were thoroughly sonicated with a Bandelin sonicator at 50% power for 15 seconds at 4°C. DTT was used to boost the concentration after homogenization and sonication to its final value of 50 mM, and samples were kept at 6°C for 35 min. After adding, it was incubated for a further 3 hours before being separated for 10 minutes at 12,000 g to get rid of any remaining insoluble material. After 3 l of sample solution were placed onto an 8 x 9 cm mini-gel and subjected to 1D-SDS-PAGE, using a Bio-Rad GS-800 Calibrated Densitometer, relative amount of protein was determined.

Electrophoresis in two dimensions:

Utilized was the CA-IEF approach for the first dimension. The Protean II xi (Bio-Rad) cell was used for isoelectric focusing. Before being extruded from tubes, second-dimension gels were equilibrated in a 50 mMTris-buffer pH 7.2 solution with 3% SDS, 7% glycerol, and 3% DTT for 25 min. The typical methodology was followed to create 8-14% linear gradient slab gels to separate proteins in second dimension.

Image analysis of gel:

Silver staining and 300 dpi scanning were used to identify protein spots on 2-DE gels. Utilizing Melanie III, images were examined. Every traditional examination included three steps: (i) gel alignment; (ii) spot matching; and (iii) Relative volume of a protein spot (%Vol) calculation, which is calculated by dividing total density of pixels in a specific place by the total pixel intensities throughout the whole gel. Additionally, sets of %Vol data for each site were subjected to a Student amount of data to determine if there was a statistically significant change in a particular protein between the two defined groups.

MALDI-TOF mass spectrometry is used to identify proteins:

Protein spots were removed at 3-DE gels, destained, and trypsin zed inside gel. They used a MALDI-TOF mass spectrometer with Microflex to perform mass spectrometry on trypsin-digested proteins. Equal parts of a solution of 2, 5-dihydroxybenzoic acid in 30% acetonitrile, then 0.1% trifluoroacetic acid also peptide sample combined, and resultant air-dried droplets were observed. The mass spectra were collected in reflection mode using internal standards; they were calibrated and had a mass range of 700-5000 daltons. The SNAP algorithm generated peak lists of peptides. Using this Mascot database search tool, proteins were found. The search criteria were Homo sapiens

taxon, NCBI protein sequence database, a mass tolerance of 100 ppm, one skipped cleavage, propionamide alterations that differ for cysteines and methionines, and oxidation for cysteines. Although they lack cleavage sites, proteins with a low molecular were more easily detected mass spectrometry utilizing nanoLC-MS/MS. The electrospray ion trap outfitted with a chip cube head was used to analyze the trypsin digest. Each sample was run for 10 minutes in an isocratic buffer with 1 µl of a reverse-phase in-chip column. Following samples uses, proteins are segregated during sixty mins using a linear gradient of choices A and B. Spectral scanning was carried out three times for all tissue hydrolysate and protein gel spots. Positive polarity eluted peptide's mass spectra were concurrently collected in both the MS and MS/MS modes in 450–1485 m/z range, with an applied 55 milliseconds of 2.1 kV buildup of 86200 ions, averaged across 3 s. The following parameters were used to identify proteins using the SwissProt Human Database: Score 8 for peptides and 25 for proteins, Minimum precursor mass tolerance is 2.9 D, maximal peptides ions charging is +5, and outcome masses tolerances is 0.8 Da. A minimum of three unsuccessful distinctions and a maximum of four peaks of the same peptide ion were required to recognize proteins.

Examination of microarray data:

They took advantage of a freshly released data collection from the GEO source. They compared 28 sets of samples. Each sample's results were normalized to its median sample value to harmonize expression signal distributions. Welch paired t-test with FDR correction was performed to evaluate a differential expression. If a probe set's average fold change was greater than 2.4 and its FDR-adjusted p-value fell below 0.01, it was deemed differentially expressed.

Analysis of over connections:

The Meta-Core software package was used to conduct all network-based studies. With a wide range of capabilities to examine high-throughput data functionally, this program uses a comprehensive and thoroughly maintained library of interactions between biological objects. They considered a gene to be over-connected if a relevant node has more connections with direct than would be expected by chance nodes of interest. Employing r - the number of interactions - in a hypergeometric distribution among interest list also investigated nodes was used to determine the significance of over connection; N represents all interactions in a database, n total amount of interactions that contain interest-getting genes, and R is degree about a studied node.

$$o(val)(q,m,Q,M) = \sum_{j=mx(q,Q+m-M)}^{\min(m,Q)} 0(j,m,Q,M) \quad (1)$$

Analysis of hidden nodes:

They used items of interest, but they also served as significant upstream regulators for such in addition. Every method as largely was previously stated, but direct linkages aren't considered, just the shortest pathways. The most convenient shortcut to any target object for a transcription factor in the MetaCore database inside the data collection is referred to as a transcriptional activation shortest path since were interested in control of transcription. To account for path's unequal distribution of inhibitory contacts, an introduced another condition. The following parameters were used in a hypergeometric distribution to determine the relevance of a node's importance: r-number of shortest routes among node and gene under study; R, the total number of a gene's shortest routes under study using transcriptional factors; n, a total count of transcript activation most transient pathways; and N, the overall count of shortest paths in databases.

Ranking aggregation:

Individually topological importance procedures generated records of genes substantially connected into an interest protein collection, ordered by matching p-values. They utilized a weighted rank aggregation method to integrate the outcomes of these two procedures. To choose the ideal aggregated list of size 20, the genetic algorithm was used in conjunction with weighted Spearman distance as a distance metric.

Network examination:

Topological analysis was combined with a look at overproduced genes and proteins by choosing related physiologically significant subnetworks enriched with interesting items. The hypergeometric distribution is used to determine the enrichment's significance. We initially employed an algorithm to uncover regulatory pathways that are probably active when there is pathology. It establishes an array of more transcription factors that directly control a collection of target genes, and a group of receptors with ligands are listed among target genes. It creates a sequence of one network per receptor.

Each network contains the ultimate locations, shortest paths taken by each receptor, and each transcriptional component. Using this method, they may identify the key regulatory systems that altered the pathological state under investigation. By p-value enrichment, networks are ordered. The objective of a second-used algorithm was to key transcriptional factors. It considers a database a transcription factor and gradually enlarges the subnetwork surrounding it to a certain size. Enrichment p-value is used for networks. Board.

Results.

Variously abundant proteins:

The amount of protein was measured using MALDI-TOF mass spectrometry after densitometric analysis of a 2D electrophoresis gel's protein spots. Ten proteins in all were at least two times overexpressed in lesional skin relative to unaffected skin: this compound Dismutase [Mn], Enolase 1, S100, Calcium-Binding and Galectin-7 Proteins A9 or A7, Squalors unit Carcinogen Antigen, and Squalors unit Carcinogen-2 are all present as shown Table 1. Previous studies have found that psoriatic plaques contain excessive amounts of several proteins. The proteins represented a variety of pathways and processes. Thus, the keratin gene family includes keratin 14, 17, and 16. Epithelial cell structural integrity is maintained by an intermediate filament protein known as keratins. Serine protease inhibitors SERPINB4 and SERPINB3 control the host immune system's response to cancer cells. The glycolytic enzyme enolase 1, often called alpha-enolase, is an isozyme of enolase and is expressed in most tissues.

Table 1. *Proteins expressed more strongly in lesions.*

Protein name	Gene	Fold change
Superoxide dismutase [MN]	SOD2	3
Antigen for squamous cell cancer; SCC antigen	SERPINB 3	10.4444446
Protein S100-A9	S100A9	Lesion only
SCCA2/SCCa1 fusion protein isoform 2	SERPINB 4	6.32323232
Keratin 17	KRT17	12.5333333
Enolase 1	ENO 1	3.152
Keratin 16	KRT16	14.5333333
Galectin-7	LGALS7B	8
Keratin 14	KRT14	13.5333333
Protein S100-A7	S100A7	Lesion only

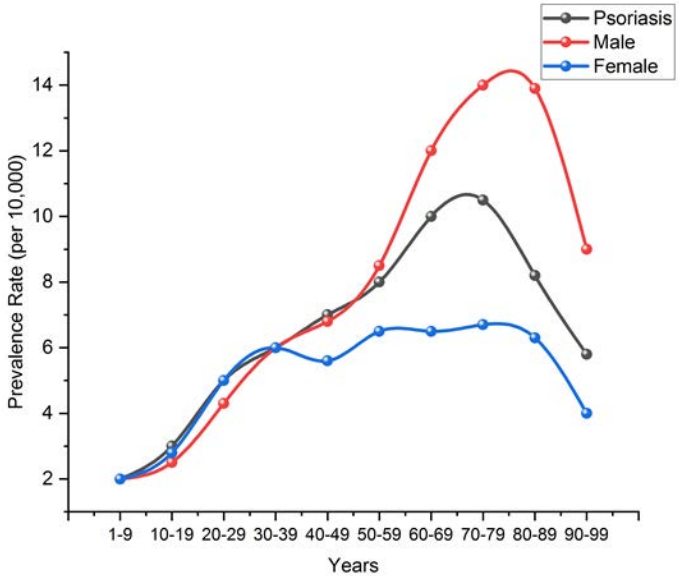

Figure 1. *Prevalence of psoriasis according to age.*

The mitochondrial electron transport chain's byproduct, dangerous superoxide, is converted into diatomic oxygen and hydrogen peroxide by the enzyme superoxide dismutase 2 (SOD2). A class of proteins known as galectins that bind beta-galactosides is thought to influence cell interactions and matrix interactions. Differential and in-depth hybridizations show that keratinocytes are the only cells producing lectin. Location inside cells and significant down-regulation of this protein in cultured keratinocytes suggest that it plays a function in cell-matrix interactions important for maintaining appropriate growth management. Several different cell types, including the S100A9 and S100A7 proteins in their cytoplasm and nuclei, are involved in controlling a number of biological functions, including cell division and cell cycle progression. In psoriatic patients' skin lesions, S100A7 is noticeably overexpressed. Using a database of more MetaCore packages, including more than 300,000 hand-vetted connections involving proteins and various shortest-route methods, they tried to link proteins into a network. Figure 1 depicts the prevalence of psoriasis according to age.

The various proteins' names are listed in this column. Each protein has a unique name and is a big protein with serves a variety of activities for our bodies. Superoxide dismutase [MN], Antigen for squamous cell cancer, SCC antigen, Protein S100-A9, SCCA2/SCCa1 fusion protein isoform 2, Keratin 17, Enolase 1, Keratin 16, Galectin-7, Keratin 14, and Protein S100-A7 are a few of the molecules included in this list. The fold change in every molecule or gene's levels of translation is described in this field. A gene's and a protein's fold change is the measurement of when significantly its transcript has changed from a control condition. Usually, it is shown as a number value. You have the fold change values for each protein or gene in the database. For instance, Collagen 17's fold change is 12.5333333, indicating a markedly higher expression level relative to the reference state. The transcription factors (TFs) STAT1, STAT3, c-Myc, SP1, NFkB and AP-1 complex members, were revealed to control the genes expressing overabundant proteins. In addition, the excess S100A9 through signal transduction kinases and RAGE receptors started the pathways upstream of these TFs' activation. A demonstration of S100A9 expression regulated an NF-kB, indicating the presence of a positive feedback loop in this network. Numerous transcriptomics investigations that demonstrated the psoriatic lesions possess an overexpression of these TFs supported the structure of this proteomics-derived network. Proteomics approaches typically fail to detect transiently expressed TFs because they usually have low protein levels.

Variously expressed genes:

They used 35 psoriasis patients' gene expression data from a recent study collected by Affymetrix. When compared to the same people's nonlesional skin, the first found to be elevated were more than 1400 probe sets. Using more strict statistical criteria, we discovered 461 overexpressed genes in lesional skin. A list of the genes that are overexpressed is in Additional File 2. The results of the proteomics revealed that seven of the 10 proteomic indicators were encoded by overexpressed genes. Enolase 1 expression was neither changed nor Keratin 14 or Galectin 7 expression as show in figure 2, Superoxide dismutase 2 (SOD2) protein concentrations in adult (A, AXE) and elderly (O, OX) rats treated. Include antioxidant damage is thought to have a role in the etiology of aging, cancer, cardiology illnesses, and neurological disorders (such as Alzheimer's and Parkinson's), SOD2 is particularly relevant in these contexts. In order to reduce antioxidant damages or enhance medical results, scientists were investigating SOD2 as a possible therapeutic target.

Common transcriptional control of differentially abundant proteins and overexpressed genes:

Despite a strong agreement between the proteome and expression datasets, direct correlation analysis is challenging given both orders of magnitude differences in list size. They used interactome approaches to analyze the shared upstream regulation of both datasets on level of transcription factors. They used a hidden nodes methodology and two recently established interactome analysis techniques to determine the sets of crucial transcription factor sets. In contrast to the number of interactions predicted to occur randomly, using a one-step overconnectivity measure with the appropriate dataset, the earlier method ranks TFs. In a second method, p-values of local subnetworks are determined using an aggregation technique, accounting for both

Table 2. *Typical transcription factors used in proteomics and expression datasets.*

Transcriptomics					Proteomics		
Enter ex-ID	**Genes**	**Over-Connection P-value**	**Hidden nodes P-values**	**Enter z-ID**	**Genes**	**Over-Connection P-value**	**Hidden nodes p-values**
5966	REL	5.864E-18	4.581E-18	6851	USF2	1.507E-04	-
3394	IRF8	8.584E-12	2.902E-12	6777	STAT5A	-	3.820E-05
4582	RELA	4.766E-16	5.121E-24	5962	STAT6	8.470E-05	3.070E-04
1059	CEBPB	2.580E-13	2.371E-15	7582	FOXL2	3.101E-03	2.423E-05
3659	IRF 1	1.345E-22	3.233E-19	6782	STAT1	2.237E-04	2.293E-04
3660	IRF2	3.796E-12	3.13E-09	7030	TFAP2A	4.807E-04	-
4790	NFICB 1	3.678E-13	2.533E-25	3092	HIF1A	2.346E	2.293E-04
10379	IRF9	8.194E-05	4.047E-17	6778	STAT58	-	3.820E-05
2506	NR3C1	3.0157E-06	2.534E-18	3024	ERG	2.387E-04	3.4528E-07
6688	SPI!	7.019E-14	1.245E-17	6824	STAT4	3.425E-06	-
7452	STAT2	4.091E-29	1.027E-22 5	3214	GLI3	5.421E-04	3.070E-04

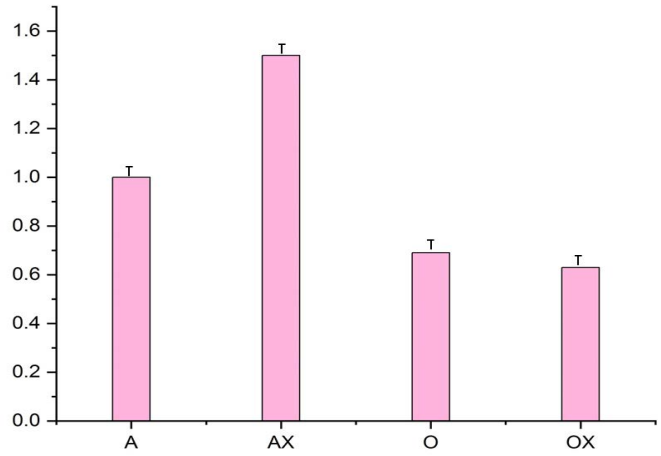

Figure 2. *Superoxide dismutase 2 (SOD2).*

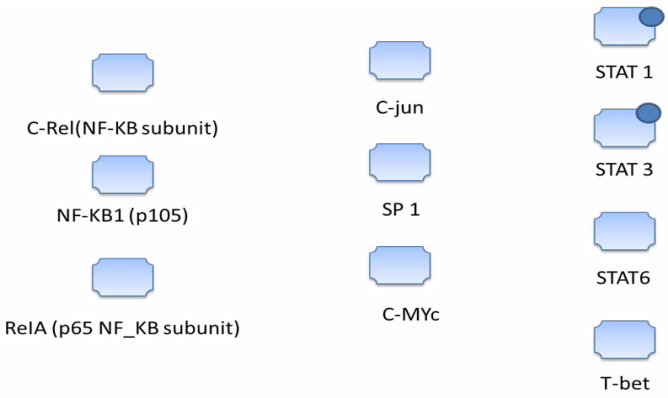

Figure 3. *Transcriptomics and proteomics transcriptional factors regulate objects.*

direct and more distance regulation. Table 2 lists the top 20 TFs in each data category as well as TFs discovered by networks analyses. Figure 3 shows the TFs common to both data kinds as 'major pathological signal transducers'.

The study of all the RNA molecules (transcripts) created in a cellular or tissues at one particular moment is known as transcriptomics. Among these is messenger RNA (mRNA), which transfers genetic data from DNA to the ribosomes so they can synthesize proteins. RNA sequencing (RNA-seq), one underlying basic transcriptomics tools, may offer a thorough understanding of the pathways of gene activation.

Identifying important receptors:

The next phase used the hidden nodes algorithm to determine which receptors had the most potential to cause a transcriptional response. In regulating 426 genes with variable expression, they found that 220 membrane receptors played a substantial role. There is a list of all receptors inside Additional File 3. Since all receptors are not always topologically significantly engaged in essential signaling, even stated in a sample, sorted depending on expressive effectiveness in this list. According to their hypothesis, psoriasis is more likely to activate pathways triggered by over-expressed ligands and receptors. They expected at least a correlation between changes in protein abundance and expression changes.

Discussion.

To better understand complicated illnesses, a meta-analysis of different OMICs studies and data formats is an increasingly important research technique for the correlation analysis of different types of datasets, such as mRNA and proteomics; several techniques and developed. The mismatching of mRNA probes and protein IDs, basic disparities technologies in OMICs, variations during experiment design conducted various organizations, etc., are only a few technical issues that need solving. In addition, biological factors, including variations in the variability of different data kinds, are impacted by RNA and protein degradation processes.

Because of this, although regarded as complementary, Transcriptome and proteome datasets typically exhibit minimal positive association. More recent research concentrated on functional parallels and divergences found for various cellular organization levels and shown in several OMICs data types. Examples include separate changed type 2 diabetes transcripts and proteins with similar interaction objects. Researchers studying leukemia discovered that various changes at the transcriptome and proteome levels represent different aspects of cellular functions that are all dysregulated. Recent study highlighted that extensive unification of mRNA and protein expression patterns, despite data types being mostly compared on a gene/protein level without any functional analysis.

Later, co-analysis of transcriptomic and proteomic data using ontology enrichment demonstrated the complementary and comparable effects of the two data sets on biological processes. The crucial question of biological causation and the functional ramifications of various regulatory processes at the cellular organization's mRNA and protein levels is addressed in detail. Enrichment analysis and other low-resolution available approaches are unable to address these issues. The most advantageous option is to employ more accurate computational techniques, including biological networks' architecture, directed binary interactions, and multi-step paths linking items amongst datasets of various sorts, independent and with directly overlapping genes and proteins. They discovered a considerable direct correlation among data that Seven out of 10 overproduced molecules discovered in psoriatic patches were identified by transcriptomics techniques and proteome are produced in various over-expressed genes. The typical correlation techniques could not be used since the size of the datasets (462 genes versus 10 proteins) differed by two orders of magnitude. Proteomics datasets further reveal systemic bias in that some other proteins operate, preferring "effector" structural and inflammatory proteins and main metabolic proteins instead of those briefly generated and swiftly degradable motioning proteins.

To find shared regulating factors the most important transcription factors and receptors for two datasets, used topological network approaches. To reconstruct the upstream signaling pathways in psoriasis, they discovered some important regulators of proteomics collection among variations in gene expression, including membrane receptors, transcription factors, and extracellular ligands. They specifically found 24 receptors that were not previously associated with psoriasis.

Conclusion.

Two unique high-throughput collections of disease data of various sizes have also been integrated and explored with success using network-based approaches. They discovered the signaling pathways that may be involved in finding common regulatory mechanisms in psoriatic lesions, which changes the regulatory network's state and is expected in the overexpression of genes and proteins. This approach makes integrating the analysis of many data kinds' simple, yields physiologically significant findings, and identifies brand-new therapeutic targets. As a result of their findings show that a wide range of diverse cascades, many of which had not previously been identified as contributing to psoriasis, can both initiate and maintain disease, suggesting that combination treatments that target many pathways may help treat the condition.

Cellular includes complex systems for chemical connections called signaling pathways which allow messages to be sent from one area of the cell to another or from one cell to another. These pathways are essential for a number of cellular functions, such as distinction, development, and responsiveness to environmental stimulus. They frequently consist of a collection of molecules, including receptors, proteins, and enzymes, that relay and amplify signals before they are eventually translated into particular physiological reactions. In order to control both physiological and pathological processes in creatures, signaling pathways are essential.

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Conflict of interest statement.

The author declares no conflict of interest.

REFERENCES

1. Zheng H, Gu L, Zhao F, et al. SerpinB7 deficiency contributes to the development of psoriasis via calcium-mediated keratinocyte differentiation dysfunction. Cell Death & Disease. 2022;13:635.
2. Theocharidis G, Tekkela S, Veves A, et al. Single-cell transcriptomics in human skin research: available technologies, technical considerations, and disease applications. Experimental Dermatology. 2022;31:655-673.
3. Radulska A, Pelikant-Małecka I, Jendernalik K, et al. Proteomic and Metabolomic Changes in Psoriasis Preclinical and Clinical Aspects. International Journal of Molecular Sciences. 2023;24:9507.
4. Krishnan V.S, Kõks S. Transcriptional basis of psoriasis from large scale gene expression studies: the importance of moving towards a precision medicine approach. International Journal of Molecular Sciences. 2022;23:6130.
5. Jin J.Q, Wu D, Spencer R, et al. Biologic insights from single-cell studies of psoriasis and psoriatic arthritis. Expert Opinion on Biological Therapy. 2022;22:1449-1461.
6. Zhou Y, Wang P, Yan B.X, et al. Quantitative proteomic profile of psoriatic epidermis identifies OAS2 as a novel biomarker for disease activity. Frontiers in Immunology. 2020;11:1432.
7. Abdallah F, Henriet E, Suet A, et al. MiR-21-3p/IL-22 axes are major drivers of psoriasis pathogenesis by modulating keratinocytes proliferation-survival balance and inflammatory response. Cells. 2021;10:2547.
8. Chen C, Yi X, Liu P, et al. CD147 Facilitates the Pathogenesis of Psoriasis through Glycolysis and H3K9me3 Modification in Keratinocytes. Research. 2023;6:0167.
9. Thrane K, Winge M.C, Wang H, et al. Single-Cell and Spatial Transcriptomic Analysis of Human Skin Delineates Intercellular Communication and Pathogenic Cells. Journal of Investigative Dermatology. 2023;S0022-202X(23)02040-7.
10. Shou Y, Zhu R, Tang Z, et al. A prediction model identifying glycolysis signature as a therapeutic target for psoriasis. Frontiers in Immunology. 2023;14:1188745.
11. Najm A, Goodyear C.S, McInnes I.B, et al. Phenotypic heterogeneity in psoriatic arthritis: towards tissue pathology-based therapy. Nature Reviews Rheumatology. 2023;19:153-165.
12. Johnsson H, Cole J, Siebert S, et al. Cutaneous lesions in psoriatic arthritis are enriched in chemokine transcriptomic pathways. Arthritis Research & Therapy. 2023;25:1-10.
13. Witte K, Jürchott K, Christou D, et al. Increased presence and differential molecular imprinting of transit amplifying cells in psoriasis. Journal of Molecular Medicine. 2020;98:111-122.
14. Theocharidis G, Tekkela S, Veves A, et al. Single-cell transcriptomics in human skin research: available technologies, technical considerations, and disease applications. Experimental Dermatology. 2022;31:655-673.

15. Łuczaj W, Gęgotek A, Skrzydlewska E. Analytical approaches to assess metabolic changes in psoriasis. Journal of Pharmaceutical and Biomedical Analysis. 2021;205:114359.
16. Niehues H, van den Bogaard E.H. Past, present, and future of in vitro 3D reconstructed inflammatory skin models to study psoriasis. Experimental dermatology. 2018;27:512-519.
17. Aggarwal D, Arumalla N, Jethwa H, et al. The use of biomarkers as a tool for novel psoriatic disease drug discovery. Expert Opinion on Drug Discovery. 2018;13:875-887.
18. Zhang Z, Zi Z, Lee E.E, et al. Differential glucose requirement in skin homeostasis and injury identifies a therapeutic target for psoriasis. Nature Medicine. 2018;24:617-627.
19. Kim J, Lee J, Kim H.J, et al. Single-cell transcriptomics applied to emigrating cells from psoriasis elucidate pathogenic versus regulatory immune cell subsets. Journal of Allergy and Clinical Immunology. 2021;148:1281-1292.
20. Grivas A, Fragoulis G, Garantziotis P, et al. They were unraveling the complexities of psoriatic arthritis by using Omics and their relevance for clinical care—Autoimmunity Reviews. 2021;20:102949.