Meeting Report: Psoriasis Stratification to Optimize Relevant Therapy Showcase

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A stratified medicine approach for the treatment of psoriasis promises greater certainty of clinical decision making through prediction of response on the basis of clinical, pharmacological, and "omics data from an individual patient. As yet, there is no predictive model for treatment response in routine clinical use for psoriasis. The Psoriasis Stratification to Optimize Relevant Therapy (PSORT) Consortium is a United Kingdom Medical Research Council-funded, academic-industrial stratified medicine consortium established with the objective of discovering the predictors and stratifiers of response of psoriasis to biologic therapies. A showcase meeting was convened and attended by 80 stakeholders at the Royal College of Physicians, London, United Kingdom on 18 November 2019. The purpose was to disseminate the research findings from the PSORT consortium discovered thus far. This report summarizes the presentations made on the day and the significant advances made by PSORT toward a stratified medicine approach to the management of psoriasis.


Introduction

Stratified medicine, where using the latest knowledge and technology to stratify patients for different treatments, promises greater certainty of clinical decision making and ideally enables the right therapy to be administered to the right patient at the right time (Bell, 2014). It is a promising concept, but no reproducible biomarker predictive of drug response is currently in routine clinical use for the treatment of psoriasis (Reid et al., 2020). Psoriasis Stratification to Optimize Relevant Therapy (PSORT) is a United Kingdom Medical Research Council-funded, academic-industrial stratified medicine consortium established in 2014 with the objective of using clinical, genetic, and immune biomarkers to predict and reproducibly stratify the response of psoriasis to biologic therapies (Griffiths et al., 2015). On 18 November 2019, a showcase day—for stakeholders, including scientists, clinicians, patient groups, and industry representatives—was held at the Royal College of Physicians, London to disseminate research findings from the consortium. This meeting report summarizes the significant advances made by PSORT toward a stratified medicine approach to the treatment of psoriasis. The meeting comprised a series of presentations made by members of the PSORT research team.

Why investigate stratified medicine for psoriasis? Perspectives from multiple stakeholders

The Psoriasis Association is a patient organization charity for people with psoriasis in the United Kingdom. A patient survey before the launch of PSORT was integral to identify whether people with psoriasis were interested in and accepting of the idea of pretreatment testing to predict response to treatment. Helen McAteer, Chief Executive Officer of the Psoriasis Association, emphasized that the mission of PSORT—to get patients on the right clinical pathway first time—was one shared by patients. Her presentation highlighted a recent priority-setting partnership supported by the Psoriasis Association. The question “What factors predict how well psoriasis will respond to a treatment?” ranked third in the top 10 priorities, highlighting the importance of answering this unmet need (Majeed-Ariss et al., 2019).

Not only is stratified medicine important for patients, but a stratified approach also holds significant potential to help industries in the research and development of medicines. Chris Chamberlain, Merck Healthcare KGA, emphasized the importance of stratified medicine from an industry perspective. Molecular stratification of disease may help inform industry-sponsored clinical
of psoriasis needed to limit the development of comorbidities, which psoriasis therapy would be effective for the treatment of specific comorbidities, and development of noninvasive tests for psoriasis comorbidities. These stakeholder perspectives, ranging from patients with PBC, industry, and international research programs made persuasive arguments for the importance of identifying scalable stratifiers for the management of psoriasis.

PSORT is structured around two integrated work strands: (i) clinical and pharmacology studies, concerned with the discovery of disease endotypes, factors associated with adherence, and the measurement of markers of drug response, and (ii) immune biomarker studies, concerned with the discovery and validation of molecular and immune signatures in the skin and blood associated with treatment response (Griffiths et al., 2015) (Figure 2). A summary overview of the outcomes from all PSORT studies to date is provided in Table 1.

Clinical and health economic outcomes
Clinical, phenotypic parameters are easily accessible, and if they are predictive of response to therapy and are valid, they could be scalable for use in the clinic. The British Association of Dermatologists Biologics and Immunomodulators Register (BADBIR) is an ongoing United Kingdom and Republic of Ireland pharmacovigilance register for patients with moderate to severe psoriasis on either traditional systemic or biologic therapies (Burden et al., 2012). The National Institute for Health and Care Excellence recommends that all patients with psoriasis on biologic therapies in England should be registered on BADBIR, thus ensuring that this cohort of patients is generalizable to the population of patients with severe psoriasis in the United Kingdom. As a large database of over 18,000 patients from 165 contributing dermatology centers, with detailed data on patient characteristics and long-term follow-up as well as aligned serial serum samples and RNA and/or DNA banked for 30% of recruited patients (Griffiths et al., 2015), BADBIR is the ideal resource for interrogation of clinical and pharmacological outcomes in psoriasis.

Zenas Yiu, University of Manchester (United Kingdom), presented data on drug survival of biologic therapies, which represents a composite proxy marker for their effectiveness and safety. Drug survival is highest for ustekinumab than for etanercept, adalimumab, and infliximab when used as a first-line or second-line biologic therapy in BADBIR (Iskandar et al., 2018; Warren et al., 2015). The predictors for first-line biologic discontinuation are female sex and smoking (Warren et al., 2015), whereas patients who discontinue their first-line biologic owing to an adverse event are more likely to discontinue their second-line biologic for the same reason (Iskandar et al., 2018). Antonia Marsden, University of Manchester (United Kingdom), presented work investigating the clinical predictors of effectiveness of adalimumab, etanercept, and ustekinumab in psoriasis using a biologic-naive cohort in BADBIR. Using a reduction in PASI by 90% from baseline (PASI 90) as the outcome for response, a multivariable logistic regression model found that female sex, nonwhite ethnicity, smoking (current or ever), higher weight, unemployment, and psoriasis involving the palms and soles are associated with a reduced likelihood of response to biologic therapy at 1 year (Warren et al., 2019). However, no predictors for a differential treatment response to adalimumab, etanercept, and ustekinumab were found.

**Figure 1.** Professor Christopher Griffiths, PSORT Director, The University of Manchester (United Kingdom) and invited Plenary Lecturer, Professor Kevin Cooper, Case Western Reserve University (Cleveland, OH). PSORT, Psoriasis Stratification to Optimise Relevant Therapy.
Medication adherence is a crucial but potentially overlooked contributor to variation in treatment response. Rachael Thorneloe, Sheffield Hallam University (United Kingdom), presented results from the Investigating Medication Adherence in Psoriasis study that was conducted in a subset of patients in BADBIR. This showed that 16.4% and 29.2% of patients on biologic and traditional systemic therapies, respectively, are nonadherent to treatment. Factors associated with nonadherence are younger age, longer duration of treatment, strong concerns before

<table>
<thead>
<tr>
<th>Study</th>
<th>Outcome</th>
<th>Predictors and/or Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>BADBIR drug survival (Ikandar et al., 2018; Warren et al., 2015)</td>
<td>Drug survival for etanercept, infliximab, adalimumab, and ustekinumab</td>
<td>Female sex, smoking (first line), discontinuing first-line biologic therapy owing to an adverse event in the order of adalimumab, ustekinumab, secukinumab</td>
</tr>
<tr>
<td>BADBIR prediction of drug response (Warren et al., 2019)</td>
<td>Nonattainment of PASI 90 at 1 year for adalimumab, etanercept, and ustekinumab</td>
<td>Female sex, nonwhite ethnicity, smoking, higher weight, unemployment, palmoplantar psoriasis</td>
</tr>
<tr>
<td>iMAP adherence (Thorneloe et al., 2018)</td>
<td>Drug nonadherence for biologic and systemic medications</td>
<td>Younger age, longer treatment duration, strong previous medication concerns, weaker medication taking habits, traditional systemic therapies</td>
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<td>BADBIR first-line biologic selection (Davison et al., 2017)</td>
<td>Predictors of biologic first-line selection</td>
<td>PsA, patient’s weight, employment status, country of registration, baseline disease severity</td>
</tr>
<tr>
<td>Modeling of the sequence of biologic therapies and cost effectiveness</td>
<td>The optimal sequence of therapies to meet cost-effectiveness threshold</td>
<td>In the order of adalimumab, ustekinumab, secukinumab</td>
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</table>

**Immunological and transcriptomic outcomes**

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<thead>
<tr>
<th>Study</th>
<th>Outcome</th>
<th>Predictors and/or Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>BSTOP adalimumab study (Wilkinson et al., 2019)</td>
<td>Minimal effective random pragmatic drug level for adalimumab to achieve PASI 75</td>
<td>Adalimumab level ≥3.2 μg/ml</td>
</tr>
<tr>
<td>BSTOP ustekinumab study (Tsakok et al., 2019)</td>
<td>Association between drug levels and PASI 75 response 6 months later</td>
<td>Drug levels are predictive of response 6 months later, whether sampled early or at a steady state</td>
</tr>
<tr>
<td>BSTOP ustekinumab pharmacokinetic and/or pharmacodynamic modeling study (Loeff et al., 2020)</td>
<td>Clustering of responders and/or nonresponders on the basis of longitudinal PK–PD simulations</td>
<td>Dose escalation improves the probability of response in partial responders but not in nonresponders; nonresponders with a high 4-week trough level may represent a primary nonresponder group where therapy should be switched</td>
</tr>
<tr>
<td>BSTOP ADA study</td>
<td>Association between ADAs and ustekinumab drug levels and treatment response</td>
<td>ADAs associated with lower median ustekinumab levels and higher absolute PASI</td>
</tr>
<tr>
<td>BSTOP genetic determinants of response</td>
<td>Interaction effect of HLA-C*0602 on treatment response for adalimumab and ustekinumab</td>
<td>HLA-C*0602 status associated with a lower likelihood of response for adalimumab than for ustekinumab; the subgroup with PsA associated with a larger magnitude of effect</td>
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**Abbreviations:** ADA, antiprader antibodies; BADBIR, British Association of Dermatologists Biologics and Immunomodulator Register; BSTOP, biomarkers of systemic treatment outcomes in psoriasis; cDC2, type 2 conventional dendritic cell; DC, dendritic cell; iMAP, investigating medication adherence in psoriasis; LPS, lipopolysaccharide; PASI 75, 75% improvement in PASI from baseline; PASI 90, 90% improvement in PASI from baseline; PK–PD, pharmacokinetic–pharmacodynamic; PsA, psoriatic arthritis; PSORT, Psoriasis Stratification to Optimise Relevant Therapy.
medication use, weaker medication-taking habits, and being on a traditional systemic therapy (Thorneloe et al., 2018). The results underscore the importance of clinicians discussing medication concerns with patients before initiation of biologic therapy to avoid subsequent intentional nonadherence.

Stratified medicine may be more cost effective as improved health outcomes, with better treatment response and lower risk of treatment-related adverse events, translate into lower costs for the healthcare system. David Wonderling, Royal College of Physicians (London, United Kingdom), presented results from studies investigating the health economics of psoriasis treatment stratification. A study from BADBIR of 3,040 patients with psoriasis using a multinomial logistic regression model (Davison et al., 2017) identified that the presence of psoriatic arthritis (PsA), patient weight, employment status, country of registration, and baseline disease severity are all associated with the selection of first-line biologic therapy. To understand patient preferences regarding the choice of biologic therapy for psoriasis, a discrete choice experiment was conducted online. This showed that patients are willing to accept delays in treatment or an increase in the risk of serious infection if they use a stratification tool providing higher positive and negative predictive power.

Particular sequences of use of biologic therapies may be more cost effective. A model using a mixture of randomized clinical trial data and published data from BADBIR involving adalimumab, secukinumab, and ustekinumab was fitted, using discrete event simulation, with 500 simulated patients in each experiment for 3 years. A cost-effectiveness threshold of £20,000 to £30,000 per quality-adjusted life-years (QALY) gained was used. QALYs were estimated using the EuroQol-5 dimension measure from BADBIR, and the unit costs for the biologics were taken from standard National Health Service sources. Preliminary analysis using a multivariable Poisson regression model found that the optimal sequence of therapies was adalimumab, ustekinumab, and then secukinumab.

Pharmacological and genomic outcomes
Measurement of pharmacogenomic factors for stratification is well-established in dermatology, with examples such as the measurement of thiopurine methyltransferase before azathioprine prescription or assessment of glucose-6-phosphate dehydrogenase deficiency before consideration of dapsone use. Blood levels of biologic drugs are routinely utilized along with clinical markers to assess treatment response for inflammatory bowel disease, but yet this is rarely implemented in the management of psoriasis. Both pharmacogenomic and pharmacological approaches may identify the predictors of significant clinical effect both before and soon after initiation of treatment.

The bioresource allied to BADBIR, Biomarkers of Systemic Treatment Outcomes in Psoriasis (BSTOP), was utilized for the discovery of pharmacological and genomic markers of biologic treatment outcome. The BSTOP bioresource was interrogated to (i) determine a therapeutic range for adalimumab and ustekinumab for the treatment of psoriasis and (ii) determine whether early measurement of adalimumab and ustekinumab drug levels in the blood predicts treatment response. Nina Wilson, Newcastle University (Newcastle upon Tyne, United Kingdom), presented a study investigating the therapeutic range for adalimumab and ustekinumab. Using random pragmatic drug level sampling, this showed that the minimally effective blood level for adalimumab to achieve a 75% reduction in PASI from baseline (PASI 75) after 1 year of treatment was ≥3.2 μg/ml (Wilkinson et al., 2019). This threshold had a sensitivity of 80% and a specificity of 58% and gives an estimated PASI 75 probability of 65% (95% confidence interval [CI] = 60–71%). Using multivariable logistic regression modeling and adjusting for potential confounders, the probability of achieving PASI 75 plateaus after a median drug level of 7 μg/ml, thereby suggesting the possibility of dose reduction for patients who achieve higher drug levels on random drug level sampling. In contrast to adalimumab, the relationship between ustekinumab drug level and treatment response is less clear (Tsakok et al., 2019). Although early (≤12 weeks after starting treatment) measurement of blood levels of ustekinumab is associated with PASI 75 response at 6 months, this is not the case for PASI 90. There is no plateau effect as observed for adalimumab.

Teresa Tsakok, King’s College London (United Kingdom), presented results from two further studies investigating pharmacokinetic–pharmacodynamic (PK–PD) modeling and the impact of antidrug antibodies (ADAs) on biologic treatment response. Longitudinal PK–PD simulations based on random blood drug level samples found two subgroup clusters of patients on ustekinumab, with one group achieving 6-month PASI 75, labeled as responders, and the other group failing to achieve PASI 50, labeled as nonresponders (Pan et al., 2020). Exploratory simulations suggest that dose escalation may improve the probability of response in partial responders but not in nonresponders. Thus, drug levels should be interpreted along with initial treatment response, that is, a good early response at week 4 may warrant an increase in dose, whereas a poor early response with high 4-week trough levels should suggest switching therapy (Pan et al., 2020).

The development of drug immunogenicity or specifically the development of ADA may contribute to poor treatment outcomes for biologic therapies. In a cross-sectional sample of patients with psoriasis on ustekinumab, the presence of ADA in a drug-sensitive radioimmunoassay and a drug-tolerant ELISA is associated with lower median ustekinumab levels (−0.62 μg/ml [95% CI = −1.19 to −0.30] and −0.74 μg/ml [95% CI = −1.09 to −0.47], respectively) and higher absolute PASI (6.6 [95% CI = 3.0–9.9]) and 1.9 [95% CI = 0.4–4.0], respectively) than in patients without ADAs (Loeff et al., 2020). The development of ADAs is greatest in patients on adalimumab, with 37.8% (drug-sensitive) and 67.3% (drug-tolerant) patients developing ADAs compared with 3.8% and 10.6%, respectively, for patients on ustekinumab.

Genetic determinants of therapeutic response are of considerable interest. The HLA-C*06:02 allele has long been known to be associated with an increased risk of developing psoriasis early in life (Chen and Tsai, 2018). Nick
Transcriptomic and immunological outcomes

Disease endotype, defined as a disease entity or subtype differentiated functionally and pathologically by molecular mechanisms and/or by treatment response, is an important concept within stratified medicine (Anderson, 2008). Although psoriasis is well-characterized phenotypically (Griffiths and Barker, 2007), distinct endotypes have not been well-described thus far. Understanding psoriasis endotypes may help develop stratification biomarkers to better classify psoriasis and predict treatment outcomes. The PSORT Discovery study involved taking blood and skin samples from 41 patients on adalimumab and 41 on ustekinumab over a period of 12 weeks. There were three strands of differential expression analysis: absolute PASI (strand A), to identify gene expression signatures associated with PASI; tissue time (strand T), to identify gene expression signatures that change over time; and clinical response (strand C), to identify gene expression signatures at baseline and week 1 that predict clinical response at week 12.

Simon Cockell, Newcastle University (Newcastle upon Tyne, United Kingdom), presented data from strand T and strand C. In strand T, a complementary method to look at differentially expressed gene sets, Correlation Adjusted Mean Rank, found that an IFN-α response gene set is upregulated in the adalimumab cohort but down-regulated in the ustekinumab cohort 1 week after starting treatment. However, at week 12, this gene set is down-regulated in both cohorts. In strand C, where a clinical response was defined by the change in PASI from baseline, the dampening of IFN signaling at baseline and at weeks 1 and 12 is associated with clinical response to ustekinumab but only at week 12 for adalimumab. These data suggest that patients with dampened IFN signaling before commencing treatment are primed to respond to ustekinumab but not to adalimumab. In contrast, dampening of the inflammasome signaling at baseline is associated with a clinical response to adalimumab but not to ustekinumab. David Watson, Queen Mary University (London, United Kingdom), introduced the audience to machine learning methods used to combine information across different genes to form a prediction model. To combine these potential biomarkers into a predictive score, random forest models were fitted, with recursive feature elimination to choose the collection of biomarkers with good predictive performance.

Monitoring immunological changes by blood sampling may give further insights into mechanisms of therapeutic response, and if validated, this approach could be scalable for clinical use. Rosa Andres Ejarque, King’s College London (United Kingdom), presented insights into the mechanisms and therapeutic response to adalimumab through the investigation of immunological markers in the blood. In a subset of patients (n = 16) on adalimumab, imaging flow cytometry was used to quantify nuclear translocation to understand changes in gene activation. NF-κB activation in lymphoid cells was inhibited by adalimumab. In dendritic cells (DCs), at baseline, increased lipopolysaccharide (LPS)-induced NF-κB translocation is associated with a nonresponder status as defined by nonattainment of PASI 75. Similarly, increased LPS-, IL-1β-, and TNF-induced NF-κB phosphorylation in type 2 conventional DCs (cDC2) at baseline is associated with nonresponse at week 12 of treatment. This indicates that NF-κB phosphorylation in cDC2 at baseline may be a biomarker for response to adalimumab. In a cohort of 28 patients, LPS-stimulated NF-κB phosphorylation of cDC2 at baseline had a sensitivity of 80% and specificity of 88.9% for predicting failure to achieve a PASI 75 response on adalimumab at week 12 (cut-off level of 0.169 log₁₀ fold change). NF-κB is responsible for the induction of DC maturation and subsequent expression of costimulatory molecules, and in vitro generated baseline DCs of patients failing to reach PASI 75 increased the upregulation of costimulatory molecules.

Conclusions

Clinical predictors and blood levels of biologic, along with HLA-C*0602, represent promising areas where translation from bench to clinic may be adopted in a meaningful way to predict the response of psoriasis to biologic therapy. Larger cohort validation of the transcriptomic predictors, such as gene expression signatures from skin biopsies and induction of NF-κB translocation or phosphorylation from blood samples, is required. Furthermore, large-scale collaborations between scientists, clinicians, and the pharmaceutical industry in the spirit of the PSORT consortium will evaluate whether predictors of response exist for other biologic classes, such as inhibitors of IL-17 or IL-23, as well as for oral small-molecule immunomodulators.

Any laboratory test or biomarker needs to be scalable, widely accessible, and cost effective within the current clinical laboratory technology framework to be adopted into routine clinical practice. A pragmatic and integrative systems medicine, algorithmic approach is needed, whereby an over-all parsimonious prediction model combining the biomarkers of the highest differential predictive value for treatment response along with more accessible clinical predictors will have to be developed and validated in large international cohorts. Such a model should be tested, ideally in a pragmatic randomized controlled trial, to evaluate...
the impact of stratified medicine on real-world clinical outcomes such as population treatment response, cost effectiveness, and improvements in drug safety.

Iain McInnes, University of Glasgow (Scotland), provided an overall summary of the legacy from PSORT. He outlined the concept of immune-mediated inflammatory diseases (IMIDs) together comprising an area of significant unmet need, introducing the term IMIDology where diseases are no longer classified by the phenotype of the affected organ but rather by molecular endotypes that cluster together by the commonality of cytokine pathways and targeted therapies. He highlighted several important messages from the PSORT consortium: (i) molecular medicine is the future direction for medicine, as demonstrated by the identification of stress-inducible proteins, inflammatory response gene sets, and blood immunological markers in PSORT as the potential stratifiers of biologic response; (ii) systems biology offers untold potential, and the PSORT findings of potential distinct endotypes will be one more step in understanding the complexity of psoriasis and its response to treatment; (iii) academic–industry partnerships are important in extending our knowledge base as exemplified by the multistakeholder partnership in PSORT; and (iv) sustainability for any findings is important and requires urgent attention. Thus, the findings from the PSORT consortium represent a systematic and significant first step on the journey to fulfill the promise of stratified medicine in the management of psoriasis and IMIDs overall.

CONFLICT OF INTEREST
CMEG reported receiving honoraria and/or research grant support (University of Manchester, United Kingdom) from AbbVie, Almirall, Boehringer Ingelheim, Bristol-Myers Squibb, Celgene, GlaxoSmithKline, Janssen, the LEO Foundation, Eli Lilly, Nestle Skin Health, Novartis, Pfizer, Sandoz, Sun Pharma, and UCB Pharma. NJR reported receiving honoraria, travel support, and/or research grants (Newcastle University, Newcastle upon Tyne, United Kingdom) from AbbVie, Almirall, Amgen, AstraZeneca, Bristol-Myers Squibb, Celgene, Genentech, Janssen, the LEO Foundation, Novartis, PSORT partners (www.PSORT.org.uk), Pfizer, Sanofi Genzyme Regeneron, Stiefel GlaxoSmithKline, and UCB Pharma. JNWNB reported receiving honoraria, research grants, and/or consulting fees from AbbVie, Almirall, Amgen, Bristol-Myers Squibb, Celgene, LEO Pharma, Novartis, Janssen, Roche, Regeneron, Eli Lilly, UCB Pharma, Samsung, Sienna, Sun Pharma, Boehringer Ingelheim, and GlaxoSmithKline. RBW received consulting fees from AbbVie, Almirall, Amgen, Arena, Avillion, Boehringer Ingelheim, Bristol-Myers Squibb, Celgene, Janssen, LEO Pharma, Eli Lilly, Novartis, Pfizer, Sanofi, Xenopur, and UCB Pharma. CHS reported receiving research support from AbbVie, GlaxoSmithKline, Novartis, Pfizer, Regeneron, Roche, and Medical Research Council consortium, which had several industry partners (see psort.org.uk). PDM has received research grants from UCB and consultancy and/or speaker honoraria from Novartis, UCB, and Janssen. The remaining authors state no conflict of interest.

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