Gastric Cancer Transcription Factors in Patient Reported Outcomes (GCTF-PRO) – Draft proposal

The GCTF-PRO seeks to examine the extent gastric cancer patients are tapping into new information particularly outside of conventional healthcare disclosures. Its significance is in assessing dimensions of QOL paradigms that frame statistical power using predictive methods. It seeks to embed evidence-based theories (perceptual and cognitive) to awareness levels in an attempt to bridge the biotechnological advances with prognostic/ diagnostic-related patient satisfactions. At present, it may complement existing GC QOL instruments and offer a novel approach on how cellular level prognoses could possibly correlate with QOL measures.

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Gastric Cancer Transcription Factors in Patient Reported Outcomes (GCTF-PRO) – Draft proposal

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Purpose, population and setting

Answerable question in PICO format:

Population	|	Gastric Cancer Patients Stage I-IV (male and female; all ages).
Intervention	|	Cellular localisation prognoses i.e. biomarkers, genes.
Comparator	|	No reference to cellular localisation.
Outcome	|	"Awareness" of transcription factors affect QOL.

→ *Can cellular localisation prognoses affect QOL in GC patients?*

This proposal explores a literature-based PROM in which cell level prognoses i.e. an awareness of transcription factors and cell level monitoring affect a patient's sense of wellbeing or QOL. The projected population are GC patients at disease progression stage I-IV. The setting is in a region e.g. Inner Sydney, with equitable allocation of health resources and access to standard treatment options.

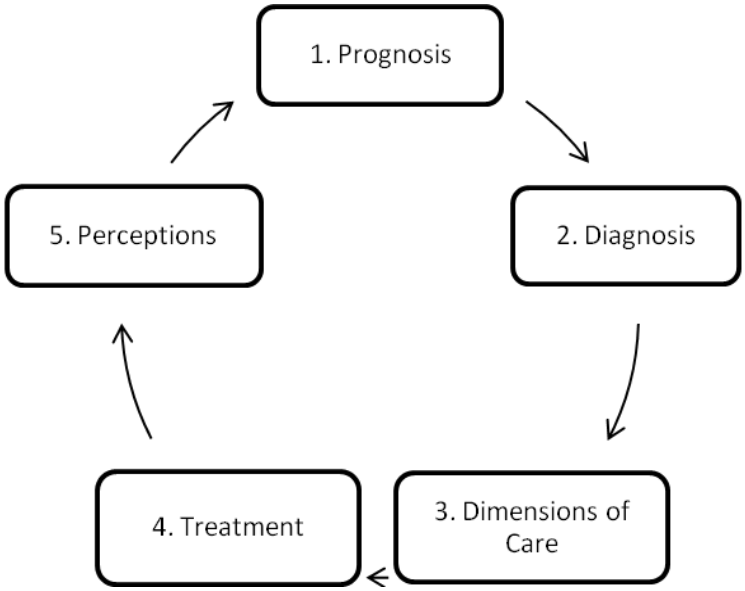
Development rationale

The rationale for GCTF-PRO's development comes from GC studies conducted in Asia and Europe, which promote cell expressions as indicators for staging prognoses (Zhou et al., 2013; Durães et al., 2014; Bilici, 2014). QOL questionnaires such as 'The Bone Metastases QOL' (Badia, Vieta & Gilabert, 2010) assess QOL for disease progression in advanced stages III and IV (palliative care), and measure new therapies for relieving symptoms. However, in gastric cancer, screening and prognoses are considered poor (Xu et al., 2013; Durães et al., 2014). Disease progressions and staging are determined by a series of observations i.e. an endoscopy; recording symptoms; blood tests; imaging scans and biopsies. Poor to modest prognosis data are predominately collated from responses to first-line chemotherapy; imaging scans are difficult to see in the region and remain contentious among treating practitioners (Durães et al., 2014 p. 374); the endoscopy is subject to the specialist's

experience; and biopsies and laparoscopies are underutilised and deemed too novel for treatment (Lin, Huang & Juan 2012 p. 3082; Hartgrink et al. 2009; Segal et al. 1975). There may also be gaps in information a patient is exposed to on the Internet and the media, and information the treating health professional discloses. This lack of evidence-based consistency between treating specialists presents a porous basis for prognosis, and may culminate to psychosocial uncertainties between a treating health professional and a patient – issues of trust, perceived neglect, and social driven factors which concern a patient's QOL. Current advances and cancer trials entail a host of compromises and risks for patients. Since most trial research is yet to translate in practice, patients and caregivers in most instances are left to make decisions based on their intuitions, beliefs, monetary supplies and other practical and psychosocial factors. Despite these risks, when the underlying prognosis is poor and treatment options palliative, patients and carers most likely will seek answers on the Internet. Areas in which their "busy" doctor failed to investigate, and expose themselves to treatment trials and research advances at a cellular level. The GCTF-PRO attempts to measure the extent to which patients are exposed and pursue cellular level information – the role transcription factors i.e. knowledge of cellular localisation, targeted cellular therapies, monitoring cell lines, biomarkers, gene therapy etc. may affect a patient's sense of wellbeing and QOL.

PROM's Description

The questionnaire is disease-specific and is self-completed. A patient fills out a questionnaire based on current disease progression. I constructed 5 domains when eyeballing and collating evidence from the literature:



The questions I had in mind:

1. Prognosis: How many transcription terms is the patient aware of?
2. Diagnosis: How many cellular related diagnoses or diagnostic terms is the patient aware of?
3. Dimensions of care: How many transcription terms did the multidisciplinary team mention?
4. Treatment: How many transcription pharmaceutical trial terms is the patient aware of?
5. Perceptions: How does the patient view information regarding transcription factors?

Domains 1, 2, 3 and 4 list GC transcription terms or diagnoses from current research literature (*Medline*, *Pubmed* databases 2013-2014; See appendix A). However, this is a literature based insight, to construct the questionnaire itself, these domains should be itemised from ideally three focus groups including experts, cross-matched to media popularity and validated in GC populations. A patient circles familiar terms and the scores are arithmetically summed (nominal scale at present). The total score measures the *awareness level* a patient has on transcription factors – the first construct. Domain 5 uses a

semantic differential rating (ordinal) scale with a series of polar opposites. The patient rates each treating professional or medium (internet) – the second construct (appendix B). This domain measures the *areas of trust* a patient has on transcription factors and is scaled by comparing estimates. At present, the raw data I gleaned from the literature (2013 - 2014) contains 247 items for domains 1 and 3; domain 2 has 32 items; domain 4 has 11 items; domain 5 has 30 items. In total there are 320 items. The recall period is dependant on disease progression and staging. The survival rate for advanced GC stages III and IV range from 2 weeks, 6 months to 2 years or more. GC is often detected in the latter stages III and IV. Treatment plans (chemotherapy, radiotherapy and/or surgery) are devised usually within a month from diagnosis. Hence the optimal time period for completing the questionnaire may depend on the optimal time period for recalling transcription factors after diagnosis and during the disease progression. The focus groups and pre-screen selection criteria may help determine an optimal time parameter for administering the questionnaire. The table below summarises the domains, items, scales and representations:

Domains	Items	Scales	Representations
1. PROGNOSIS	247 combined with domain 3	Nominal (potential ordinal)	Level of awareness on transcription factors; summing known items (construct 1).
2. DIAGNOSIS	32	Nominal	Level of awareness on cellular level prognoses; summing known items (construct 1).
3. DIMENSIONS OF CARE	247 combined with domain 1	Nominal (potential ordinal)	Level of awareness on transcription factors; summing known items (construct 1).
4. TREATMENT	11	Nominal (potential ordinal)	Level of awareness on transcription factors; summing known items (construct 1).
5. PERCEPTIONS	30	Ordinal	Areas of trust on transcription factors from scaled comparisons (construct 2).

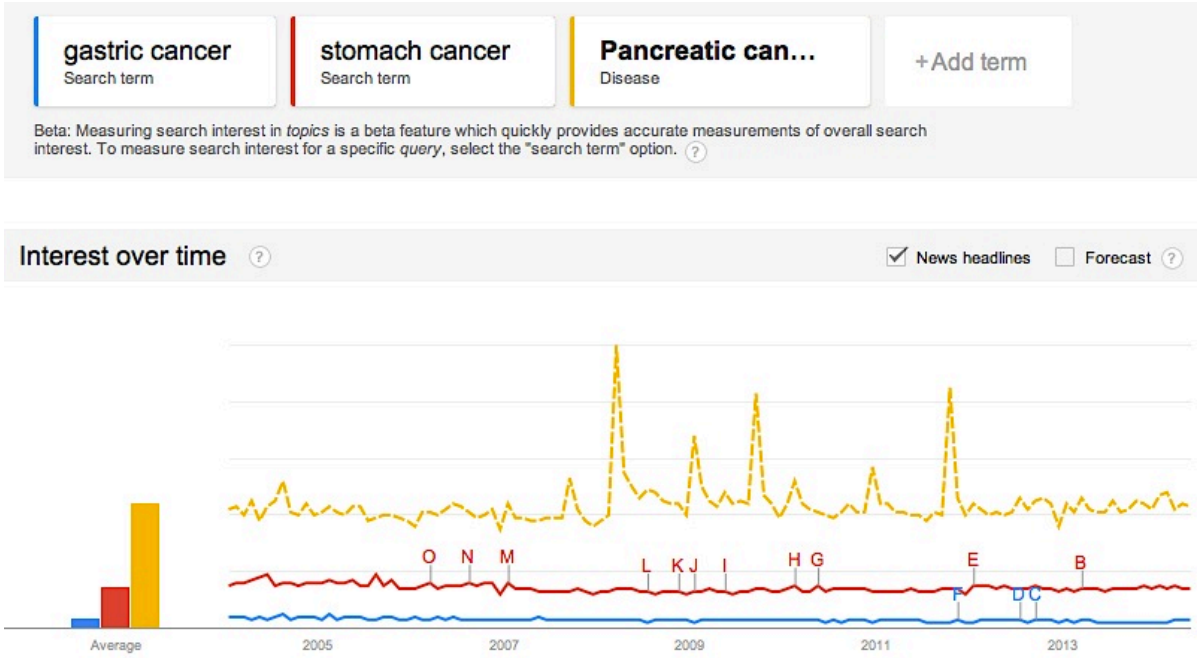
PROM's purpose

The purpose of GCTF-PRO is to complement existing PRO instruments measuring QOL in GC patients: EORTC QLQ-C30, EORTC QLQ-STO22, FACT-G, FACT-Ga, MDASI-GI (Xu et al., 2013; Khanna et al., 2014), and systematically assess whether exposure to potentially undisclosed information such as transcription factors, influence a patient's sense of wellbeing. It may be argued – information empowers. A more informed patient – no matter how intricate the information – is a happier patient. On the other hand, information overload or intangible, idiosyncratic information – may harm a patient. With advancing technologies e.g. recombinant DNA and new therapies involving genetic amalgamations, the patient may be more exposed to these elements, and are increasingly attuned to these potentials (Gotay et. al 2008, p. 1361). For advanced gastric patients, palliative treatment plans offer little hope. So much of the literature today, report cutting-edge, gene-driven frontiers and the information is readily available on the Internet. Do any parts of these published findings offer hopeful incentives? Do they affect a patient's QOL? The GCTF-PRO attempts to explore if such exposures affect a patient's QOL. Ideally, it will eventually be a QOL instrument with latent variables rooted in perceptual and cognitive theories, modelled with heuristics mapped to QOL factors – rather than remaining a questionnaire measuring attitudes towards transcription factors.

Development history

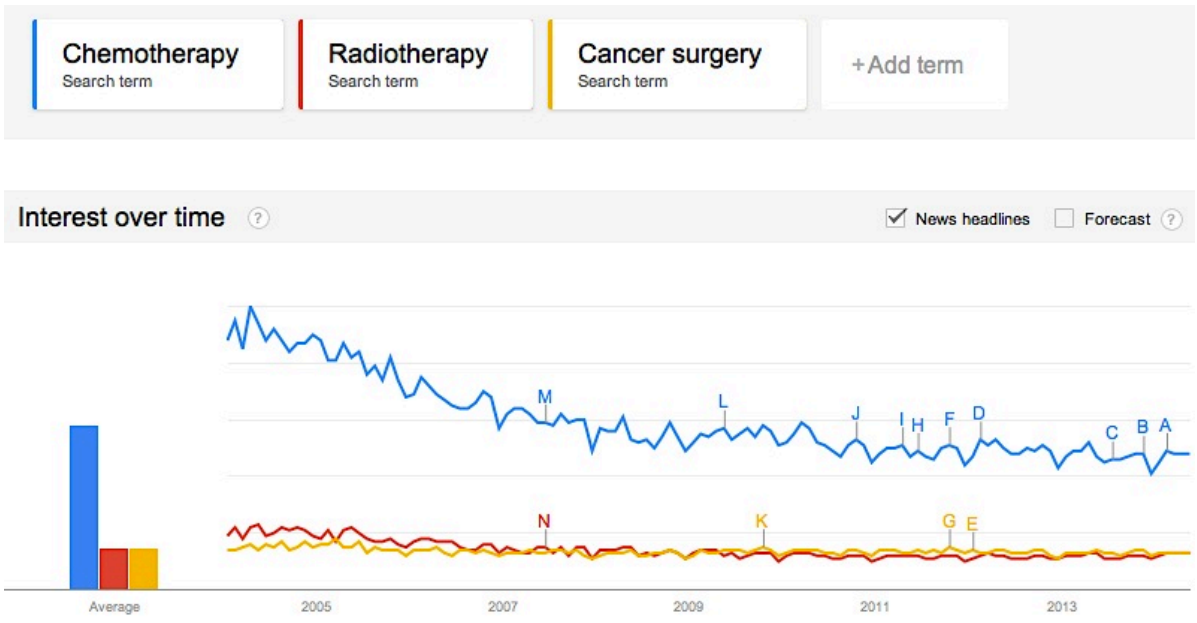
Trends in *Google* (an internet based search engine) generally show a steady trend when searching for gastric cancer related terms between 2004 and 2014:

Fig. 1: Search term trends for 'Gastric cancer,' 'Stomach cancer,' and 'Pancreatic cancer.' Screenshot taken May 26th, 2014.



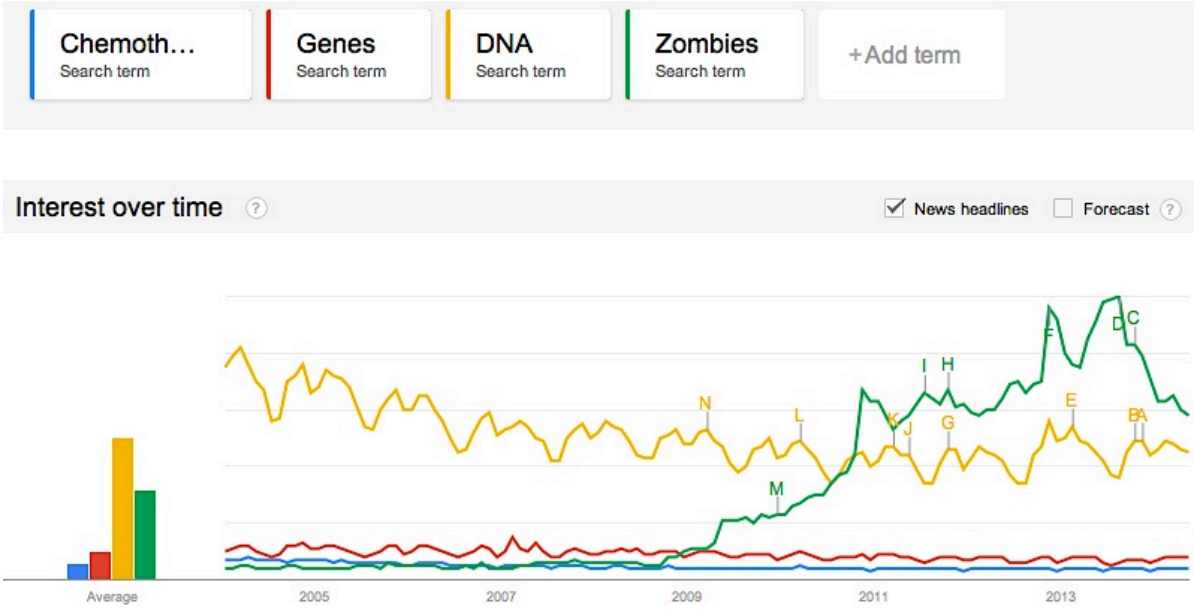
However, there is an overall decline in searching for traditional cancer related treatments.

Fig. 2: Search term trends for 'Chemotherapy,' 'Radiotherapy,' and 'Cancer surgery,' 2004-2014. Screenshot taken May 26th, 2014.



Trials and cancer related gene therapies did not yield a significant trend. The database may lack sensitivity and specificity. However, broad interests in transcription factors i.e. 'Genes,' 'DNA,' can be noted. This is not to imply a surging interest in gene therapies in gastric cancer patients, but a potential awareness and interest in transcription factors overall in the worldwide general population.

Fig. 3: Search term trends comparing 'Chemotherapy,' 'Genes,' 'DNA' and 'Zombies,' 2004-2014. Screenshot taken May 26th, 2014.



Sensitive and specific search terms taken from search engine databases could provide insights into which transcription factors impact the general public. This may not be specific to GC patients, but could nevertheless be validated in this population. The information is then cross-matched to impact factors (H-index) on specific transcription factors, ranked and pooled and verified among experts. Some transcription terms may not be evidence-based. Focus groups may generate novel items intrinsic to QOL. For example, Santa Claus and presents is a more likely association, than a furry robed stranger climbing down your chimney, raiding your fridge, gobbling up your cookies and leaving a sooty mess. Santa is easily identifiable like a transcription term, but associations beyond that is usually beyond

the scope of understanding for a young child – or some might say. Transcription factors adorned with promises and catchy letters may in fact be more memorable than those with awkward letters. Focus groups could assist with which transcription factor terms are romanticised, memorable, hopeful- and contribute to a patient's sense of wellbeing and QOL. The below is a simplified timeline for GCTF-PRO's development:

Phase	**Details**
First	Literature search Theory search – concept mapping
Second	Algorithm sequencing – concept mapping Preliminary analysis part I
Third	Expert opinion interviews Preliminary analysis part II
Fourth	Construct open discussion questionnaire Recruit focus group Focus group interviews
Fifth	Itemise: cross-match with theory – concept defining Adopt Formal analysis methods
Sixth	Construct formal questionnaire Pilot test run questionnaire on individuals
Seventh	Construct outcome estimate model
Eighth	Recruit focus group Validate questionnaire
Ninth	Formal analysis
Tenth	Design web server, upload results, invite health researchers to comment, replicate, monitor and maintain

General approach

The approach is psychometric. Whether the items reflect 'awareness' and 'trust' are two psychosocial dimensions requiring further investigation. At present, without focus groups, it is difficult to assert if the items overwhelm the patient because it is literature-based without the balance of a popularised flourish from the media, or, if a patient's intelligence is a confounder; word associations a confounder; other potential confounders and biases. Each item is anticipated to have its own history, impact and an underlying cognitive association. For example, are certain transcription letter strings more identifiable and contribute to a patient's QOL? The challenge seems to be in establishing effect indicators between 'awareness' and transcription factors; 'trust' and transcription factors; and discerning each item as a tip of a network on perception and cognitive theories. For example, *HER2 positive* is a transcription term featured in the literature, and popularised in the media for causing breast cancer. However, there are other transcription factors like *FOXO* genes that cause growth in cancer cells (Li et al., 2013). *HER* seems appealing and marketable because it is a gender word – *HER* positive breast, whereas *FOXO* is cited often with the gene *RUNX3* for gastric cancers. Then again, RUNNING from a FOX is more memorable than the *IL-6* gene – a skinny set of letters attached to a slouching 'devil' numeral. Item parameters (history, impact, cognitive strength etc.) nestled in a network of real and imagined transcription terms, may serve to facilitate mathematical models that construct ratios – a stronger form of evidence. However, the approach for this draft proposal is to explore a literature-based (transcription factors) GC scale that could readily complement existing GC QOL instruments i.e. the main-effect (QOL improvements) in a QOL instrument, compared to a disease-specific scale adopting a multifactorial analysis (five domains: prognosis, diagnosis, dimensions of care, treatment, and perceptions).

Item generation method

I conducted a literature-search on *Medline(Ovid)* and *Pubmed* from 2013-2014 (See appendix C for search terms). This however presents a literature-based dimension. Ideally, a focus group (GC patients) in an open discussion – given a series of situational vignettes, letter priming and cognitive-based tasks, could present insights on psychosocial dimensions of 'awareness' and 'trust.' Perceptual and cognitive theories should be the driving basis with items generated from GC content. At present, it is difficult to establish how these items affect a patient's QOL unless to complement existing GC QOL instruments, because the scale is disease-specific and overlooks QOL factors. Running algorithms on search engines may generate popularised transcription terms. Interviewing experts on diagnoses and transcription factors may also generate items. This PROM essentially has four item generation methods: A literature search, focus groups, expert opinions (key informant interviews) and algorithm sequencings.

Item selection method

The literature search for domains 1 and 3: I read the abstracts in each article and selected each acronym with a transcription reference. I looked up each term online to ensure each item was related to transcription factors. Domain 2 and 4: I eyeballed and selected terms from reviews and GC PROMs on prognoses, diagnoses, trials and treatments (appendix A). In algorithm sequencings: item selection is anticipated to come from frequency distributions of transcription related terms. However, it would be difficult to filter for GC patients. In focus groups: letter primes and cognitive-based tasks may help facilitate item rank and selection. Transcripts from focus groups and expert opinions could undergo qualitative analysis such as grounding, so that key word selections directly or indirectly associated with transcription factors reflect latent variables intrinsic to a proposed theory.

Aggregation

Domains 1 and 3 consist of 247 items presented concurrently. The resulting score is aggregated to domain 2 consisting of 32 items and domain 4 with 11 items. Domain 5 is a separate construct to the other domains and when completed total 30 items. The total single score possible is 320 (appendix A & B). Items should be pooled and categorised proper after input adjustments from focus groups, expert opinions and algorithm sequencings. The domain scores are compared using multifactorial analysis methods. This PROM is novel – the cut-off thresholds at present, have little a priori basis to be set at certain levels that detect awareness and confidences in trust. Hence in the early stages, observing summary scales between-patients could help direct methods that employ appropriate and effective aggregation strategies.

Instruction comprehensibility

The instructions should be clear from the questionnaire's onset. There is a possibility when unblind, the study's true intention may prompt biased responses. This will require further investigation during the questionnaire's design phase. A general definition of transcription factors prior to administering the questionnaire may ensure the task does not overwhelm participants. Instruction pitfalls to avoid will include ensuring the questions are not ambiguous; double-barrelled, jargon left from item generation and selection, value-laden; worded both positive and negative. Pilot runs and software programs could assist appropriate wording, and selection criteria more sensitive so that the instructions do not confound and bias the outcome.

Item comprehensibility

Given the complexity in transcription terms, numerous content revisions are anticipated so that the items are translatable. Even if the items themselves cover the construct of interest, whether this comprehensibility translates to an exact recall is doubtful. The focus groups and cognition-based heuristics may facilitate some of these anticipated challenges. Recall triggers within letter strings, modifying presentations into meaningful groups, transcription factors embedded in vignette scenarios and so on, are mechanisms that could assist translatability. Sampling issues: Age-related factors may also influence translatability. For instance, are younger age brackets more prone to fluent recall, thus elevates QOL? Recalling transcription terms may not even be a true reflection of 'awareness' and 'trust.' Packaging transcription terms into observable treatment outcomes may in fact show the true effect, as opposed to a theory-based design. Such disparate issues should be discerned when collecting data from focus groups, expert opinions and when sequencing algorithms.

Layout and format

Items on scales should not be worded too long or too short. Holden et al. (1985) found on average, items containing 10 to 20 characters had validity coefficients four times higher than items containing 70 to 80 letters and a load on working memory (Streiner & Norman 2003, p. 82). Item characteristics are an important component to this PROM, and are anticipated attributes in latent variables, hence items should be laid out clearly: such as complex letter strings packaged in evidence-based digestible formats, and tried and tested layouts. It should mimic the preferred layout and format in a GC population – subject to feedback, and analysed for statistical consistency.

Content validity

If the items are translatable i.e. item and semantic equivalence, content validity for domains 1 and 3 should reflect the construct of interest. That being, patient recall of transcription terms and those mentioned by the multidisciplinary team reflect underlying latent traits 'awareness' and 'trust.' However, comprehensibility issues are anticipated: when items are grouped and modified, initial domain inferences and the internal consistency may alter. Hence domains 1, 2, 3 and 4 are dependent on performance differences between-patients, and expert judgements on item content. Domain 5 adheres to Heise's *Affect Control theory* with a pre-determined set of sentiments. Actions are associated with identities (appendix B). Whether this domain accurately depicts areas of trust (construct of interest) is uncertain. It has the potential to yield low content validity and is therefore at present, assigned a full score upon completing the questionnaire.

Face validity

The items on the surface need to appear to measure what it purports to measure. That way, at face value, it is easy to see the nature and purpose of this instrument. This PROM seeks to complement existing GC QOL instruments, to make easy administration and scoring, so that researchers and policy makers can more readily adopt it. Patients could also rate the questionnaire on a 5-point scale (extremely suitable to irrelevant) so that offensive superficial particulars can be re-formatted.

Criterion validity

There appears to be no gold standard for measuring an awareness or trust of transcription factors – not from my literature search. Psychosocial dimensions: 'level of awareness' and 'areas of trust' seem arbitrary concepts requiring further investigation. A clear definition may depend on which perceptual and cognitive theories are included. Rigorous selection criteria and pre-screen selections could ensure patients have had an adequate exposure to transcription factors. This way, specificity can be tested by comparing samples with the exposure to those who have not. Predictive validity: An awareness of cellular level prognoses could correlate with future QOL criteria. However, 'awareness' of transcription factors needs to be translatable when compared to other scale criteria.

Construct validity

The most concern to construct validity is ensuring the content (transcription terms) is translatable. Extensively modifying items so that it is translatable may bias the validity and at worse, end up testing another construct. There are numerous potential biases and confounds when incorporating input from focus groups, expert opinions, and sequencing algorithms. It is anticipated each group recalls transcription factors differently. The challenge is ensuring the questionnaire is cohesive, and adequate methods are employed so that each item is rigorously mapped to principally reflect the construct of interest. A conceptual mapping system can weigh each item in terms of frequency, impact and other factors relative to other items. Bayesian methods (probable estimates) could mathematically deduce a web of strengths and correlations. Although I am uncertain how this effects conventional validity constructs. Whether the scores converge or diverge may depend on which perceptual and cognitive theories are included and defined during the design phase.

Responsiveness

Measuring overall trends in responsiveness, may entail identifying the correlates of change. The questionnaire itself could be the intervening variable for eliciting change. This dilemma means that measuring from the same sample may bias subsequent measures. This could manifest in patients developing a 'motivation' for transcription factors upon completing the questionnaire, instead of remaining true to the construct of interest. Subsequent measures derived from the same sample may in fact be testing other latent variables, such as 'motivation.' At present, measuring responsiveness may be feasible when adopting a Bayesian effect model based on a priori (theory) change correlates. Responsiveness can then be measured continuously in a "metapopulation" that remain true to the construct of interest. However, I am uncertain whether this notion exists and can hold statistically.

Measurement error

At present, the main concern is item translation. Presenting a questionnaire with a list of acronyms may not measure anything at all – hence a tendency in responses to commit non-differential errors and bias the outcome towards the null. No validation, consistency and evidence of agreement in self-reports i.e. failed re-tests or lack of internal consistency, may lead to various measurement errors: inconsistent (numerical) variable responses in the same item within-patients, or inconsistent variable extents between-patients on the true underlying trait. The exposure is also uncontrolled and screened prior, so there could be recall biases leading to differential misclassification errors and bias towards or away from the null. Initial systematic errors are anticipated due to item complexity – all patients may find the items unintelligible- causing bias. Random errors may also arise from inconsistent variable exposures, personal issues, aptitude levels and other unmeasurable pre-screen

selection factors – leading to more inconsistent variability in measurements without a recognisable pattern.

Test-retest reliability

In this PROM, conducting two tests in the same sample is not feasible, and may inflate or deflate intra-individual variability. Exposure to the items first time round makes it more likely the patient recognises the items second time round. Split-half reliability tests may be more feasible. At present, latent variables remain undefined in a psychosocial construct: level of awareness and areas of trust. However, it is anticipated, theory-based criteria may determine re-test variation extents within and between-patients: scores on tests re-adjusted and scaled relative to an a priori estimate criteria within a Bayesian effect model.

Internal consistency

Ideally, *Cronbach's alpha* should be between 0.8-0.9. Factor analysis (when items are grouped and constructed into meaningful chunks) can help eliminate and group items based on correlations. Different aspects of the same construct can also be revealed through item loadings. For instance, highly popularised or memorable transcription terms are anticipated to yield consistent variance, whereas obscure terms may not add much meaningful value to the outcome because it yields no consistent variance. At present, based on nominal scales in which items are either identified or not – *Kuder Richardson Formula 20* could test for correlations and provide a psychometric quality to individual items which can facilitate the removal of certain items and contribute to theory-driven directions.

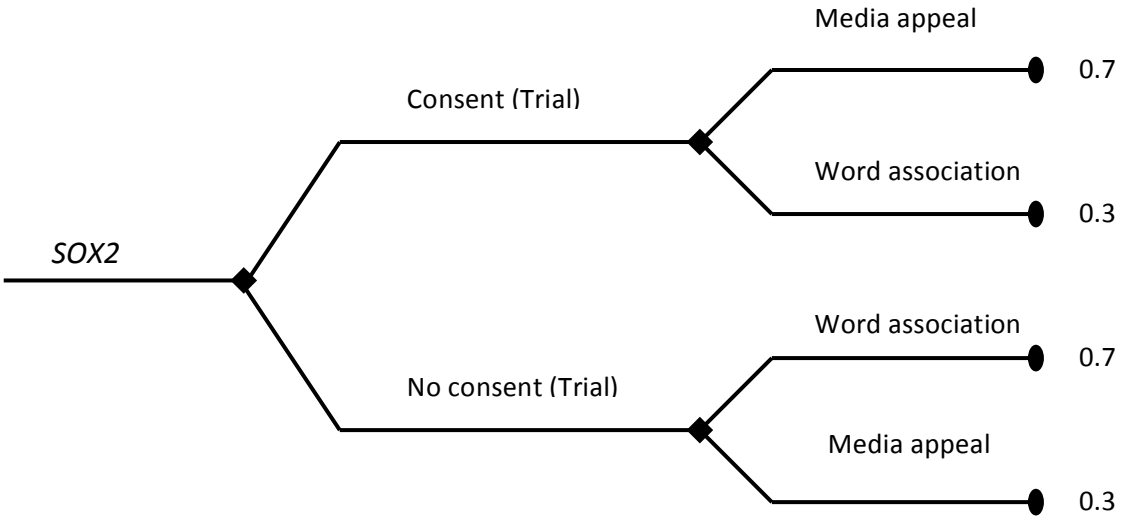
Reducing error methods

Focus groups, expert opinions and algorithm sequencings may better package items into meaningful chunks, groups or vignettes based on perception and cognition theories. This provides an a priori basis for measuring specific factors that can be analysed with existing studies. Clearly defining and translating items may decrease the likelihood of committing exposure related measurement errors. Validating the PROM in intervention studies comparing GC patients and control groups may refine measurement methods that test and control for variability in changes over time. Information collected on potential confounders prior to administering the PROM, may also decrease errors in confounder measurements. Repeating and grouping items in different forms, for instance, *HER* receptors in a popularised vignette and in a list, may assist with verification methods for addressing non-differential biases. Assigning a level of exposure on transcription factors and being stringent with selection criteria may reduce selection errors. Assessing reasons for participating or not participating in the PROM may also assist with reducing errors.

Preference-based utility model

At present, this PROM seeks to complement existing GC QOL instruments (multi-attribute health status classification systems: EORTC QLQ-C30, EORTC QLQ-STO22, FACT-G, FACT-Ga, MDASI-GI) and does not address the design features associated with QOL, so a *cost-effective analysis* might be more appropriate (Drummond et al. 2005, p. 138). However, if considering utility measures, the population of interest would need to be more attuned to recombinant DNA technology and cellular level prognosis so that their preferences, QALYs and trade-offs can be precisely mapped. In this setting, the risk attitudes in preference measurements may entail consenting to a GC cancer trial because the transcription factor looks promising and it

features often in the media. These decision options could be mapped using normative or behavioural models i.e. perceptual and cognitive findings. The below maps an example with the transcription factor *SOX2* in a population screened with the exposure (dummy results):



The relative values for each decision is weighed and assessed and measures cardinal preferences (standard gamble method). On the other hand, *consumer choice* methods (ordinal utility) might be more appropriate for measuring media appeal. Later on, utilities and classification systems in this PROM may include descriptions of transcription factors that extend across disease states in a QOL paradigm. For now, the type of utility measure adopted could complement existing instruments depending on who the party of interest is.

Clinical results implications

The clinical implications of cost-effective ratios in a cost-utility analysis may mean enacting policies in health parastatals that re-envision efficient and effective prognostic/ diagnostic methods. This may include communicating across transcription factors to GC patients using biotechnology devices. Health professionals may need to be re-trained on transcription

factors pioneered in research industries, so that patients can ask questions confidently (Tzelepis et. al, 2014). Influences from the media can also be made accountable when debunked and addressed by a health professional. A patient no longer needs to consider risks alone when consenting to a trial – if there are adequate support systems to ensure health professionals are communicating with research sectors and to a patient, this may improve long-term efficiency in treatment outcomes and to a patient's QOL.

Resource results implications

Monitoring and communicating across transcription factors require access to biotechnology information and devices. It may in fact be inevitable more and more people in the future will naturally gravitate towards knowing more about their genetic make up and other cellular level information. Research in this area is also rapidly expanding with intelligent technological advances. The results from cost-utility analyses on transcription factors may be more widely funded when it is arguably commensurate to consumer need. At present, cost-effectiveness as utility weights, measure artefacts on assessment processes shaped by theory-driven constructs but not the patient's real-world choices (Lenert & Kaplan 2000, p. II-139; See diagram below). Algorithm sequencings, expert opinions and focus groups, may not reflect real-world choices, particularly for this PROM because such preference systems for transcription factors do not yet exist. Hence it would be unreliable to conduct multiple regressions or descriptive analyses alone for calculating preference-based utility measures (Dobrez et al. 2007). However, a Bayesian approach for interpreting utility data could estimate the patient's utility *before* measurement. Researchers can then aggregate these measurements and form estimate mean utilities in populations and groups.

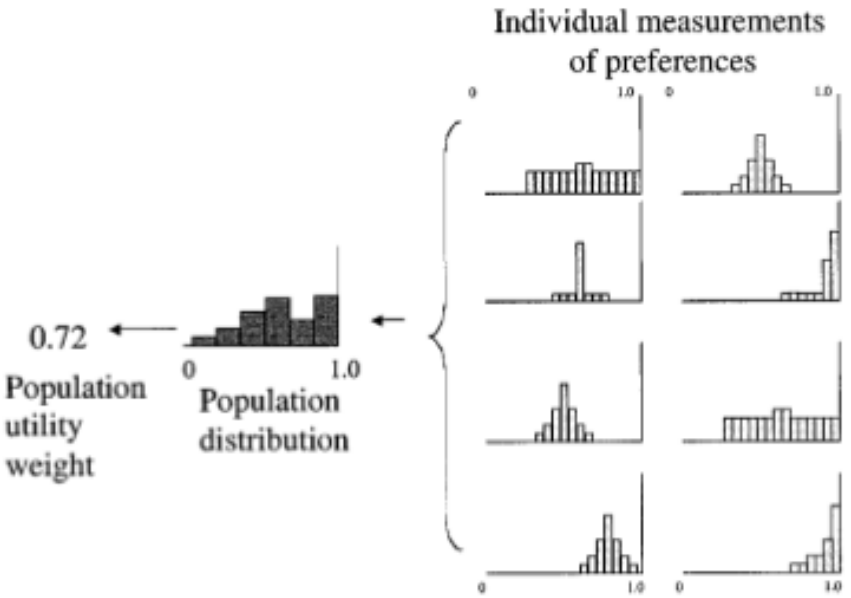


FIG. 1. Model illustrating factors underlying a population preference weight. The population weight is a summary of the population distribution of preferences. This in turn is a summary of the posterior (eg, postassessment) distributions of individuals' true preferences.

This approach can further be modelled to obtain a preference elicitation (Lenert & Kaplan 2000, p. II-141).

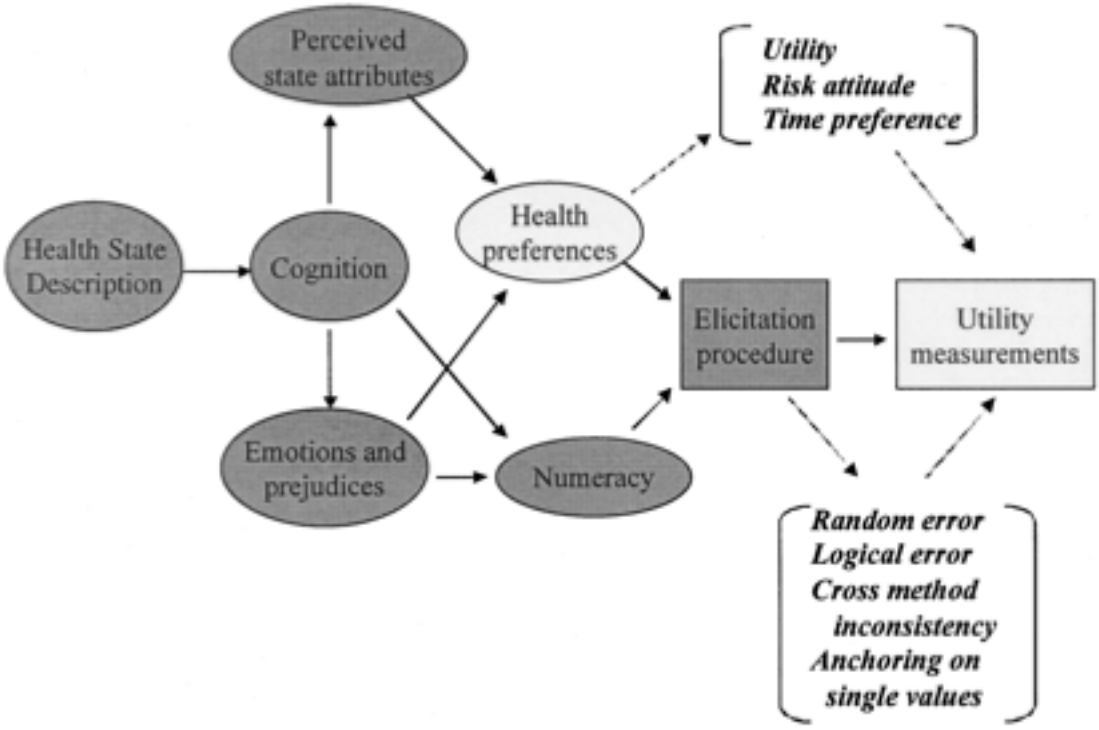


FIG. 2. Model of the process of obtaining a preference elicitation.

Without going into detail, a preference elicitation framework for transcription factors may provide a broader platform to the processes concerning utility weights, and disseminate more openly on resource allocation policies.

Study question, hypotheses and outcomes

Answerable question in PICO format:

Population	|	Gastric Cancer Patients Stage I-IV (male and female; all ages).
Intervention	|	Cellular localisation prognoses i.e. biomarkers, genes.
Comparator	|	No reference to cellular localisation.
Outcome	|	"Awareness" of transcription factors affect QOL.

→ *Can cellular localisation prognoses affect QOL in GC patients?*

Hypothesis: "Awareness" of transcription factors affects QOL in GC patients.

$\mathbf{H_0}$: No association between transcription factors and QOL.

$\mathbf{H_1}$: "Awareness" of transcription factors affects QOL.

The study question examines whether exposure to cellular localisation prognoses affect the GC patient's sense of wellbeing or QOL (outcome).

Outcome measures, endpoints, frequency, assessment duration

Outcome measures

The outcome measure is based on whether exposure to transcription factors affects a patient's sense of wellbeing or QOL. Since this PROM seeks to complement existing instruments, the 'affect' is determined by the underlying latent variables embedded in the instrument: between those exposed to transcription factors and those who have not. The outcome measure (this PROM) aggregates a single score on transcription 'awareness levels' (dichotomous scale; at present) and 'areas of trust' (semantic differential rating scale).

Endpoints

At present, a higher score assumes a higher awareness level with a maximum score of 320. A single endpoint is anticipated so the outcome is clearly defined and directly transferable.

Frequency

The PROM is administered once but it needs to be presented at a point in time when there is a general tendency for GC patients exposed to cellular level prognoses.

Assessment duration

The questionnaire is self-completed, at present it is uncertain how long the questionnaire will take to complete. It is however a one off assessment with no follow-up.

Repeated measures, missing values, multiple testing

The problem with repeated measures and multiple testing in this PROM – the exposure to the questionnaire may prime the patient to seek further transcription related information. Different time points could be measured in different staging for different groups, but repeated measures on the same population at different time points, most likely will end up measuring something else. What could be done, is a Bayesian effect estimate of a future time point based on a GC population at an earlier time point. The future time point is then validated in another GC population. For example, the PROM is administered to stage III locally advanced GC patients and estimated by stage IV to present a more higher level of awareness, and distrust in health professionals. The Bayesian modelled effect (modified to summary statistics; Frison & Pocock, 1992) can then compare *its* estimate to a future time point in a GC population stage IV. This way, a priori theories embedded in the Bayesian effect model can be refined and offer a consistent numerical framework for monitoring

changes over time. Ideally, this approach means that attrition rates, missing values and other issues cannot considerably skew the overall analysis.

Results presentation

The results should be presented succinct with accompanying flow diagrams. For example, a tree diagram with probable estimates. A summary of transcription terms – its impact factor, association and other algorithm sequencings could be categorised in a summary in respect to outcome. The analysis for each transcription term should be attached to the appendix so the proposed model can be replicated, distributed and updated by other researchers. Disseminating the results will most likely entail publishing in a journal and conforming to guidelines: Labelled figures and tables, summary of the findings and presentations in line with the CONSORT statement, COCHRANE, APA or other standards depending on which study or review was used to validate and present the PROM.

Significance of proposed PROM

The GCTF-PRO seeks to examine the extent GC patients are tapping into new information particularly outside of conventional healthcare disclosures. Its significance is in assessing dimensions of QOL paradigms that frame statistical power using predictive methods. It seeks to embed evidence-based theories (perceptual and cognitive) to awareness levels in an attempt to bridge the biotechnological advances with prognostic/ diagnostic-related patient satisfactions. At present, it may complement existing GC QOL instruments and offer a novel proposal on how cellular level prognoses could possibly correlate with QOL.

New knowledge

Is the population developing a symbiosis with biotechnology? Should we be re-envisioning conventional treatment strategies i.e. dysfunctional multidisciplinary teams and paternalism despite "patient-centred" exteriors, to one where a patient is informed and can monitor their progress at a cellular level, and can truly make informed decisions about their treatment? These are practical problems this PROM alone cannot answer, but nevertheless seeks to raise social discourse on potential solutions. In terms of QOL, this PROM seeks to frame theories from existentialism – Sartre: Human beings cannot be fully questioned and understood with science or morals. "Authenticity" demonstrates a practical, embodied individual, true to one's personality and self. This is the "quality" and new knowledge the GCTF-PRO seeks to operationalise.

Results implication

The results will most likely have adopted predictive methods not normally applied in health research. This implies an emphasis on design and modelling before collecting data and conducting interviews in a manner that reflect the underlying theory. If an awareness of transcription factors is correlated with QOL, then these results may promulgate further methods. For instance, deducing correlation extents; constructing causal pathways; explore in depth the areas it taps into; refine methods to test errors, validity, reliability, selection and measurement; construct time measures (end points) based on numerical models that can be updated collectively by health researchers. Validate predictive and probabilistic models, as opposed to relying on disparate populations to detect change and validate instruments. Generally, this approach may allow health researchers to be a step ahead and to numerically forecast the patient's QOL based on a unified model.

Health service provisions and outcomes for target patient group

If there is a correlation between QOL and transcription factors, further studies may look into which cellular level prognostic/diagnostic coherency alleviates a patient's sense of wellbeing. Provision of health services may include: formally educating GC patients on transcription factors and regularly communicating across cellular level activities; targeted therapies based on genetic and epigenetic profiles (Søreide & Søreide, 2013); utilising proteomic therapeutics (Lin, Huang & Juan, 2012); investing in commercial devices which can monitor cellular level activities at home; synchronise the patient's progress into a seamless program – containing pathology reports, medical scans, updates, appointments and other notes; design efficient systems which can ensure open and transparent dialogue between health professionals and research industries. These are overarching health service provisions not necessarily limited to GC patients. However, for GC patients, when the prognosis is poor, this uncertainty may set off a chain of mixed attitudes that determine their QOL. The GCTF-PRO attempts to explore how GC patients are affected by these uncertainties in respect to their sense of wellbeing and QOL, and avenues in which health services can sufficiently address the needs of GC patients.

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Appendix

Appendix A

Table 1: Transcription terms for domain 1 and 3 grouped alphabetically.

A, B, C, D, E	F, G, H, I, J	K, L, M, N, O
1. ADAMTS1 expression	61. Fanconi anemia (FA) gene	103. KAP1 (Kinesin II-associated protein)
2. ADAMTS9 expression	62. F-box leucine rich protein 11	104. KLF4 (Kruppel-like factor 4) expression
3. AFP (Alpha-fetoprotein)	63. FOX01 (Forkhead Box Protein)	105. KLF5
4. AhR (Aryl hydrocarbon receptor)	64. FOXM1	106. KLF8 transcription factor
5. Alpha-308	65. FOX06	107. KLK12 expression (kallikrein)
6. Angiogenesis	66. FOXD3	108. KRAS mutations
7. Apoptosis	67. FOXP3	109. Leptin receptor
8. AKT signaling	68. Fibroblast	110. LIGHT genes
9. ASCL2 (Achaete scute-like 2) transcription factor	69. FISH	111. MAWD expression MEG-3 (Maternally expressed gene 3)
10. AUY922	70. FGFR (Fibroblast Growth Factor Receptor)	112. MCG-803 gastric cancer cell
11. BMP pathway	71. FGFR2	113. MCL1 expression
12. BMP2 pathway	72. Gastrin receptor	114. MET amplification
13. BTF3 (Basic transcription factor 3)	73. GATA1 expression	115. MERTK signaling
14. Beta signaling	74. GATA2	116. MicroRNA-18a
15. BEZ235	75. GATA3	117. MicroRNA-21
16. BCL-2 expression	76. GES-1	118. MicroRNA-27a
17. BYL719	77. Gli-1	119. MicroRNA-181c
18. CagPAI effector	78. GPR48	120. MicroRNA-106a
19. Carcinogenesis	79. GRIM-19	121. Micro-RNA-183
20. Caveolin-1	80. HDAC1 (Histone deacetylase 1) expression	122. MicroRNA polymorphs
21. CCAT1 expression	81. Hedgehog (Hh) singalling	123. MIR-22
22. CCK2R (cholecystokinin 2 receptor)	82. HGFR (Hepatocyte Growth Factor Receptor)	124. MIR-129
23. CDC25A	83. HER1	125. MIR-202-3p
24. CD8	84. HER2	126. MIR-196b
25. CD44	85. HER-2/neu	127. MIR-301a
26. CD100 subset gastric cancer cells	86. HER3	128. MIR-365
27. CD133 expression	87. HER4	129. MIR-370
28. CAC1 (CDK-associated Cullin1)	88. HIC1 gene	130. MKN-1 cells
29. CDX1 expression	89. HIF-1alpha expression	131. MKN-45 cell line
30. CDX2 expression	90. HMGB3 expression	132. MLHI promoter
31. CpG hypomethylation		133. MMP-2 (Matrix metalloproteinase-2)

32. CK19 RT-LAMP 33. Cordon-bleu protein-like 1 34. COX-2 35. COL4A3 expression 36. CXCR1 37. Cyclin D1 38. Cyclooxygenase-2 gene 39. Cytology 40. Cytokine signaling 41. Damage-specific DNA binding protein 1 42. DDX6 protein 43. DLX2 expression (Distal-less homeobox 2 44. E2F family of transcriptions 45. E2F2 46. E2F4 47. E2F8 48. EBV (Epstein-Barr virus) 49. E-cadherin (CDHI) 50. ECRG4 gene expression 51. Eculizumab 52. EGFR expression 53. EGCG inhibitor (green tea) 54. Epithelial-mesanchymal 55. EGR1 56. EMT (Epithelial-mesenchymal transition) 57. ERBB2 58. ERCC1 59. ETV1 transcription factors 60. Everolimus	91. HopQ 92. HOXA10 93. HSP90A 94. Hypermethylation 95. IHC 96. IFI-27 (Interferon alpha-inducible protein 27) 97. IL-6 expression 98. IL-8 99. IL-26 expression 100. JAK2 signalling 101. JNK (c-June N-terminal kinase) 102. JWA expression	134. MMP-9 135. MMP-14 136. MMR (Mismatch repair) 137. Monoclonal antibodies 138. MSI (Microsatellite Instability) 139. mTOR (Mammalian Target of Rapamycin) 140. MUC1 expression 141. MUC4 expression 142. Myeloid cell trafficking Metastasis-associated gene (MTA3) 143. MSC (Mesenchymal stem cells) 144. MZF1 expression 145. NDRG1 expression 146. Neoplasm 147. Non-small cell lung carcinoma 148. NOD1 (nucleotide-binding oligomerization domain 1) 149. Notch2 activation 150. NF-B transcription factor 151. NFYA expression 152. NFYB expression 153. NFYC expression 154. NUCB2 155. Nuclear receptor coactivator-6 156. OPB-31121 inhibitor

P, Q, R, S, T	U, V, W, X, Y, Z	Numerals
157. P13K (Phosphatidylinositol-3-kinase) 158. PAI-1 (plasminogen activator inhibitor-1) 159. PDS5B gene 160. PEGFP-ZNRF3 161. PIK3CA 162. Phosphatase 163. PG100 164. PKM2 (Pyruvate kinase M2) 165. PLAC 166. Proteomics 167. Protease-activated receptor-2 168. P21 169. P27 170. p53 171. Protein 4 172. PRKAA1 gene 173. PSMB1 (proteasome subunit) 174. PSK (Protein-bound polysaccharide K) 175. PS-101 phenotype screening 176. PTEN (Phosphatase and tensin homolog) 177. rhGH (recombinant human growth hormone) 178. PTGER4 gene 179. RASSF proteins 180. REL expression 181. RT-PCR 182. RT-qPCR 183. RUNX3 184. Semaphorin 6B 185. SGC7901 cell line 186. SGOL1 gene 187. Signet-ring cell 188. SIP1 (small interacting protein 1)	228. UFT (tegafur uracil) 229. UHRF1 epigenetic regulator 230. VEGFA 231. VEGFR-1 232. VEGFR-2 233. VEZT 234. VGLL4 235. WDR62 expression 236. Wnt signaling 237. WWOX gene 238. YCC-3 cell line 239. YAP 240. YB-1 gene 241. YF476 242. ZEB1 gene 243. ZEB2 gene 244. ZNRF3 (Zinc and ring finger 3) 245. ZFX (Zinc finger transcription)	246. 1alpha-Hyroxylase 247. 5-gene signature

189. SISH 190. Smad4 191. Smad7 192. Snail 193. SNU-5 cell line 194. SOCS3 195. SOX2 expression 196. SOX9 expression 197. SOX17 expression 198. SP1 expression 199. SRY expression 200. STAT3 binding site 201. T4SS (Type IV secretion system) 202. STC2 (stanniocalcin 2) 203. STKM-1 204. TCF signaling 205. TBX5 expression 206. TEAD1 207. Tensin homolog 208. TERT (Telomerase reverse transcriptase) 209. TFF3 (Trefoil factor 3) 210. TFR2 expression 211. TGF-alpha (Tumor necrosis factor-alpha) 212. TGF-beta 213. TH17 214. TIMP1 (TIMP metallopeptidase inhibitor 1) 215. TLN2 (talin 2) 216. TLR4 signalling 217. TNF-alpha 218. Trastuzumab 219. TRC (Transcriptase-reverse transcriptase concerted reaction) 220. TR3 receptor 221. Trefoil factor 1 222. Treg cells 223. TSG101 expression 224. Tumorigenesis 225. TXN (Thioredoxin) 226. TXNIP expression		

227. WNT signalling		

Table 2: Transcription terms for domain 2 listed alphabetically.

1. Adenocarcinoma	17. Lump
2. Aggressive	18. Lymph nodes
3. Bad cells	19. Malignant
4. Cells	20. Metastasis
5. Chemotherapy	21. Nasty
6. Complete Blood Count (CBC)	22. Pathology
7. CT scan	23. PET scan
8. Endoscopy	24. Peritoneum
9. Gastric lining	25. Protein
10. Good cells	26. Radiotherapy
11. Helicobacter pylori	27. Scirrhous
12. Hemoglobin	28. Spread
13. HER2	29. T-cells
14. Immune system	30. TNM staging
15. Laparoscopy	31. Tumor
16. Locally advanced	32. White cells

Table 3: Transcription terms for domain 4 listed alphabetically based on Duraes et al. 2014 review.

1. LoGIC trial (lapatinib chemo combination)	7. SHINE study
2. EXPAND study (cetuxmab chemo combination)	8. MetGastric trial (onartuzumab chemo combination)
3. ENRICH study	9. GRANITE-1 (everolimus placebo combination)
4. FLEX trial	10. ToGA trial (trastuzumab chemo combination)
5. AVAGAST GC trial (bevacizumab chemo combination)	11. Immunochemotherapy
6. REGARD (ramucirumab chemo combination)	

Appendix B

Evaluation Potency Activity (EPA)

Note: Explanation of transcription factors given prior to administering the questionnaire.

When it comes to transcription factors, please rate 'cancer health specialists' in terms of the following qualities. Mark X in the slot.

Evaluation (Status)

Helpful ___ : ___ : ___ : ___ : ___ : ___ : ___ Unhelpful

Attentive ___ : ___ : ___ : ___ : ___ : ___ : ___ Paternal

Informed ___ : ___ : ___ : ___ : ___ : ___ : ___ Uninformed

Networked ___ : ___ : ___ : ___ : ___ : ___ : ___ Distant

Modern ___ : ___ : ___ : ___ : ___ : ___ : ___ Conventional

Potency (Power)

Powerful ___ : ___ : ___ : ___ : ___ : ___ : ___ Powerless

Strong ___ : ___ : ___ : ___ : ___ : ___ : ___ Weak

Deep ___ : ___ : ___ : ___ : ___ : ___ : ___ Shallow

Safe ___ : ___ : ___ : ___ : ___ : ___ : ___ Risk

Knowledgeable ___ : ___ : ___ : ___ : ___ : ___ : ___ Unknowledgeable

Expressivity (Expressivity)

Noisy ___ : ___ : ___ : ___ : ___ : ___ : ___ Quiet

Informative ___ : ___ : ___ : ___ : ___ : ___ : ___ Uninformative

Forward ___ : ___ : ___ : ___ : ___ : ___ : ___ Behind

Light ___ : ___ : ___ : ___ : ___ : ___ : ___ Dark

Open ___ : ___ : ___ : ___ : ___ : ___ : ___ Closed

Appendix C

Medline (results as of 26th May 2014)

1. gastric cancer.mp. or Stomach Neoplasms (77991 articles)

2. With the above and below terms (2218) results. Confined to 2013-2014 (225 articles)

GATA6 Transcription Factor/ or Ikaros Transcription Factor/ or E2F7 Transcription Factor/ or SOX Transcription Factors/ or Transcription Factor AP-2/ or Sp Transcription Factors/ or ARNTL Transcription Factors/ or Transcription Factor TFIID/ or GATA Transcription Factors/ or STAT4 Transcription Factor/ or Transcription Factor RelB/ or E2F4 Transcription Factor/ or Sp1 Transcription Factor/ or E2F2 Transcription Factor/ or MSX1 Transcription Factor/ or Otx Transcription Factors/ or GATA1 Transcription Factor/ or TCF Transcription Factors/ or Sp3 Transcription Factor/ or Paired Box Transcription Factors/ or Transcription Factor CHOP/ or Pol1 Transcription Initiation Complex Proteins/ or Transcription Factors, TFII/ or Winged-Helix Transcription Factors/ or E2F6 Transcription Factor/ or Transcription Factor Brn-3A/ or Transcription Factors, TFIII/ or Transcription Factor TFIIH/ or Transcription Factor AP-1/ or Basic-Leucine Zipper Transcription Factors/ or GATA5 Transcription Factor/ or Transcription Termination, Genetic/ or Maf Transcription Factors/ or COUP Transcription Factor I/ or Transcription Factor Brn-3/ or STAT2 Transcription Factor/ or NFI Transcription Factors/ or Transcription Factors, General/ or Transcription Factor 7-Like 1 Protein/ or STAT1 Transcription Factor/ or Transcription Factor Brn-3B/ or Transcription Factors/ or GATA3 Transcription Factor/ or SOXB1 Transcription Factors/ or Octamer Transcription Factor-2/ or MafF Transcription Factor/ or SOX9 Transcription Factor/ or Transcription Factor Pit-1/ or Twist Transcription Factor/ or COUP Transcription Factor II/ or MafK Transcription Factor/ or PAX2 Transcription Factor/ or E2F1 Transcription Factor/ or Octamer Transcription Factor-6/ or STAT5 Transcription Factor/ or PAX9 Transcription Factor/ or p300-CBP Transcription Factors/ or NF-E2 Transcription Factor/ or SOXB2 Transcription Factors/ or Sp2 Transcription Factor/ or Transcription Factor TFIIB/ or E2F5 Transcription Factor/ or STAT Transcription Factors/ or Transcription Factor 3/ or Reverse Transcription/ or Onecut Transcription Factors/ or SOXC Transcription Factors/ or SOXF Transcription Factors/ or SOXD Transcription Factors/ or E2F3 Transcription Factor/ or Activating Transcription Factor 3/ or Activating Transcription Factor 2/ or Transcription Factor 7-Like 2 Protein/ or YY1 Transcription Factor/ or GATA4 Transcription Factor/ or Transcription Factor TFIIA/ or STAT6 Transcription Factor/ or Transcription, Genetic/ or GA-Binding Protein Transcription Factor/ or GATA2 Transcription Factor/ or MafG Transcription Factor/ or Sp4 Transcription Factor/ or T Cell Transcription Factor 1/ or E2F Transcription Factors/ or PAX7 Transcription Factor/ or SOXE Transcription Factors/ or Transcription Initiation Site/ or Octamer Transcription Factor-1/ or Fushi Tarazu Transcription Factors/ or Transcription Factor TFIIIA/ or Octamer Transcription Factors/ or STAT3 Transcription Factor/ or MEF2 Transcription Factors/ or Early Growth Response Transcription Factors/ or NF-E2 Transcription Factor, p45 Subunit/ or Activating Transcription Factor 1/ or COUP Transcription Factors/ or Forkhead Transcription Factors/ or Transcription Factor DP1/ or Transcription Factor TFIIIB/ or AraC Transcription Factor/ or Transcription Elongation, Genetic/ or Maf Transcription Factors, Large/ or Activating Transcription Factor 4/ or Transcription Factor Brn-3C/ or Octamer Transcription Factor-3/ or Basic Helix-Loop-Helix

Transcription Factors/ or Transcription Initiation, Genetic/ or NFATC Transcription Factors/ or MafB Transcription Factor/ or Basic Helix-Loop-Helix Leucine Zipper Transcription Factors/ or Activating Transcription Factor 6/ or Transcription Factor RelA/ or Kruppel-Like Transcription Factors/ or Maf Transcription Factors, Small/ or transcription.mp. or Microphthalmia-Associated Transcription Factor/ or Activating Transcription Factors/

Pubmed:

Search terms: ((Gastric cancer) AND Stomach neoplasms) AND Transcription

2184 articles: results as of 26th May 2014.